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PREVALENCE OF SHORT STATURE AND ITS ASSOCIATION WITH ANAEMIA IN CHILDREN AGED 1–14 YEARS: A HOSPITAL-BASED CROSS-SECTIONAL STUDY FROM UTTAR PRADESH, INDIA

Dr. Ritik Chandra*	Junior Resident, Department of Paediatrics, Dr. KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, India (Affiliated to Atal Bihari Vajpayee Medical University, Lucknow) *Corresponding Author
Prof. (Dr.) Mohd Kashif	Professor and Head, Department of Paediatrics, Dr. KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, India
Prof. (Dr.) Sadaf Mirza	Professor and Head, Department of Pathology, Dr. KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, India
Dr. Yusuf Ahmed	Assistant Professor, Department of Paediatrics, Dr. KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, India

ABSTRACT

Background: Short stature and anaemia are prevalent nutritional disorders in children from low- and middle-income countries, yet their concurrent occurrence and the quantitative haemoglobin–height relationship remain inadequately characterised in hospital-based Indian cohorts spanning the full childhood age range. **Objectives:** To determine the prevalence of short stature (HAZ < −2 SD) and anaemia (WHO criteria), to establish the correlation between haemoglobin and height, and to characterise associated nutritional and socioeconomic determinants. **Methods:** A hospital-based, observational, cross-sectional study using consecutive sampling enrolled 255 children aged 1–14 years from the Department of Paediatrics, Dr. KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, over 18 months. Standardised anthropometric measurements and automated haematology analysis (Sysmex XN-1000) were performed. Dietary intake was assessed by a single structured 24-hour recall. Data were analysed using IBM SPSS v26. **Results:** Short stature was noted in 16 children (6.3%), with rural children showing a significantly higher prevalence (9.3%) than urban children (1.9%) (χ^2=4.605; p=0.033). Anaemia was present in 204 children (80.0%): mild 38.4%, moderate 31.0%, severe 10.6%; mean haemoglobin 9.1 ± 1.2 g/dL. Morphologically, 70.1% of anaemic children showed a microcytic hypochromic pattern consistent with iron deficiency. A statistically significant moderate positive correlation was demonstrated between height and haemoglobin (r = 0.43, p < 0.001). Short stature prevalence showed a graded increase: 2.0% in non-anaemic children rising to 25.9% in those with severe anaemia (χ^2=18.7; p < 0.001). Iron intake was 52.4% below the ICMR Recommended Dietary Allowance. **Conclusion:** Short stature and anaemia demonstrate a significant positive association within this hospital-based paediatric cohort from Uttar Pradesh and share common nutritional and socioeconomic determinants. Integrated clinical assessment and programmatic responses addressing iron deficiency and undernutrition as co-linked targets are warranted. Prospective longitudinal studies are needed to establish the direction and causality of this relationship.

KEYWORDS : Short Stature; Anaemia; Iron Deficiency; Haemoglobin; Paediatric Malnutrition; Height-for-age z-Score; India

INTRODUCTION

Short stature and anaemia are two of the most consequential nutritional disorders affecting children in low- and middle-income countries (LMICs), imposing substantial burdens on physical growth, cognitive development, immune competence, and long-term human capital. Both conditions share deep common roots in micronutrient deficiency-particularly iron, zinc, and folate-recurrent infections, poverty, and inadequate complementary feeding, yet they are frequently evaluated and managed as distinct clinical entities.

Globally, approximately 149.2 million children under five were stunted in 2020, with South Asia bearing the largest regional burden (34.9%).

[3] India's National Family Health Survey-5 (NFHS-5, 2019–21) documented stunting in 35.5% of under-five children nationally, with Uttar Pradesh recording 39.7% — among the highest in the country.

Anaemia is equally pervasive: 67.1% of Indian children aged 6–59 months were anaemic per NFHS-5, a figure that rose from 58.6% in NFHS-4, representing a reversal of a previously declining trend.[4,5] Iron deficiency anaemia, the most prevalent micronutrient deficiency globally, is the dominant contributor, driven by cereal-predominant diets low in bioavailable iron, recurrent gastrointestinal infections, and early cow's milk feeding in the Indian context.[6,7]

The biological interdependence of anaemia and linear growth has been well characterised: iron is essential for the GH–IGF-1 axis that regulates post-infancy bone elongation, for collagen synthesis enzymes in bone matrix formation, and for mitochondrial energy production in osteoblasts.[8,9,10]Despite this, robust hospital-based data quantifying the concurrent prevalence of short stature and anaemia across the full paediatric age range (1–14 years) and establishing a quantitative haemoglobin–height correlation are sparse from Uttar Pradesh.[12] Most Indian studies are limited to under-five children; the school-age group (5–14 years) has received comparatively little attention.[13] Furthermore, failure to screen for treatable iron deficiency before labelling a child as having idiopathic short stature represents an important clinical gap.[14,15]

The present study was therefore designed to determine the prevalence of short stature (HAZ < −2 SD) and anaemia (WHO haemoglobin criteria) in children aged 1–14 years attending a tertiary care centre in Barabanki, Uttar Pradesh; to establish the association between haemoglobin concentration and height; to characterise the morphological type of anaemia; and to describe the nutritional and socioeconomic determinants of both conditions.

METHODS

Study Design, Setting, and Sampling

This was a hospital-based, observational, cross-sectional study conducted in the Department of Paediatrics, Dr. KNS

Memorial Institute of Medical Sciences (MIMS), Barabanki — a 500-bed tertiary care teaching hospital affiliated to Atal Bihari Vajpayee Medical University, Lucknow. The study was conducted over 18 months using consecutive sampling of eligible children attending the paediatric outpatient department (OPD) or admitted to the inpatient ward during the study period. All participants were enrolled in the order of presentation until the target sample was achieved. The study was conducted following Institutional Ethics Committee (IEC) approval, and written informed consent was obtained from a parent or legal guardian for all participants. The study is reported in accordance with the STROBE guidelines for cross-sectional observational studies.

Study Population and Sample Size

Children aged 1–14 years (completed) of either sex, with informed consent and capacity for standardised measurement, constituted the study population. Sample size was calculated a priori using an estimated stunting prevalence of 35% from NFHS-5 Uttar Pradesh data, with a 95% confidence interval and an allowable absolute error of 6%, using the formula $n = Z^2 \times p \times q/d^2 = 242.7$. Adding a 5% attrition allowance yielded a final target of 255 children, which was fully achieved. No data were missing for the primary outcome variables (haemoglobin and height); complete data were available for all 255 participants for the principal analysis.

Inclusion and Exclusion Criteria

Children aged 1–14 years of either sex with written informed consent were included. Excluded were children with: chromosomal disorders (Down syndrome, Turner syndrome) or skeletal dysplasias; haemoglobinopathies, including sickle cell disease and beta-thalassaemia major or intermedia — identified by prior confirmed diagnosis in medical records, corroborating family history, and, where clinically indicated, haemoglobin electrophoresis or high-performance liquid chromatography (HPLC); severe acute malnutrition requiring inpatient ready-to-use therapeutic food (RUTF) protocol; active systemic infections, specifically meningitis, septicaemia, and active tuberculosis (confirmed by clinical, microbiological, or radiological criteria); other chronic or subclinical infections including chronic respiratory disease and clinically suspected or confirmed parasitic infestations (assessed by presenting symptoms, clinical examination, and available stool microscopy records — children with documented recent deworming within three months or active helminthic infestation confirmed clinically were excluded); malignancies or ongoing cytotoxic or hormonal therapy; and significant limb deformities precluding accurate height measurement.

Anthropometric Measurements

All measurements followed WHO anthropometric assessment protocols. Height was measured using a calibrated wall-mounted stadiometer (Seca 217, precision ±0.1 cm) in children aged ≥24 months; recumbent length was recorded using an infantometer (Seca 210) for younger children. Weight was measured on a calibrated digital platform scale (Seca 874, precision ±0.1 kg). Short stature was defined as a height-for-age z-score (HAZ) below −2 SD from WHO 2006/2007 growth standards, computed with WHO Anthro v3.2.2 (under-5) and WHO AnthroPlus v1.0.4 (5–14 years). The term 'short stature' is used throughout in the clinical sense of HAZ < −2 SD; the term 'stunting' is reserved for community-based epidemiological usage in under-five children and is used solely to contextualise NFHS-5 comparisons. BMI-for-age z-scores (BAZ) were classified per WHO/IAP 2022 criteria. Mid-upper arm circumference (MUAC) was assessed in children under five (n=81) using WHO-standard non-stretch tapes: SAM <12.5 cm, MAM 12.5–13.4 cm, normal ≥13.5 cm.

Haematological Assessment and Definition of Anaemia

Venous blood (3–5 mL) was collected into EDTA-coated vacutainer tubes and processed within two hours using the Sysmex XN-1000 automated haematology analyser (subject to daily internal quality-control calibration). Parameters measured included haemoglobin (g/dL), packed cell volume (%), MCV (fL), MCH (pg), MCHC (g/dL), and RDW-CV (%). Anaemia was defined and severity-classified using WHO 2024 age-specific haemoglobin thresholds: children aged 6–23 months, Hb <10.5 g/dL; children aged 24–59 months, Hb <11.0 g/dL; children aged 5–11 years, Hb <11.5 g/dL; and children aged 12–14 years, Hb <12.0 g/dL.16 Severity grading: mild anaemia, Hb 10.0 g/dL to threshold; moderate, 7.0–9.9 g/dL; severe, <7.0 g/dL. Peripheral blood smears stained with Leishman stain were examined at 100× oil-immersion magnification by an experienced pathologist blinded to clinical details. Morphological classification combined red cell indices with smear findings to assign each anaemic child to one of four categories: microcytic hypochromic (MCV <80 fL, MCH <27 pg),17 consistent with iron deficiency or thalassaemia trait; normocytic normochromic; macrocytic (MCV >100 fL); or mixed/dimorphic (normal or near-normal MCV with RDW-CV >15% and bimodal red cell population on smear), consistent with concurrent iron and B12/folate deficiency.17 MCV and RDW cutoff values are based on established haematological reference criteria.17 It is acknowledged that morphological classification is inferential; confirmatory biochemical indices (serum ferritin, TIBC, transferrin saturation, vitamin B12, and folate) were not obtained, which represents a methodological limitation.

Dietary Assessment

Dietary intake was assessed by a single structured 24-hour dietary recall administered in Hindi by trained research personnel using standardised household measurement aids (cups, spoons, and pictorial food models) to estimate portion sizes. Interviews were conducted during a face-to-face consultation; interviewers were trained in standardised recall methodology and used a pre-tested, structured proforma to minimise interviewer variability and recall bias. Caloric and micronutrient content was estimated using the Indian Food Composition Tables (IFCT 2017, NIN, Hyderabad) and compared against age- and sex-adjusted ICMR Recommended Dietary Allowances (2020). Dietary inadequacy was defined as intake below 80% of the RDA for the respective age and sex category. It is acknowledged that a single 24-hour recall may not accurately capture habitual dietary intake given day-to-day variability, and this constitutes a limitation of the nutritional data.

Statistical Analysis

Data were analysed using IBM SPSS Statistics v26.0. Prior to parametric testing, normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Continuous variables conforming to a normal distribution are expressed as mean ± SD; non-normally distributed data are expressed as median with interquartile range (IQR). The primary statistical outcome — the linear relationship between height (cm) and haemoglobin (g/dL) — was assessed using Pearson's correlation coefficient (r) following confirmation of normality; Spearman's rank correlation was used for any pair of variables deviating from normality. Categorical comparisons used Pearson's chi-square test or Fisher's exact test as appropriate. Group differences for continuous variables were assessed using independent samples t-test, Mann–Whitney U, one-way ANOVA with Tukey's HSD post-hoc, or Kruskal–Wallis H test as appropriate. It is acknowledged that the primary analysis reported here is bivariate and is not adjusted for potential confounders including age, sex, infection burden, and socioeconomic status; multivariable regression was considered but not performed owing to power constraints on subgroup cell sizes, and this is recognised as a limitation. All tests were two-tailed; $p < 0.05$ was the threshold for statistical significance.

RESULTS

A total of 255 children were enrolled with complete data for all primary outcome variables. The mean age was 8.93 ± 4.07 years; 52.5% were female and 47.5% male. Urban and rural children comprised 41.2% and 58.8% respectively. Approximately 70.6% of families belonged to lower-middle or low-income socioeconomic strata, and 60.0% of guardians had educational attainment at or below primary level. Immunisation was complete in 60.4% and incomplete or absent in 39.6% of children. Demographic characteristics are presented in Table 1.

Table 1. Demographic and Socioeconomic Characteristics of the Study Population (n = 255)

Characteristic	n	%
Total enrolled	255	100.0
Age: 1–2 years	26	10.2
Age: 3–5 years	55	21.6
Age: 6–8 years	48	18.8
Age: 9–11 years	62	24.3
Age: 12–14 years	64	25.1
Gender: Female	134	52.5
Gender: Male	121	47.5
Residence: Urban	105	41.2
Residence: Rural	150	58.8
SES: Low income (<₹5,000/month)	66	25.9
SES: Lower middle (₹5,001–10,000)	114	44.7
SES: Upper middle / High (>₹10,000)	75	29.4
Immunisation: Complete	154	60.4
Immunisation: Incomplete/None	101	39.6
Guardian education: ≤Primary level	153	60.0
Mean age (years ± SD)	8.93 ± 4.07	

SES: Socioeconomic status (Modified Kuppuswamy Scale). Mean values expressed as mean ± SD.

Nutritional and Anthropometric Status

BMI-for-age z-score classification showed thinness (Grade I–III) in 66.3% of children, with only 29.0% normally nourished. Among under-five children (n=81), MUAC assessment identified SAM in 22.2% and MAM in 35.8%, indicating a high burden of acute-on-chronic malnutrition. These MUAC-based indicators of acute malnutrition and BAZ-based indicators of chronic nutritional status reflect different dimensions of the malnutrition spectrum and are not directly comparable to HAZ-based short stature. The most severe caloric deficit by 24-hour recall was in the youngest age group (1–2 years: 22.5% below ICMR RDA). Iron intake was 52.4% below the age-adjusted RDA (mean 8.1 mg/day versus recommended ~17 mg/day), folate 69.0% below, and zinc 47.5% below — indicating a compound micronutrient deficit that substantially exceeds the aggregate caloric deficit alone (18.0% below RDA).

Prevalence and Severity of Anaemia

Anaemia was detected in 204 of 255 children (80.0%), distributed as: mild 38.4%, moderate 31.0%, severe 10.6%. Mean haemoglobin was 9.1 ± 1.2 g/dL. Red cell indices showed a microcytic, hypochromic pattern: mean MCV 71.6 ± 8.4 fL (reference 75–96 fL), MCH 24.3 ± 1.4 pg (reference 27–31 pg), MCHC 30.8 ± 1.9 g/dL (reference 32–36 g/dL), and an elevated RDW of 15.9 ± 1.9% (reference ≤14.5%), indicating significant anisocytosis. Severe anaemia was most prevalent in the youngest age group (1–2 years: 23.1%) and declined with age (12–14 years: 3.1%), reflecting the heightened physiological vulnerability of toddlers following exhaustion of fetal iron stores. Morphologically, microcytic hypochromic anaemia consistent with the iron deficiency pattern dominated at 70.1% of anaemic children, followed by normocytic normochromic (16.2%), mixed/dimorphic (10.8%), and macrocytic (2.9%). Anaemia data are presented in Table 2.

Table 2. Prevalence, Severity, and Morphological Classification of Anaemia (n = 255)

Anaemia Category	n	% (n=255)
Normal Haemoglobin	51	20.0
Mild Anaemia	98	38.4
Moderate Anaemia	79	31.0
Severe Anaemia	27	10.6
Total Anaemic	204	80.0
Mean Haemoglobin (g/dL)	9.1 ± 1.2	
Mean MCV (fL)	71.6 ± 8.4	
Mean RDW-CV (%)	15.9 ± 1.9	
— Morphological Type (n=204 anaemic) —		% of 204
Microcytic Hypochromic (IDA-consistent pattern)	143	70.1
Normocytic Normochromic	33	16.2
Mixed/Dimorphic (Fe + B12/Folate consistent)	22	10.8
Macrocytic (B12/Folate deficiency consistent)	6	2.9

WHO 2024 haemoglobin thresholds applied by age group. Morphological classification based on red cell indices (MCV, MCH, MCHC, RDW-CV) and peripheral blood smear examination; this is an inferential classification. Biochemical confirmation (serum ferritin, TIBC, B12, folate) was not performed.

Prevalence of Short Stature and Urban–Rural Comparison

Short stature (HAZ < −2 SD) was identified in 16 of 255 children (6.3%), a figure that is notable within this hospital-based clinical sample but should not be directly equated with community stunting estimates, which use a different sampling frame and are generally restricted to under-five children. A statistically significant rural–urban difference was observed: rural children had a notably higher short stature prevalence (9.3%) than urban children (1.9%) (^{2}=4.605; p=0.033). Concurrent short stature and anaemia (any severity) was documented in 15 children (5.9%), significantly more prevalent in rural (8.7%) than urban (1.9%) children (^{2}=5.82; p=0.016). The concurrent burden of short stature with severe anaemia was also higher in rural children (4.0% vs. 1.0%; p=0.046). These data are presented in Table 3.

Table 3. Short Stature Prevalence, Urban–Rural Comparison, and Anthropometric Status (n = 255)

Parameter	Urban (n=105)	Rural (n=150)	Total (n=255)
Short Stature (HAZ < −2 SD)	2 (1.9%)	14 (9.3%)	16 (6.3%)
Normal Stature	103 (98.1%)	136 (90.7%)	239 (93.7%)
χ^2 = 4.605, p = 0.033*			
Concurrent Short Stature + Anaemia (any)	2 (1.9%)	13 (8.7%)	15 (5.9%)
Concurrent Short Stature + Severe Anaemia	1 (1.0%)	6 (4.0%)	7 (2.7%)
χ^2 (concurrent) = 5.82, p = 0.016*			
BMI: Any Thinness (Grade I–III)	—	—	169 (66.3%)
BMI: Normal Nutrition	—	—	74 (29.0%)
SAM by MUAC (<12.5 cm)†	—	—	18 (22.2%)
MAM by MUAC (12.5–13.4 cm)†	—	—	29 (35.8%)

HAZ: Height-for-age z-score. SAM: Severe acute malnutrition. MAM: Moderate acute malnutrition. †MUAC assessed in under-five children only (n=81); BAZ and MUAC reflect distinct dimensions of malnutrition (chronic vs. acute). * p < 0.05, chi-square test.

Association Between Haemoglobin and Height: Primary Outcome

Pearson's correlation analysis, following confirmation of approximate normality of both variables by the Kolmogorov–Smirnov test, demonstrated a statistically significant moderate positive association between height and haemoglobin ($r = 0.43$, $p < 0.001$). This indicates a moderate, though statistically robust, relationship between haemoglobin concentration and linear growth in this cohort. Haemoglobin also correlated with BMI ($r = 0.41$), MUAC ($r = 0.39$), and weight ($r = 0.37$), all at $p < 0.001$. A graded increase in short stature prevalence was observed across anaemia severity categories: 2.0% (normal haemoglobin) → 2.0% (mild) → 7.6% (moderate) → 25.9% (severe anaemia) ($\chi^2=18.7$; $p < 0.001$). Children with severe anaemia showed approximately 13-fold higher short stature prevalence compared to non-anaemic children within this sample. Primary outcome data are presented in Table 4.

Table 4. Association Between Anaemia Severity and Short Stature — Primary Outcome (n = 255)

Anaemia Category	Total n	Short Stature n (%)	Normal Stature n (%)
Normal Haemoglobin	51	1 (2.0%)	50 (98.0%)
Mild Anaemia	98	2 (2.0%)	96 (98.0%)
Moderate Anaemia	79	6 (7.6%)	73 (92.4%)
Severe Anaemia	27	7 (25.9%)	20 (74.1%)
Total	255	16 (6.3%)	239 (93.7%)
$\chi^2 = 18.7$; $p < 0.001$*			
Pearson r (Height vs Haemoglobin) = 0.43; $p < 0.001$*			

Pearson r (height cm vs haemoglobin g/dL): $r = 0.43$ (moderate association), $p < 0.001$ (two-tailed, $n=255$). Association is unadjusted for potential confounders. * $p < 0.05$, chi-square test.

DISCUSSION

This hospital-based cross-sectional study of 255 children aged 1–14 years in Barabanki, Uttar Pradesh, demonstrated three principal findings that were consistent with the a priori hypothesis. First, short stature (HAZ < −2 SD) was present in 6.3% of children within this clinical sample, with a notable rural excess (9.3% vs. 1.9% urban; $p=0.033$). Second, anaemia was highly prevalent at 80.0%, with a mean haemoglobin of 9.1 ± 1.2 g/dL; the morphological pattern was most consistent with iron deficiency in 70.1% of anaemic children, concurrent iron and B12/folate deficiency in 10.8%, and normocytic or macrocytic patterns in the remainder. Iron intake estimated by 24-hour recall was 52.4% below the ICMR RDA. Third, and most importantly for the primary objective, a statistically significant moderate positive association was demonstrated between height and haemoglobin ($r = 0.43$, $p < 0.001$), with a graded increase in short stature prevalence across anaemia severity categories from 2.0% in non-anaemic to 25.9% in severely anaemic children ($\chi^2=18.7$; $p < 0.001$). Secondary outcomes revealed that 66.3% of children had thinness by BAZ classification, 22.2% of under-five children had SAM by MUAC, and caloric and micronutrient deficits were pervasive across all age groups, with the youngest children bearing the greatest proportional deficit.

The haemoglobin–height association ($r = 0.43$) is consistent with comparable hospital-based studies from similar settings: Gad et al. from Egypt ($r = 0.38$, $p < 0.001$),[1] Singh et al. from Lucknow ($r = 0.38$),[19] Gautam et al. from Bihar ($r = 0.41$),[20] and Kapoor et al. from Uttarakhand ($r = 0.29$).[17] The cross-setting consistency supports the biological plausibility of the association, though it does not exclude the possibility of shared confounders — particularly poverty, dietary diversity, and infection burden — operating simultaneously across these settings. Multiple mechanisms could plausibly underlie this relationship: iron is required for osteoblast collagen synthesis and mitochondrial energy production essential for bone elongation, and iron deficiency may suppress the GH–IGF-1 axis through elevated phosphorylated IGFBP-1 in tissue-hypoxic states.[8,9,10] However, since no mechanistic biomarkers were measured in this study, these pathways represent explanatory frameworks consistent with the data rather than confirmed mediators. The morphological predominance of the microcytic hypochromic pattern (70.1%) aligns with the documented dietary iron deficit; the 10.8% mixed/dimorphic prevalence — characterised by a near-normal MCV masking bimodal red cell populations on peripheral smear through elevated RDW-CV — is clinically significant as it may be under-recognised if only MCV is evaluated.[17] This pattern suggests concurrent iron and B12/folate deficiency, plausible in this predominantly vegetarian, low-income population, though biochemical confirmation was not obtained in the present study.

The principal strengths of this study are the extension of the age range to the full paediatric spectrum (1–14 years) beyond the under-five focus of most national datasets; comprehensive haematological profiling including peripheral smear morphological classification; structured dietary recall providing nutritional context; and the inclusion of urban and rural children enabling comparative analysis. However, several methodological constraints must be acknowledged. The hospital-based consecutive sampling design introduces selection bias that likely inflates prevalence estimates relative to community rates, and the prevalence of short stature (6.3%) should not be interpreted as equivalent to community stunting rates, which differ in both sampling frame and age restriction. The comparison with NFHS-5 stunting data (under-five community estimates) is not directly equivalent to this study's hospital-based 1–14-year cohort and is presented for contextual orientation only. Iron deficiency anaemia was inferred morphologically in the absence of serum ferritin, TIBC, or transferrin saturation; misclassification risk exists, particularly for thalassaemia trait and anaemia of chronic disease, which may share similar morphological appearances. The single 24-hour dietary recall is subject to day-to-day variability and recall bias, potentially limiting the accuracy of nutrient intake estimates. The primary analysis is bivariate and unadjusted for potential confounders including age, sex, infection burden, and socioeconomic status, limiting the independence of the reported association. The claim of "100% data completion" refers specifically to the absence of missing values for haemoglobin and height, the two primary outcome variables; it is not a claim of complete data across all secondary variables.

The central controversy this study raises is the question of causality. The graded dose–response relationship between anaemia severity and short stature prevalence ($\chi^2=18.7$; $p < 0.001$) is consistent with a biologically plausible causal pathway from iron deficiency to growth impairment. However, as a cross-sectional study, the data capture exposure and outcome simultaneously, and it is not possible from these data to determine whether anaemia preceded growth faltering, whether growth faltering preceded or worsened anaemia, or whether both conditions are parallel consequences of the same underlying nutritional deprivation. The dose–response gradient, while one element of causal reasoning under Bradford Hill's criteria, is insufficient without temporality.23 The observed moderate correlation ($r = 0.43$) must be interpreted as an association of moderate strength, not a strong or causal relationship. A further controversy is the appropriate threshold for defining 'significant' morphological iron deficiency without biochemical confirmation: the microcytic hypochromic pattern used here is standard clinical practice but carries a non-trivial misclassification rate in settings where thalassaemia trait is prevalent.[17]

The findings are consistent with evidence that supports

consideration of haemoglobin estimation as part of the initial evaluation of children presenting with growth faltering, before attributing short stature to idiopathic or constitutional causes — particularly in high-anaemia-burden settings such as Uttar Pradesh.[14,15] Systematic RDW evaluation in all anaemic children is advisable, as the mixed/dimorphic pattern requires a targeted therapeutic approach distinct from iron monotherapy; however, combined supplementation should ideally be guided by biochemical confirmation of deficiency. The rural–urban gradient observed suggests that rural children in this catchment area may warrant prioritised screening, though this finding should be interpreted with the selection bias of hospital-based sampling in mind. These findings add to a growing body of evidence from hospital-based Indian cohorts supporting an association between iron deficiency and impaired linear growth, and are broadly consistent with the programmatic rationale of the Anemia Mukt Bharat and POSHAN Abhiyaan frameworks, which integrate iron supplementation, dietary diversification, deworming, and growth monitoring as convergent interventions. Prospective longitudinal studies that track children with documented anaemia through structured iron supplementation protocols — measuring height velocity, serum ferritin, and IGF-1 as primary outcomes, while adjusting for infection status, repeated dietary assessments, and socioeconomic confounders using multivariable modelling — represent the most pressing research direction this study highlights. Such studies would enable determination of temporality and, ultimately, the reversibility of anaemia-related growth impairment in this population.[24,25]

CONCLUSION

This study demonstrates that anaemia (80.0%) and short stature (6.3%, notable within this hospital-based sample) co-exist at substantial frequency in children aged 1–14 years attending a tertiary care centre in Barabanki, Uttar Pradesh. A statistically significant moderate positive association was identified between height and haemoglobin ($r = 0.43$, $p < 0.001$), with a graded increase in short stature prevalence from non-anaemic to severely anaemic children ($\chi^2=18.7$; $p < 0.001$). The morphological pattern most consistent with iron deficiency dominated at 70.1% of anaemic children, supported by documented dietary iron intake 52.4% below the ICMR RDA. Rural children bore a disproportionately higher burden of both conditions. These findings are consistent with a plausible biological association between iron deficiency and impaired linear growth, though the cross-sectional design precludes causal inference. Integrated clinical assessment of haemoglobin and growth parameters in children presenting with either condition is supported by these findings. Prospective longitudinal studies incorporating biochemical markers, repeated dietary assessments, and multivariable modelling are needed to establish the direction and causal nature of the observed association and to assess the impact of iron supplementation on linear growth outcomes in this population.

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