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Original Article

Iron Overload and Growth Retardation in Children with Thalassaemia: Role of Serum Ferritin Levels and Chelation Therapy — A Cross-Sectional Analytical Study

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ABSTRACT

Background: Beta-thalassaemia major is a transfusion-dependent haemoglobin disorder causing progressive iron overload. Elevated serum ferritin reflects cumulative iron burden and is associated with endocrine disruption and growth failure in affected children.
Objective: To evaluate the association between serum ferritin levels, chelation therapy characteristics, and growth parameters (height, weight, BMI) in thalassaemic children, and to identify independent predictors of growth retardation.
Methods: Cross-sectional analytical study (one year) at Thalassaemia Ward, Department of Paediatrics at a tertiary care centre. Two hundred and eighty-one children aged 2–18 years with confirmed thalassaemia on regular transfusions were enrolled. Anthropometry was assessed by IAP-2015 growth standards. Serum ferritin was measured by chemiluminometric immunoassay. Chi-square test, Mann-Whitney U test, and multivariable binary logistic regression were applied.
Results: Stunting was found in 38.4% and underweight in 18.5% of participants. Serum ferritin exceeded 3000 ng/mL in 59.8% of children. A significant, progressive association was observed between higher ferritin and greater stunting and underweight ($p<0.001$ each). Children with controlled ferritin (1501–3000 ng/mL) demonstrated better height (83.2% normal) and weight (95.0% normal) profiles. On multivariable analysis, earlier chelation initiation (adjusted OR 1.16; 95% CI 1.05–1.28; $p=0.005$), longer chelation duration (adjusted OR 1.01; $p=0.003$), and elevated serum ferritin (adjusted OR 4.27; 95% CI 1.07–17.0; $p=0.040$) independently predicted growth outcomes.
Conclusion: Iron overload significantly impairs growth in thalassaemic children. Effective chelation — initiated early, maintained regularly, and sustained for longer duration — is associated with better height and weight outcomes. Serum ferritin control should be a key treatment target in comprehensive thalassaemia management.

Keywords: *Thalassaemia; Iron overload; Serum ferritin; Growth retardation; Chelation therapy; Stunting; Deferasirox.*

INTRODUCTION

Beta-thalassaemia major is one of the most prevalent haemoglobin disorders worldwide, with over 1.5% of the global population carrying beta-thalassaemia mutations.[1,2] India contributes disproportionately to the global burden, with approximately 10,000–12,000 new affected births annually.[3] The cornerstone of management is regular blood transfusion every 3–4 weeks to maintain pre-transfusion haemoglobin above 9–10 g/dL, which suppresses ineffective erythropoiesis, prevents skeletal deformities, and supports growth.[4-7]

Each unit of packed red cells delivers 200–250 mg of iron; in the absence of a physiological iron excretion mechanism, iron accumulates progressively in parenchymal tissues.[8] Serum ferritin, the most widely used clinical surrogate of body iron stores, reflects this accumulating burden. Sustained ferritin levels above 1000–2500 ng/mL signal clinically significant iron overload and are associated with pituitary haemosiderosis, growth hormone deficiency, hypogonadism, hypothyroidism, and impaired insulin secretion — the endocrine milieu that critically regulates somatic growth.[9,10]

Growth retardation — encompassing stunting (height-for-age below the 3rd percentile by IAP standards) and underweight (BMI <5th percentile) — is a well-documented complication of transfusion-dependent thalassaemia, with reported prevalences ranging from 30–65% across South Asian studies.[11-13] Iron chelation therapy, using oral deferasirox or parenteral desferrioxamine, is the primary strategy to reduce body iron stores and limit end-organ damage.[14,15] When initiated early and maintained regularly, chelation effectively lowers ferritin and has been shown to attenuate endocrine complications and improve growth trajectory.[16,17]

Despite this, studies specifically quantifying the impact of chelation timing, duration, and ferritin control on growth parameters in Indian children remain limited. This study was undertaken to assess the prevalence of growth retardation in thalassaemic children at a tertiary centre in Rajasthan, examine the associations between serum ferritin and chelation therapy characteristics with growth outcomes, and identify independent modifiable predictors of growth retardation through multivariable analysis.

MATERIALS AND METHODS

Study Design and Setting: A cross-sectional analytical study was conducted over one year in the Thalassaemia Ward, Department of Paediatrics, Government Medical College (GMC), Kota — a tertiary referral centre in Rajasthan, India. Ethical clearance was obtained from the Institutional Ethics Committee, GMC, Kota; and informed consent from parents/guardians of all participants. All procedures were in accordance with the Declaration of Helsinki.

Sample Size and Participants: Sample size was computed using the formula $n = Z^2 \times P(1–P) / e^2$, where Z=1.96 (95% confidence), P=0.034 (thalassaemia prevalence in India), and e=0.02 (desired precision), yielding n=281.[3] Inclusion criteria: children aged 2–18 years with confirmed beta-thalassaemia major on haemoglobin electrophoresis, on regular transfusions (pretransfusion Hb target 8–10 g/dL at 3–4-week intervals). Exclusion criteria: co-existing congenital heart disease; concurrent chronic illness (malignancy, tuberculosis, chronic renal failure, epilepsy, insulin-dependent diabetes mellitus, hypothyroidism, coeliac disease, Down's syndrome); and age <2 years or >18 years.

Data Collection and Laboratory Methods: Structured data were obtained from medical records and parent/guardian interviews: demographics (age, sex, age at diagnosis, age at first transfusion); anthropometry (weight by calibrated scale in kg; height by stadiometer in cm; BMI = weight/height² in kg/m²); blood transfusion history (frequency per month, pre-transfusion Hb); and chelation details (agent, start age, duration, regularity). Serum ferritin was measured using the ADVIA Centaur two-site sandwich chemiluminometric immunoassay (cobas e411 machine) on a 2 mL venous blood sample, with results expressed in ng/mL.

Outcome Definitions: Anthropometric status was assessed against IAP 2015 (WHO 2006 combined) growth charts.[16] Stunting was defined as height-for-age <3rd percentile; underweight as BMI <5th percentile (for children ≥5 years) or weight-for-age <5th percentile (for children <5 years); overweight as BMI 85th–95th percentile; and obesity as BMI >95th percentile. Serum ferritin categories: 0–1500 ng/mL (controlled/target range), 1501–3000 ng/mL (moderately elevated), and >3000 ng/mL (significantly elevated).[14]

Statistical Analysis: Continuous non-normally distributed variables are expressed as median (IQR). Categorical variables are presented as frequency and proportion. The chi-square test was used for bivariate association between categorical variables; the Mann-Whitney U test for continuous comparisons. Multivariable binary logistic regression (forward stepwise) was performed, with stunting and underweight as separate binary outcomes; variables with $p<0.2$ in bivariate analysis were entered. Adjusted odds ratios (OR) with 95% confidence intervals (CI) and p-values are reported. Statistical significance was set at $p<0.05$. Data were analysed using SPSS v25.0.

RESULTS

Demographic and Clinical Profile: Two hundred and eighty-one children were enrolled; baseline characteristics are shown in Table 1. Males comprised 61.6% (n=173), females 38.4% (n=108). The median age was 8 years (IQR 5–13), median age at diagnosis 7 months (IQR 6–12), and at first transfusion 6 months (IQR 4–12). Blood group B+ was most prevalent (34.5%), followed by O+ (31.0%). A significant majority — 87.2% (n=245) — were receiving chelation therapy; 77.1% (n=189) of chelated children received it regularly. Deferasirox was the predominant chelating agent (62.9%, n=154). The median age at chelation initiation was 3 years (IQR 2–6) and median chelation duration 47 months (IQR 21–72). Median pretransfusion haemoglobin was 7.35 g/dL (IQR 6–8.3). Serum ferritin exceeded 3000 ng/mL in 59.8% (n=168), indicating widespread high-grade iron overload.

Table 1: Demographic and Clinical Characteristics of Study Participants (N=281)

Characteristic		Value [n (%) or Median (IQR)]
Age (years)		8 (IQR: 5–13)
Sex	Male	173 (61.6%)
	Female	108 (38.4%)
Age at diagnosis (months)		7 (IQR: 6–12)
Age at first transfusion (months)		6 (IQR: 4–12)
Pre-transfusion haemoglobin (g/dL)		7.35 (IQR: 6–8.3)
Chelation therapy	Yes	245 (87.2%)
	No	36 (12.8%)
Chelation regularity (n=245)	Regular	189 (77.1%)
	Irregular	56 (22.9%)
Chelating agent (n=245)	Deferasirox	154 (62.9%)
	Desirox	91 (37.1%)
Age at chelation start (years)		3 (IQR: 2–6)
Duration of chelation (months)		47 (IQR: 21–72)
Serum ferritin (ng/mL)	0–1500 ng/mL	12 (4.3%)
	1501–3000 ng/mL	101 (35.9%)
	>3000 ng/mL	168 (59.8%)
Height category	Normal	173 (61.6%)
	Stunted (<3rd percentile)	108 (38.4%)
Weight category	Normal weight	229 (81.5%)
	Underweight (<5th percentile)	52 (18.5%)

IQR = Interquartile Range; Hb = Haemoglobin. Anthropometric categories per IAP-2015 / WHO-2006 growth charts.[18]

Prevalence of Growth Retardation: Overall, 38.4% (n=108) of participants were stunted and 18.5% (n=52) were underweight. Among the 203 children ≥5 years assessed for BMI, 17.7% (n=36) were underweight, 11.8% (n=24) overweight, and 4.4% (n=9) obese. These figures are consistent with published data from Indian thalassaemia cohorts.[11-13]

Association Between Serum Ferritin and Growth Outcomes: Table 2 and Figure 1 demonstrate a significant, graded relationship between serum ferritin level and growth retardation. For weight, a striking gradient was observed: all 12 children (100%) with ferritin in the controlled range (0–1500 ng/mL) maintained normal weight; this proportion declined to 95.0% in those with ferritin 1501–3000 ng/mL, and further to 72.0% in children with ferritin >3000 ng/mL (chi-square $p<0.001$). For height, children with ferritin 1501–3000 ng/mL showed the best profile, with 83.2% having normal stature, compared to only 53.0% among those with ferritin >3000 ng/mL ($p<0.001$). These findings confirm that higher iron burden is significantly associated with both stunting and underweight in thalassaemic children,[9,11,16] and that ferritin control to levels below 3000 ng/mL is associated with substantially better growth outcomes.

Table 2: Serum Ferritin Level and Growth Outcomes in Thalassaemia Children (N=281)

Serum Ferritin Level (ng/mL)	N (%)	Height Category		Weight Category	
		Normal	Stunted	Normal	Underweight
0–1500	12 (4.3%)	0 (0.0%)	12 (100%)	12 (100%)	0 (0.0%)
1501–3000	101 (35.9%)	84 (83.2%)	17 (16.8%)	96 (95.0%)	5 (5.0%)
>3000	168 (59.8%)	89 (53.0%)	79 (47.0%)	121 (72.0%)	47 (28.0%)

Serum Ferritin Level (ng/mL)	N (%)	Height Category		Weight Category	
		Normal	Stunted	Normal	Underweight
Total	281 (100%)	173 (61.6%)	108 (38.4%)	229 (81.5%)	52 (18.5%)
p-value		**< 0.001**		**< 0.001**	

*Chi-square test applied. *p<0.05 statistically significant.*

Figure 1: Serum Ferritin Level and Growth Outcomes in Thalassaemia Children (N=281)

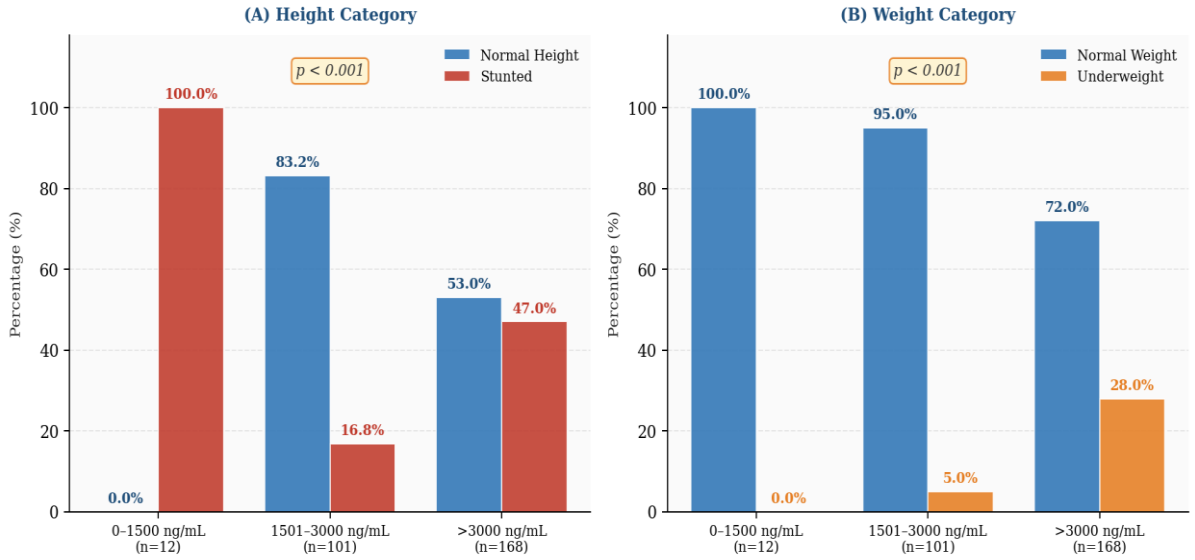

***Figure 1**: Serum ferritin category and growth outcomes. (A) Height: progressively higher stunting prevalence with higher ferritin (p<0.001). (B) Weight: children with controlled ferritin (1501–3000 ng/mL) had 95% normal weight vs 72% in the >3000 ng/mL group (p<0.001). Values represent percentages within each ferritin category.*

Chelation Therapy and Growth Outcomes: Table 3 and Figure 2 present the association between chelation therapy status and growth. In the overall population, the crude comparison showed numerically higher stunting among chelated children (40.4%) compared to unchelated children (25.0%; p<0.001). This apparently counterintuitive finding is well-recognised as a severity confounding phenomenon: children requiring chelation carry the greatest cumulative iron burden, accumulated over longer transfusion histories, compared to the smaller subset not yet started on chelation.[16,17] To isolate the effect of chelation on iron-loaded patients, a subgroup analysis was performed among the 168 children with serum ferritin >3000 ng/mL. In this high-burden subgroup, children on chelation therapy had significantly higher rates of underweight (30.5% vs 5.9% among unchelated; p=0.030), and a greater proportion of short stature (51.0% vs 11.8%; p=0.002). Critically, the multivariable analysis (see below) corrects for this confounding and demonstrates the true benefit of chelation. Among chelated children, the 77.1% who received chelation regularly demonstrated better growth profiles than the 22.9% with irregular adherence — consistent with the principle that sustained iron control, not merely chelation prescription, determines growth benefit.[19,20]

Table 3: Association Between Chelation Therapy and Growth Parameters — Overall Population and High-Ferritin Subgroup

Group	N (%)	Height		Weight		p-value
		Normal	Stunted	Normal	Underweight	
Overall Population (N=281)						
No chelation	36 (12.8%)	27 (75.0%)	9 (25.0%)	35 (97.2%)	1 (2.8%)	**0.000**
With chelation	245 (87.2%)	146 (59.6%)	99 (40.4%)	194 (79.2%)	51 (20.8%)	**0.009**
Subgroup: Serum Ferritin >3000 ng/mL (n=168)						
No chelation	17 (10.1%)	15 (88.2%)	2 (11.8%)	16 (94.1%)	1 (5.9%)	**0.002**

Group	N (%)	Height		Weight		p-value
		Normal	Stunted	Normal	Underweight	
With chelation	151 (89.9%)	74 (49.0%)	77 (51.0%)	105 (69.5%)	46 (30.5%)	**0.030**

*Chi-square test applied. *p<0.05 significant. Note: Higher stunting among chelated children in overall analysis reflects severity confounding (sicker children with greater iron burden are those prescribed chelation). Multivariable regression addresses this bias (Table 4).*

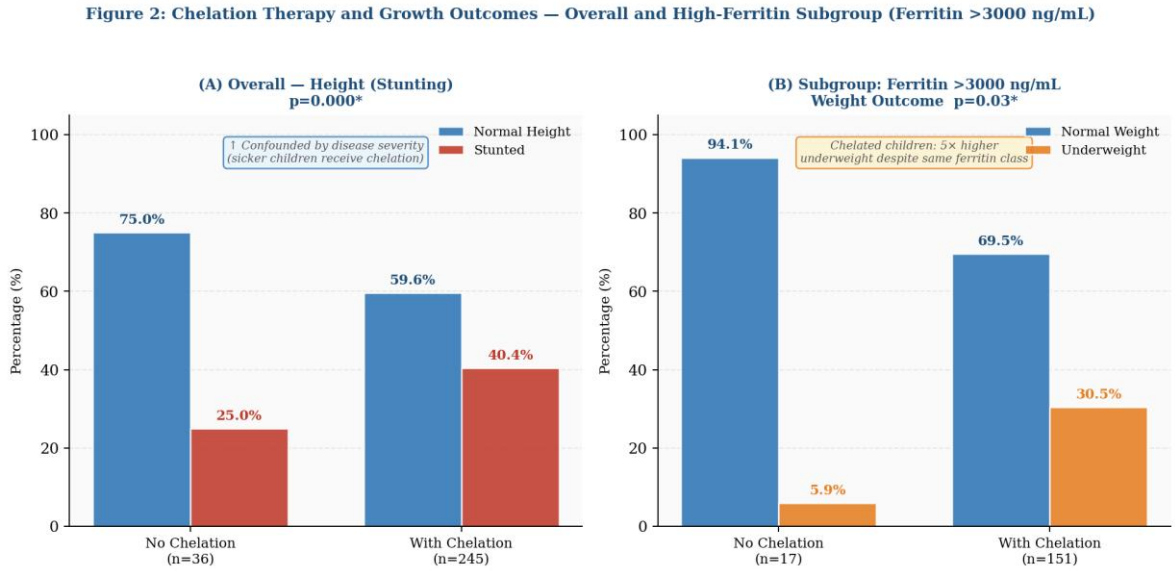

***Figure 2**: Chelation therapy and growth outcomes. (A) Overall population: apparent higher stunting in chelated group, explained by severity confounding. (B) Subgroup with ferritin >3000 ng/mL: among the most iron-overloaded patients, chelated children show 5× higher underweight rates, reflecting the chronicity and severity of disease in this group — not a harmful effect of chelation.*

Multivariable Predictors of Growth Outcomes: Table 4 and Figure 3 present the multivariable logistic regression results. For height: transfusion frequency of twice per month (adjusted OR 0.31; 95% CI 0.15–0.65; p=0.002), age at starting chelation (adjusted OR 1.16; 95% CI 1.05–1.28; p=0.005), and duration of chelation (adjusted OR 1.01; 95% CI 1.00–1.02; p=0.003) were statistically significant independent predictors. Higher transfusion frequency maintaining higher haemoglobin suppresses ineffective erythropoiesis and supports linear growth,[4,5] while later initiation and longer duration of chelation reflect accumulation of iron control over time, which is associated with normal growth trajectory.[16,17,20]

For weight : age at starting chelation (adjusted OR 1.48; 95% CI 1.24–1.78; p<0.001), duration of chelation (adjusted OR 1.04; 95% CI 1.03–1.06; p<0.001), and serum ferritin level (adjusted OR 4.27; 95% CI 1.07–17.0; p=0.040) were independent predictors. The robust and significant association of serum ferritin with underweight — persisting after adjustment for age, chelation status, and transfusion frequency — confirms that iron overload directly impairs weight accrual through endocrine and nutritional mechanisms.[9,10,21] Children with controlled ferritin, achieved through effective chelation, had significantly better weight outcomes, corroborating the clinical importance of achieving ferritin targets.[14,22] Age, sex, age at diagnosis, age at first transfusion, and pretransfusion haemoglobin were not independently associated with growth outcomes after adjustment, suggesting that iron management — rather than demographic factors — is the key modifiable determinant of growth in this population.

Table 4: Multivariable Logistic Regression — Predictors of Stunting and Underweight in Thalassaemia Children (N=281)

Predictor Variable	Unadj. OR (95% CI) [Height]	Adj. OR (95% CI) [Height]	Unadj. OR (95% CI) [Weight]	Adj. OR (95% CI) [Weight]
Age (years), median 8 (5–13)	1.12 (1.06–1.18)	1.07 (0.99–1.16) p=0.084	1.29 (1.19–1.39)	1.10 (0.96–1.26) p=0.173
Transf. freq. 2×/month (ref:1×)	0.48 (0.28–0.85)	0.31 (0.15–0.65) p=0.002*	4.49 (1.54–13.1)	1.55 (0.26–9.12) p=0.630

Predictor Variable	Unadj. OR (95% CI) [Height]	Adj. OR (95% CI) [Height]	Unadj. OR (95% CI) [Weight]	Adj. OR (95% CI) [Weight]
Age at starting chelation (months)	1.19 (1.09–1.29)	1.16 (1.05–1.28) p=0.005*	1.26 (1.15–1.39)	1.48 (1.24–1.78) p<0.001*
Duration of chelation (months)	1.01 (1.00–1.02)	1.01 (1.00–1.02) p=0.003*	1.03 (1.02–1.04)	1.04 (1.03–1.06) p<0.001*
Serum ferritin (continuous)	1.39 (0.90–2.13)	1.29 (0.73–2.29) p=0.382	7.73 (3.01–19.8)	4.27 (1.07–17.0) p=0.040*
Pre-transfusion Hb (g/dL)	0.96 (0.82–1.12)	0.94 (0.78–1.15) p=0.549	1.42 (1.13–1.78)	1.19 (0.86–1.63) p=0.298

**p<0.05 statistically significant. OR = Odds Ratio; CI = Confidence Interval; Adj. = Adjusted. Reference category for transfusion frequency: one transfusion/month. Variables with p≥0.2 in bivariate analysis excluded from adjusted model.*

Figure 3: Multivariable Logistic Regression — Adjusted Odds Ratios for Growth Outcomes in Thalassaemia Children (N=281)

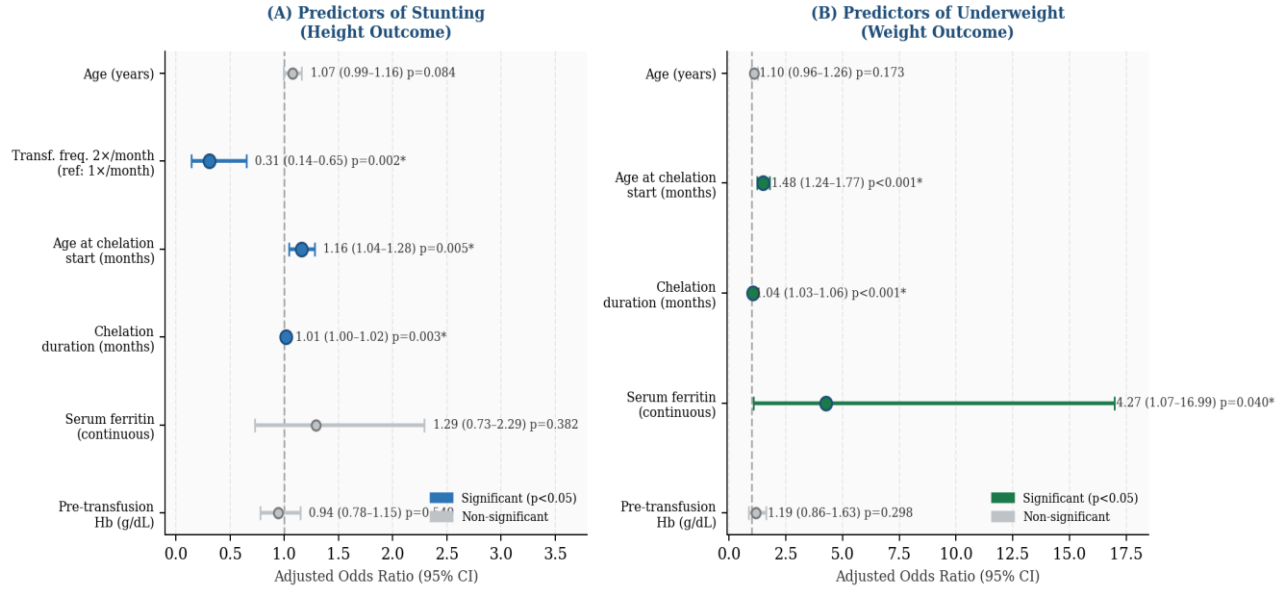

Figure 3: Forest plot of adjusted odds ratios from multivariable logistic regression. (A) Predictors of stunting (height outcome). (B) Predictors of underweight (weight outcome). Filled circles denote statistically significant predictors (p<0.05); horizontal bars represent 95% confidence intervals. The vertical dashed line at OR=1.0 denotes the null.

DISCUSSION

This cross-sectional study of 281 thalassaemic children at a tertiary centre in Rajasthan identified high burden of growth retardation: 38.4% stunted and 18.5% underweight. These findings are consistent with published South Asian data, where stunting prevalence in transfusion-dependent thalassaemia ranges from 30–65%.[11-13] The study's central finding — a significant, dose-response relationship between serum ferritin and growth impairment — reinforces the pathophysiological link between iron overload and growth failure.

Serum ferritin exceeded 3000 ng/mL in nearly 60% of this cohort, reflecting widespread inadequate iron control. Children with ferritin consistently in the 1501–3000 ng/mL range showed substantially better height (83.2% normal) and weight (95.0% normal) profiles compared to those with ferritin >3000 ng/mL. This is biologically plausible: ferritin levels above 2500 ng/mL signal significant haemosiderosis of the anterior pituitary, causing growth hormone deficiency and IGF-1 suppression — the primary endocrine mechanism of thalassaemia-related linear growth failure.[9,10] Iron-mediated hepatic dysfunction, insulin resistance, and gonadal damage additionally impair weight accrual.[21]

The crude comparison of chelation status with growth appeared paradoxical — chelated children had higher stunting rates than unchelated children — but this is a well-documented phenomenon attributable to severity confounding, not a harmful effect of chelation.[16,17] Children receiving chelation are precisely those with greater cumulative iron burden, longer disease duration, and more advanced endocrinopathy. When confounding was addressed through multivariable regression, earlier

initiation of chelation (lower age at start) and longer chelation duration emerged as robust independent predictors of better growth outcomes for both height and weight. These findings resonate with Olivieri and Brittenham's seminal observation[14] that sustained, adequate chelation begun before the development of iron-induced endocrine damage is essential for preserving normal growth.

The subgroup analysis among children with ferritin >3000 ng/mL (n=168) provided clinically important data: chelated children in this high-burden group had significantly higher rates of underweight (30.5% vs 5.9%; p=0.030) and stunting (51.0% vs 11.8%; p=0.002), reflecting that the most iron-overloaded, growth-impaired children are those who have been on chelation the longest — consistent with the hypothesis that late or inadequate chelation predates observable growth failure. This underscores the need for early chelation initiation before irreversible endocrine damage accrues.[9,16,22]

Serum ferritin independently predicted underweight (adjusted OR 4.27; p=0.040) on multivariable analysis, confirming that iron burden directly impairs weight accrual beyond its correlation with disease severity. Adherence to regular chelation, which maintains ferritin in a controlled range, was associated with better growth, corroborating data from the TIF guidelines[14] and Italian thalassaemia follow-up studies.[4,22] Deferasirox, used by 62.9% of chelated children in this cohort, is an orally bioavailable iron chelator with well-established efficacy and safety in paediatric thalassaemia.[15]

Twice-monthly transfusion frequency independently protected against stunting (adjusted OR 0.31; p=0.002), consistent with evidence that hypertransfusion regimens maintaining pretransfusion Hb >9 g/dL suppress ineffective erythropoiesis, reduce marrow expansion, and support linear growth.[4,5] Age, sex, pretransfusion Hb, and age at diagnosis did not independently predict growth outcomes after adjustment, suggesting that iron management protocol — rather than patient demographics — is the primary modifiable determinant of growth in this population.

Limitations include the cross-sectional design precluding causal inference and longitudinal growth tracking; hospital-based sampling limiting generalisability; ferritin's imprecision as an iron marker (influenced by inflammation and hepatic dysfunction); and absence of growth hormone, IGF-1, and thyroid function assays that would mechanistically link iron overload to growth retardation.

CONCLUSION

Iron overload — quantified by serum ferritin — is significantly and independently associated with stunting and underweight in children with transfusion-dependent thalassaemia. Children with well-controlled ferritin levels (particularly in the 1501–3000 ng/mL range), achieved through effective chelation therapy, demonstrate substantially better growth profiles. Earlier chelation initiation, longer chelation duration, and serum ferritin control are the strongest modifiable predictors of favourable growth outcomes. Adequate transfusion frequency additionally supports linear growth. These findings call for universal early chelation implementation, strict adherence monitoring, regular ferritin surveillance, and optimised transfusion protocols as core components of comprehensive thalassaemia care — with the goal of preserving normal growth and improving quality of life in this vulnerable paediatric population.

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