Original Research Paper

Volume - 16 | Issue - 07 | July - 2026 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

General Medicine

RELATIONSHIP BETWEEN SERUM INSULIN-LIKE GROWTH FACTOR-1 AND SERUM FERRITIN IN PATIENTS WITH BETA-THALASSEMIA MAJOR: A HOSPITAL-BASED CASE-CONTROL STUDY

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ABSTRACT **Background and Objectives:** Iron overload from repeated blood transfusions is a recognized cause of hypothalamic-pituitary dysfunction in beta-thalassemia major (BTM), manifesting as growth hormone (GH) deficiency and reduced insulin-like growth factor-1 (IGF-1) levels. The present study aimed to compare serum IGF-1 levels between transfusion-dependent BTM patients and healthy controls, and to evaluate the relationship between serum IGF-1 and serum ferritin in cases. **Methods:** This hospital-based case-control study enrolled 60 confirmed BTM patients (cases) and 60 age- and sex-matched healthy individuals (controls) at the Department of Medicine, Assam Medical College and Hospital, Dibrugarh. Serum IGF-1 and serum ferritin were measured in all participants. Anthropometric parameters (height, weight, BMI) and body composition by DEXA scan (lean body mass, body fat percentage) were also assessed. Statistical analysis included independent samples t-test and Pearson correlation (significance at $p < 0.05$). **Results:** Mean serum IGF-1 was significantly lower in cases (114.94 ± 56.64 ng/mL) than controls (349.53 ± 192.69 ng/mL; $p < 0.001$). Mean serum ferritin was markedly elevated in cases (1660.47 ± 810.28 ng/mL) versus controls (117.97 ± 24.88 ng/mL; $p < 0.001$). In the case group, IGF-1 demonstrated a weak negative correlation with serum ferritin that did not reach statistical significance ($r = -0.109$, $p = 0.407$). Significant positive correlations were observed between IGF-1 and lean body mass ($r = 0.491$, $p < 0.001$) and body fat percentage ($r = 0.441$, $p < 0.001$) in cases. Thalassemia patients exhibited significantly lower weight, BMI, lean body mass, and body fat percentage compared to controls (all $p < 0.001$). **Conclusions:** BTM patients demonstrate profound IGF-1 deficiency with significant iron overload, reflecting GH-IGF-1 axis dysfunction. Although a direct statistically significant inverse correlation between serum ferritin and IGF-1 was not established, the co-existence of severe iron overload and reduced IGF-1 is consistent with iron-mediated pituitary somatotroph injury. IGF-1 levels are significantly associated with body composition parameters in thalassemia, underscoring the anabolic importance of this axis. Annual endocrine screening including serum IGF-1 is recommended for all transfusion-dependent thalassemia patients.

KEYWORDS : Beta-thalassemia Major; IGF-1; Serum Ferritin; Iron Overload; Growth Hormone; Body Composition; DEXA

1. INTRODUCTION

Beta-thalassemia major (BTM), also known as Cooley's anemia, is the most severe form of the beta-thalassemia syndromes, resulting from homozygous or compound heterozygous mutations in the HBB gene that lead to absent or markedly reduced beta-globin chain synthesis. The resulting severe hemolytic anemia mandates lifelong regular packed red cell transfusions, typically every two to four weeks, to maintain adequate hemoglobin levels and suppress pathological erythropoiesis. Despite improved survival attributable to modern transfusion protocols and oral iron chelation therapy, the chronic transfusional iron burden predisposes patients to multi-organ siderosis and a broad spectrum of endocrine complications.

Among the most clinically consequential but often underrecognized endocrine complications is dysfunction of the growth hormone (GH)–insulin-like growth factor-1 (IGF-1) axis. Iron deposition in the somatotroph cells of the anterior pituitary impairs GH secretory capacity, while concurrent hepatic siderosis reduces the hepatic synthesis and secretion of IGF-1. The net effect is a state of GH resistance and IGF-1 deficiency that manifests clinically as growth retardation, delayed puberty, and altered body composition—characterized by reductions in lean body mass and fat mass. These abnormalities are particularly pronounced during adolescence, when GH pulsatility and IGF-1 levels are critically required for the pubertal growth spurt and accrual of peak bone mass.

Serum IGF-1 has emerged as a practical, clinically accessible surrogate for assessing GH axis integrity. Because direct GH stimulation tests (insulin tolerance test, arginine test) are logistically demanding and carry procedural risk, measurement of basal serum IGF-1 provides a useful screening parameter, with levels below the age- and sex-specific reference range suggesting GH deficiency or resistance. Conversely, serum ferritin—though an acute phase reactant and thus imperfect—remains the most widely available surrogate marker of total body iron stores in resource-limited settings, and elevated ferritin levels have been associated with increased endocrine morbidity in transfusion-dependent thalassemia.

The specific relationship between the degree of iron overload as reflected by serum ferritin and the degree of GH-IGF-1 axis suppression in BTM patients has been explored in several international studies but remains incompletely characterized in the Indian subcontinent context, where genetic mutation spectra, nutritional background, and chelation compliance patterns differ from Western cohorts. The present study was therefore designed to compare serum IGF-1 levels between BTM patients and healthy age- and sex-matched controls at a tertiary care centre in Northeast India, to evaluate the correlation between serum ferritin and serum IGF-1 in cases, and to assess the relationship between IGF-1 and body composition parameters as measured by dual-energy X-ray absorptiometry (DEXA).

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This was a hospital-based, cross-sectional case-control study conducted at the Department of Medicine, Assam Medical College and Hospital (AMCH), Dibrugarh, Assam, India—a tertiary referral center serving the northeastern region of India—over a period of one year.

2.2 Study Population

Cases comprised 60 patients with a confirmed diagnosis of beta-thalassemia major who were registered in the thalassemia day-care unit of AMCH, meeting the following criteria: age ≥ 13 years, documented diagnosis of BTM established by hemoglobin electrophoresis (CE-HPLC), transfusion dependence (regular transfusions since childhood), and ongoing iron chelation therapy with deferasirox.

Controls were 60 healthy volunteers matched to cases on age (±2 years) and sex. Exclusion criteria for both groups included chronic liver disease, chronic kidney disease, diabetes mellitus, active infections, malignancies, thyroid disorders, ongoing glucocorticoid therapy, pregnancy, and any other condition known to independently alter IGF-1 metabolism. Written informed consent was obtained from all participants (or their guardians for minors). The study was approved by the Institutional Ethics Committee of AMCH.

2.3 Clinical and Anthropometric Assessment

Detailed clinical history including transfusion frequency (number of transfusions per year), duration and type of chelation therapy, and

number of hospitalizations was recorded. Anthropometric measurements—height (to the nearest 0.1 cm), weight (to the nearest 0.1 kg), and body mass index (BMI = weight/height² in kg/m²)—were obtained using standardized techniques.

2.4 Laboratory Investigations

Fasting venous blood samples were collected from all participants. Serum IGF-1 was measured by enzyme-linked immunosorbent assay (ELISA) using commercially available kits. Age- and sex-specific reference intervals were applied for interpretation of IGF-1 results. Iron profile—serum iron (µg/dL), serum ferritin (ng/mL), total iron binding capacity (TIBC; µg/dL), and transferrin saturation (%)—was assessed using automated biochemical analyzers. Complete blood count and liver function tests were obtained to exclude confounding hepatic disease.

2.5 Body Composition Assessment

Body composition was assessed by dual-energy X-ray absorptiometry (DEXA scan) in all participants. Parameters recorded included lean body mass (LBM; kg), lean body mass index (LBMI; kg/m²), and body fat percentage.

2.6 Statistical Analysis

Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Continuous variables are expressed as mean ± standard deviation (SD). Between-group comparisons were made using the independent samples Student's t-test for normally distributed variables and the Mann-Whitney U test for non-normal distributions. Categorical variables were compared using Fisher's exact test. Pearson's correlation coefficient (r) was used to assess the linear relationship between serum IGF-1 and serum ferritin (primary objective), and between IGF-1 and body composition parameters. A p-value < 0.05 was considered statistically significant.

3. RESULTS

3.1 Baseline Characteristics

The study enrolled 60 BTM cases (27 males, 33 females) and 60 controls (29 males, 31 females). The male-to-female ratio was 0.82:1 in cases and 0.94:1 in controls. The mean age of cases was 24.93 ± 13.67 years (range: 13–61 years), and 29.40 ± 15.06 years in controls (range: 13–65 years; p = 0.091). Age and sex distributions were comparable between groups, confirming adequate matching.

All 60 cases were transfusion-dependent with a documented history of regular packed red cell transfusions; 56.7% received more than 10 transfusions annually, and 43.3% received 1–10 transfusions per year. All cases were on deferasirox as oral chelation therapy. Regarding hospitalization burden, 56.7% had been admitted more than 10 times, 40.0% between 6–10 times, and only 3.3% had ≤5 admissions.

Table 1. Baseline Demographic and Anthropometric Characteristics

Parameter	Cases (n=60) Mean ± SD	Controls (n=60) Mean ± SD	p value	Signi-ficance
Age (years)	24.93 ± 13.67	29.40 ± 15.06	0.091	NS
Height (cm)	154.10 ± 12.24	161.84 ± 6.43	< 0.001	***
Weight (kg)	40.25 ± 8.83	59.49 ± 7.76	< 0.001	***
BMI (kg/m²)	16.76 ± 2.28	22.68 ± 2.32	< 0.001	***
Males/Females	27/33	29/31	0.717	NS

*NS = not significant; *** p < 0.001; SD = standard deviation; BMI = body mass index*

3.2 Iron Profile

Iron profile parameters were markedly deranged in BTM cases compared to controls (Table 2). Mean serum ferritin was 1660.47 ± 810.28 ng/mL in cases, versus 117.97 ± 24.88 ng/mL in controls (p < 0.001)—a greater than 14-fold elevation. All 60 cases had serum ferritin above the normal threshold: 23.3% had mild iron overload (300–999 ng/mL), 58.3% had moderate overload (1000–2499 ng/mL), and 18.3% had severe overload (≥2500 ng/mL). Serum iron, transferrin saturation, and TIBC also differed significantly between groups.

Table 2. Comparison of Iron Profile Parameters Between Cases and Controls

Iron Profile Parameter	Cases (n=60) Mean ± SD	Controls (n=60) Mean ± SD	p value	Signi-ficance
Serum Iron (µg/dL)	155.52 ± 29.10	112.69 ± 18.94	< 0.001	***
Serum Ferritin (ng/mL)	1660.47 ± 810.28	117.97 ± 24.88	< 0.001	***
TIBC (µg/dL)	226.43 ± 12.37	291.80 ± 53.94	< 0.001	***
Transferrin Saturation (%)	68.52 ± 13.95	38.47 ± 7.96	< 0.001	***

*TIBC = total iron binding capacity; *** p < 0.001*

3.3 Serum IGF-1 Levels

Mean serum IGF-1 was profoundly reduced in BTM cases compared to controls: 114.94 ± 56.64 ng/mL versus 349.53 ± 192.69 ng/mL respectively (p < 0.001; Table 3). The median IGF-1 in cases was 106.63 ng/mL (range: 15.63–261.42 ng/mL) compared to 300.46 ng/mL (range: 81.76–825.09 ng/mL) in controls. The difference was clinically substantial, with cases having approximately one-third the IGF-1 concentration of age- and sex-matched healthy individuals.

When stratified by age group in cases, low IGF-1 was predominantly observed among adolescents (11–15 years: 45.8%; 16–20 years: 45.8%), while normal IGF-1 was most frequent in adults aged 21–50 years (71.9%). Elevated IGF-1 was seen exclusively in older adults (51–70 years), possibly reflecting compensatory or reactive hormonal changes in this subgroup.

Table 3. Comparison of Serum IGF-1 and Body Composition Between Cases and Controls

Parameter	Cases (n=60) Mean ± SD	Controls (n=60) Mean ± SD	p value	Signi-ficance
Serum IGF-1 (ng/mL)	114.94 ± 56.64	349.53 ± 192.69	< 0.001	***
Lean Body Mass (kg)	31.07 ± 6.90	39.63 ± 8.02	< 0.001	***
Body Fat (%)	14.48 ± 3.90	34.44 ± 6.48	< 0.001	***
LBMI (kg/m²)	12.94 ± 1.86	15.12 ± 2.74	< 0.001	***

*IGF-1 = insulin-like growth factor-1; LBMI = lean body mass index; *** p < 0.001*

3.4 Correlation Between Serum IGF-1 and Serum Ferritin

In the case group, serum ferritin demonstrated a weak negative correlation with serum IGF-1 (r = −0.109, p = 0.407), which did not achieve statistical significance at the 5% level (Table 4). Scatter plot analysis revealed a non-linear distribution of data points, with wide variability in IGF-1 across all ranges of ferritin, suggesting that the relationship between iron overload and IGF-1 suppression is multifactorial and not directly captured by a simple linear correlation.

In the control group, a weakly positive but statistically significant correlation was observed between serum ferritin and IGF-1 (r = 0.316, p = 0.013), consistent with the normal physiological relationship between nutritional iron status and anabolic hormone milieu in healthy individuals.

Table 4. Pearson Correlation Coefficients for Serum IGF-1

Variable	Group	n	r value	p value
IGF-1 vs Serum Ferritin	Cases	60	−0.109	0.407 (NS)
IGF-1 vs Serum Ferritin	Controls	60	+0.316	0.013*
IGF-1 vs Lean Body Mass	Cases	60	+0.491	< 0.001***
IGF-1 vs Body Fat (%)	Cases	60	+0.441	< 0.001***

*NS = not significant; * p < 0.05; *** p < 0.001. Pearson correlation coefficient.*

3.5 Correlations Between IGF-1 and Body Composition

In the case group, serum IGF-1 showed a moderate positive correlation with lean body mass (r = 0.491, p < 0.001), indicating that higher IGF-1 levels were associated with greater muscle mass. Similarly, a moderate positive correlation was noted between IGF-1 and body fat percentage (r = 0.441, p < 0.001). These findings suggest that IGF-1 is an important determinant of both lean and adipose tissue accrual in BTM patients. In contrast, neither correlation was significant in the control group (LBM: r = 0.055, p = 0.388; body fat: r = 0.083, p = 0.264), consistent with the broader GH-IGF-1 regulatory dynamics in healthy individuals where body composition is governed by multiple overlapping hormonal and metabolic axes.

4. DISCUSSION

The present study demonstrates that transfusion-dependent BTM patients have markedly impaired GH-IGF-1 axis function, evidenced

by profoundly reduced serum IGF-1 levels compared to age- and sex-matched healthy controls. This finding is consistent with an extensive body of international literature. Soliman et al. documented significantly reduced IGF-1 in thalassemia patients, particularly in pubertal and adolescent cohorts, which they attributed to GH deficiency and hepatic iron deposition impairing local IGF-1 synthesis. Scacchi and colleagues similarly demonstrated low IGF-1 in adult thalassemia patients, correlating with impaired GH secretory dynamics on formal stimulation testing. The 2010 study by Poggi et al. specifically established a high prevalence of GHD in adults with BTM receiving regular transfusions, with low IGF-1 as a prominent biochemical feature.

In our cohort, the mean serum ferritin in cases was 1660.47 ± 810.28 ng/mL, with nearly one-fifth of patients exhibiting severe iron overload (ferritin ≥ 2500 ng/mL). Despite this degree of iron loading, the Pearson correlation between serum ferritin and serum IGF-1 in cases was weak and non-significant ($r = -0.109$, $p = 0.407$). This absence of a statistically significant direct linear correlation is clinically informative and biologically plausible. The pathophysiology of iron-mediated GH deficiency is not simply a dose-response relationship between circulating ferritin and IGF-1; rather, it reflects the cumulative burden of tissue iron deposition—particularly in the pituitary gland and liver—which is incompletely captured by serum ferritin alone. Serum ferritin is an acute phase reactant that overestimates iron stores during inflammatory states, and its relationship with organ-specific iron deposition (as measured by MRI-based liver iron concentration or cardiac T2* imaging) is imperfect. This methodological limitation is well-recognized in the thalassemia literature.

Additionally, IGF-1 suppression in thalassemia is not exclusively mediated through iron toxicity to somatotrophs. Contributory mechanisms include chronic hypoxia from anemia impairing hepatic IGF-1 secretion, malnutrition reducing hepatic protein synthesis, delayed puberty attenuating the estrogen-stimulated amplification of GH pulsatility, and direct iron toxicity to hepatocytes reducing local IGF-1 production. The multifactorial nature of IGF-1 suppression means that serum ferritin alone—even if substantially elevated—does not uniquely predict the degree of IGF-1 deficiency. Similar findings have been reported by other investigators: De Sanctis et al. noted that the relationship between ferritin and endocrine dysfunction in thalassemia is modulated by chelation efficacy, baseline pituitary reserve, and genetic factors. Prospective cohort studies with MRI-based quantification of pituitary iron and pituitary volume assessments would be better positioned to delineate this mechanistic link.

The significant positive correlations observed between serum IGF-1 and lean body mass ($r = 0.491$, $p < 0.001$) and body fat percentage ($r = 0.441$, $p < 0.001$) in BTM cases are physiologically coherent and clinically meaningful. IGF-1 exerts well-characterized anabolic effects on skeletal muscle through stimulation of protein synthesis, satellite cell activation, and inhibition of muscle proteolysis. The positive IGF-1–lean mass relationship in our case group mirrors findings by Soliman et al. in 2014, who reported that IGF-1 deficiency was associated with decreased muscle mass and impaired body composition in thalassemia, and by Peter Wong et al. in 2014, who showed that lean body mass and fat mass were positively correlated with endocrine and metabolic factors in transfusion-dependent thalassemia. The clinical implication is that BTM patients with lower IGF-1 levels are at compounded risk for sarcopenia and functional impairment, both of which adversely affect quality of life and potentially increase infection risk during intercurrent illness.

The anthropometric data from our study reinforce the systemic impact of GH-IGF-1 axis dysfunction in BTM. Mean BMI in cases was 16.76 ± 2.28 kg/m², placing the average case patient firmly in the underweight range, with a calculated z-score of approximately −2.55 against WHO/IAP growth standards—below the 3rd percentile. Mean weight deficit of approximately 19 kg compared to controls and mean lean body mass approximately 8.5 kg below the control group reflect the combined effects of chronic anemia, endocrine dysfunction, iron overload, and nutritional deficiencies in this population.

From a clinical management standpoint, these findings support international and Indian guidelines recommending annual endocrine screening for all transfusion-dependent thalassemia patients from adolescence onward. The Thalassemia International Federation (TIF) 2014 guidelines specifically recommend annual assessment of growth parameters, pubertal staging, and basal IGF-1/IGFBP-3 levels as first-line screening for GH axis dysfunction. Formal GH stimulation testing (insulin tolerance test or arginine test) remains the gold standard for diagnosis of GHD and should be pursued in patients with low IGF-1 before initiating recombinant human growth hormone (rhGH) therapy. The Endocrine Society recommends rhGH replacement at 0.17–0.33 mg/kg/week in confirmed GHD when there are no contraindications. Optimization of iron chelation therapy, which is the most impactful preventive strategy, must accompany any hormonal assessment.

Several limitations of the present study merit discussion. First, direct GH stimulation testing was not performed; hence, GH deficiency is inferred through IGF-1 levels rather than formally established. Second, MRI-based organ iron quantification (liver iron concentration, cardiac T2*, pituitary volume and signal characteristics) was not available, preventing a more mechanistically refined analysis of the ferritin-IGF-1 relationship. Third, the cross-sectional design precludes causal inference; longitudinal data tracking changes in ferritin and IGF-1 over time would be more informative. Fourth, the study was conducted at a single centre in Northeast India, and findings may not be fully generalizable to other thalassemia populations. Fifth, nutritional assessment including micronutrient status (zinc, vitamin D) was not systematically performed, which could confound both IGF-1 levels and body composition parameters.

5. CONCLUSION

This study demonstrates that transfusion-dependent beta-thalassemia major is associated with profound IGF-1 deficiency, with mean serum IGF-1 approximately one-third of age- and sex-matched healthy controls. Serum ferritin is markedly elevated in BTM patients but does not show a statistically significant direct linear inverse correlation with serum IGF-1. This finding does not negate the biological role of iron overload in suppressing GH-IGF-1 axis function; rather, it highlights that serum ferritin is an incomplete surrogate for organ-level siderosis, and IGF-1 suppression in thalassemia is multifactorial. Serum IGF-1 is significantly and positively correlated with lean body mass and body fat percentage in BTM cases, emphasizing the important anabolic role of this axis in governing body composition in these patients.

Annual screening of IGF-1 levels, particularly in adolescent and young adult thalassemia patients, should be integrated into routine clinical follow-up. Future studies incorporating MRI-based iron quantification, GH stimulation testing, pituitary imaging, and longitudinal follow-up designs are required to fully elucidate the relationship between iron organ deposition and GH-IGF-1 axis suppression, and to evaluate the impact of rhGH therapy on growth and body composition outcomes in Indian thalassemia patients.

DECLARATIONS

Ethics Approval and Consent to Participate: The study was approved by the Institutional Ethics Committee of Assam Medical College and Hospital, Dibrugarh. Written informed consent was obtained from all participants (or their legal guardians for minors).

Conflict of Interest: The authors declare no conflicts of interest.

Funding: No external funding was received for this study.

Author Contributions: AB: concept, data collection, analysis, manuscript drafting. AD: study supervision, critical revision. MD: biochemical investigation design, critical revision. All authors read and approved the final manuscript.

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