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Original Research Article

Comparison of Thyroid Profile in Beta-Thalassemia Major Patients on Regular Blood Transfusion and Iron Chelation Therapy with Age-Matched Controls: A Cross-Sectional Study

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Abstract

Background: Beta-thalassemia major (BTM) is a transfusion-dependent hereditary hemoglobin disorder associated with iron overload and multiple endocrine complications, including thyroid dysfunction. Early detection of thyroid abnormalities is essential to reduce morbidity in these patients.

Objective: To evaluate the prevalence and pattern of thyroid dysfunction in children with beta-thalassemia major receiving regular blood transfusion and iron chelation therapy, and to compare findings with age-matched healthy controls.

Methods: This hospital-based cross-sectional comparative study included 50 children (≥8 years) with confirmed BTM and 50 age-matched healthy controls. Clinical evaluation, anthropometric measurements, and laboratory investigations including serum T3, T4, thyroid-stimulating hormone (TSH), and serum ferritin were performed. Thyroid status was classified as euthyroid, subclinical hypothyroidism, primary hypothyroidism, or secondary hypothyroidism. Statistical analysis was conducted using SPSS version 21.0.

Results: The mean age of cases and controls was comparable. Thalassemia patients had significantly lower mean height and weight than controls. Overall, 26% of BTM patients exhibited thyroid dysfunction, with subclinical hypothyroidism being most common (20%), followed by primary hypothyroidism (6%). In contrast, 12% of controls had subclinical hypothyroidism, with no cases of overt hypothyroidism. Although mean T3, T4, and TSH levels did not differ significantly between groups, elevated TSH was associated with thyroid dysfunction. Higher serum ferritin levels and longer duration of chelation therapy were significantly associated with thyroid abnormalities. No significant association was found with age at diagnosis, transfusion frequency, or transfusion burden.

Conclusion: Thyroid dysfunction, predominantly subclinical hypothyroidism, is a common endocrine complication in transfusion-dependent beta-thalassemia major patients despite ongoing chelation therapy. Regular screening of thyroid function and monitoring of iron overload are recommended for early detection and management.

Keywords: Beta-thalassemia major, Iron overload, Thyroid dysfunction, Subclinical hypothyroidism, Serum ferritin.

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Introduction

Thalassemia is the most common monogenic disorder worldwide, affecting millions of individuals predominantly in the Mediterranean basin, Middle East, Indian subcontinent, and Southeast Asia [1][2]. Beta-thalassemia major (BTM), the severe form of β-thalassemia, results from mutations in the β-globin gene leading to reduced or absent synthesis of β-globin chains, causing severe hemolytic anemia, ineffective erythropoiesis, and extramedullary hematopoiesis

[1]. The global incidence of thalassemia major is approximately 100,000 new cases annually, with approximately 10,000 cases born in India alone [3]. Despite recent therapeutic advances, thalassemia major remains a significant public health burden in endemic regions [1].

Beta-thalassemia major typically manifests between 4-6 months of age as hemoglobin F (fetal hemoglobin) levels decline during infancy [2][3]. Historically considered fatal before the second decade of life, survival rates have improved dramatically with modern management strategies [3]. Regular blood transfusions and iron chelation therapy have extended median life expectancy significantly [4]. However, these life-sustaining interventions result in serious secondary complications, particularly transfusion-related iron overload [5].

The human body lacks a physiological mechanism for iron excretion. Chronic blood transfusions result in progressive iron accumulation, with each unit of transfused packed cells containing approximately 250 mg of iron [6]. This leads to severe iron overload (secondary hemosiderosis), which deposits in virtually all organs including the myocardium, liver, pancreas, and endocrine glands [6-8]. Iron deposition in endocrine tissues causes hemosiderosis, resulting in multiple endocrinopathies including hypogonadism, diabetes mellitus, hypothyroidism, and hypoparathyroidism [9]. Thyroid dysfunction represents one of the most common endocrine complications in chronically transfused thalassemia patients. Primary hypothyroidism is the predominant form of thyroid dysfunction observed in thalassemia [10]. The frequency of thyroid dysfunction demonstrates significant geographic and management-related variation, with reported prevalence ranging from 13-60% in thalassemia patients after 10 years of age [11]. Most cases present as subclinical hypothyroidism (compensated hypothyroidism with elevated TSH and normal T4) [11].

Recent cross-sectional studies demonstrate subclinical hypothyroidism in 21.8% to 32% of thalassemia patients, primary hypothyroidism in 6.9% to 12%, with clinical hypothyroidism requiring treatment occurring in approximately 5% of patients [5][6]. The progression from euthyroidism to subclinical and clinical hypothyroidism reflects progressive iron deposition in thyroid parenchyma [8].

Despite extensive literature on endocrinopathies in thalassemia, thyroid dysfunction remains inadequately managed and monitored in many resource-limited settings. The specific interplay between iron overload, chelation therapy, transfusion burden, and thyroid function requires investigation in diverse populations with varying genetic backgrounds, transfusion protocols, and chelation regimens.

The present study aims to systematically characterize thyroid dysfunction in a cohort of transfusion-dependent BTM patients managed with modern chelation therapy and to correlate thyroid parameters with clinical, biochemical, and demographic variables. Understanding these relationships can optimize screening protocols, refine therapeutic strategies, and improve quality of life in this chronically ill population.

Research Question: What is the prevalence and pattern of thyroid gland dysfunction in beta-thalassemia major children aged ≥8 years maintained on regular blood transfusion and iron chelation therapy compared to healthy age-matched controls?

Material and Methods

Study Design and Setting: This was a hospital-based cross-sectional comparative study conducted at the Department of Pediatrics, Dr. Baba Saheb Ambedkar Medical College and Hospital, Rohini, New Delhi from November 2016 to June 2017 (8-month study period). The study was approved by the Institutional Ethics Committee prior to commencement.

Study Population

Case Group (Thalassemia Patients): The case group comprised 50 confirmed cases of beta-thalassemia major selected from patients registered in the Thalassemia Clinic and receiving inpatient management for routine blood transfusion at the Department of Pediatric Medicine. Inclusion criteria were:

- Age ≥8 years
- Confirmed diagnosis of homozygous beta-thalassemia based on:
    - Clinical presentation and hematological findings
    - Peripheral blood smear examination
    - Hemoglobin electrophoresis demonstrating predominantly hemoglobin A2 and fetal hemoglobin with absent or markedly reduced hemoglobin A
- Regular blood transfusion (at minimum monthly transfusions)
- Active participation in iron chelation therapy
- Parental written informed consent

Exclusion criteria were:

- Absence of parental consent
- History of hereditary growth disorders
- Major congenital anomalies
- Active acute or chronic infections (HIV, hepatitis B, hepatitis C)

- Acute or chronic liver disease (beyond hepatomegaly secondary to iron overload)
- Obvious malnutrition (BMI <-3 SD by WHO standards)
- Chronic corticosteroid use or other medications affecting growth and thyroid function

Control Group

The control group consisted of 50 healthy subjects age-matched (±2 years) to the case group. Control subjects were recruited from:

- Outpatient Department of Pediatrics attending for minor unrelated illnesses
- Healthy siblings of thalassemia patients to reduce selection bias

Inclusion criteria for controls:

- Age 8-18 years
- Normal growth and development
- No clinical or historical features suggestive of thyroid disease
- No chronic illness or medication use
- Absence of hepatosplenomegaly or other systemic findings
- Parental consent

All controls were clinically examined to exclude thyroid dysfunction before inclusion.

Methodology

Informed Consent and Data Collection: Written informed consent was obtained from parents or legal guardians of all study subjects after detailed explanation of the study purpose and procedures. A structured pro forma captured the following information:

For Thalassemia Cases:

- Personal details: name, age, sex, address
- Disease characteristics: age at diagnosis, mode of diagnosis
- Transfusion details: average monthly frequency, annual transfusion requirement, compliance with transfusion protocol
- Chelation therapy: type of chelator (Deferiprone, Deferasirox, combination), duration of therapy, compliance
- Family history and pedigree information
- Complications: cardiac status, endocrine symptoms, hepatic complications

For Controls:

- Personal details
- General medical and family history
- Medication history

Physical Examination and Anthropometry

Height Measurement: Standing height was measured without shoes using a Harpenden stadiometer by a single trained observer to ensure reproducibility. Measurements were recorded to the nearest 0.1 cm.

Weight Measurement: Body weight was measured using a calibrated digital weighing machine (Capri Electronics, Mumbai, India, and Model MI 120T) with subjects in light clothing and without shoes. Measurements were recorded to the nearest 0.1 kg.

BMI Calculation: BMI was calculated using the standard formula: BMI = weight (kg) / height² (m^2).

Anthropometric Assessment: Height, weight, and BMI were compared with age and sex-specific WHO growth reference standards. Height-for-age, weight-for-age, and BMI-for-age were expressed as Z-scores relative to WHO reference medians.

Systemic Examination: All the patients were examined for Hepatomegaly, Splenomegaly, and Auscultation for cardiac murmurs and signs of cardiac compromise, Screening for neurological signs, Assessment for goiter, clinical features of hypo/hyperthyroidism and findings recorded.

Laboratory Analysis

Blood Collection and Processing:

From thalassemia cases:

- 3 ml blood in plain vial (for serum ferritin, T3, T4, TSH)
- 2 ml blood in EDTA vial (for hemoglobin estimation)

From controls:

- 2 ml blood in plain vial (for thyroid profile only)

Thyroid Hormone Assays: All hormonal estimations were performed using immune-enzymatic colorimetric ELISA methods with standardized kits from Meril Diagnostics (Vapi, India):

Free T3 (FT3): Meril FT3 ELISA Kit (Code: ITTELI-01), measured at 450 nm.

- Reference range: 55-180 ng/dl

Free T4 (FT4): Meril FT4 ELISA Kit (Code: ITFELI-01), measured at 450 nm

- Reference range: 5-14.3 µg/dl (laboratory-specific range: 5.0-14.0)

Thyroid Stimulating Hormone (TSH): Merilisa TSH ELISA Kit (REF: TSIELI-01), measured at 450-630 nm

- Reference range: 0.5-4.5 mIU/L (laboratory-specific normal: 0.5-4.0 mIU/L)

Serum Ferritin Assay: Serum ferritin levels were measured using Fortress Diagnostics Ferritin

ELISA Kit (BXEO039A), measured at 450 nm and 620-630 nm with microplate reader (Robonic, India).

- Reference range: <300 ng/ml (values in thalassemia patients typically range 1,000-8,000 ng/ml)

Hemoglobin Estimation:

Hemoglobin was estimated on the same day of collection from EDTA sample using automated hematology analyzer.

- Reference range: 12-16 g/dl for normal children

Classification of Thyroid Status:

Based on combination of T4 and TSH values, subjects were classified as:

1. **Euthyroidism:** Normal T4 (5.0-14.0 µg/dl) + Normal TSH (0.5-4.0 mIU/L)
2. **Primary Hypothyroidism:** Low T4 (<5.0 µg/dl) + High TSH (>4.0 mIU/L)
3. **Subclinical Hypothyroidism:** Normal T4 (5.0-14.0 µg/dl) + High TSH (>4.0 mIU/L)
4. **Secondary/Central Hypothyroidism:** Low T4 + Low/Normal TSH

Statistical Analysis: All data were entered into a Microsoft Excel spreadsheet with double-entry verification, and statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) version 21.0 (IBM Corporation, USA).

Categorical variables were presented as number and percentage (%), while continuous variables were expressed as mean ± standard deviation (SD) and median with interquartile range. Normality of data distribution was assessed using the Kolmogorov–Smirnov test. For analytical statistics, comparison of quantitative variables between groups was performed using the unpaired t-test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. Qualitative variables were compared using the Chi-square (χ^2) test or Fisher's exact test when the expected cell frequency was less than 5.

Correlation analysis was conducted using Spearman's rank correlation coefficient to assess the association between serum ferritin and anthropometric parameters (BMI, height, and weight). A p-value of <0.05 was considered statistically significant, and 95% confidence intervals were calculated where appropriate.

Results

A total of 100 subjects were enrolled: 50 thalassemia major patients (cases) and 50 healthy controls. All subjects completed the study with complete data collection. The mean age of cases was 11.45 ± 3.65 years, and that of controls was 10.78 ± 2.42 years, with no statistically significant difference. [Table no. 1] The sex distribution was also comparable between the two groups, with 34% of the cases were female while 48% were female in control group, and rest were male. [Figure no. 1] Anthropometric assessment showed that thalassemia cases had significantly lower mean weight and height than controls. [Table no. 2]

However, BMI did not differ significantly between the two groups. [Figure no. 2] On WHO-based growth assessment, a larger proportion of cases fell below the expected height-for-age reference range. [Figure no. 3] Thyroid function testing showed that the mean T3, T4, and TSH levels were not significantly different between cases and controls. [Table no. 3] However, when thyroid status was categorized, 74% of thalassemia patients had euthyroidism, 20% had subclinical hypothyroidism, and 6% had primary hypothyroidism, whereas 88% of controls were euthyroid and 12% had subclinical hypothyroidism. [Table no. 4] No case of secondary hypothyroidism was detected in either group.

Table 1: Age of cases and controls

	Case	Control	P value
Age (In Years)			0.798
Sample size	50	50	
Mean ± Std. dev.	11.45 ± 3.65	10.78 ± 2.42	
Median	10	10.5	
Min-Max	8-24	8-18	
Inter quartile Range	9 – 13	9 – 12	

Table 2: Comparison of Weight and Height between cases and controls

	Cases	Controls	Significance
No. of subjects	50	50	
Weight in kg (mean) (± SD)	26.12 ± 8.62	30.56 ± 9.47	p-value = 0.002
Weight range	10.2 - 50.2	16.86 – 56.4	
Height in cm. (mean) (± SD)	131.24 ± 13.79	139.11 ± 12.32	p-value = 0.003
Height Range (in cm.)	103.9 - 160.2	112.5 – 167	
BMI (mean) (± SD)	15.07 ± 2.29	15.58 ± 2.6	p-value = 0.461
BMI range	11.7 - 22.32	10.9 - 21.5	

Table 3: Comparison of Thyroid profile between cases and controls

Hormone / Parameter	Case Group (Mean ± SD)	Control Group (Mean ± SD)	Range (Case)	Range (Control)	P-value
T3 (ng/dl)	125.94 ± 26.95	119.98 ± 38.06	70 – 173	55 – 180	0.369
T4 (µg/dl)	9.42 ± 2.83	10.08 ± 2.35	3 – 14	5 – 14.3	0.467
TSH (mIU/L)	5.15 ± 2.83	4.0 ± 2.45	0.8 – 13	0.5 – 14.5	0.290

Table 4: Comparison of thyroid profile in cases and controls

Thyroid profile	Groups		Total
	Case	Control	
Eutyhroid	74%	88%	81.00%
Primary hypothyroidism	6%	0%	3.00%
Subclinical hypothyroidism	20%	12%	16.00%
Total	100.00%	100.00%	100.00%

(X2 = 4.605, df = 2, p value=0.100)

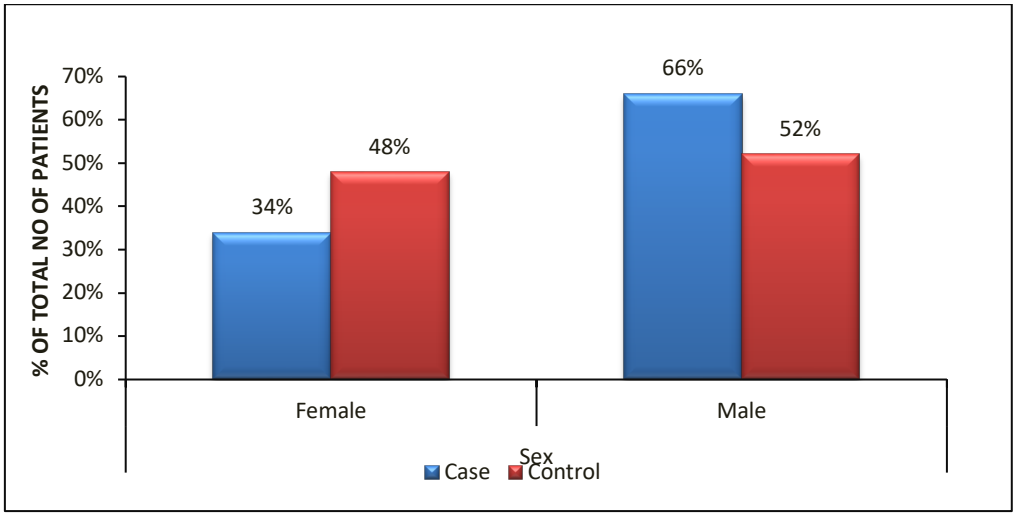

Figure 1: Sex distribution of cases (Thalassemia patients) and healthy controls

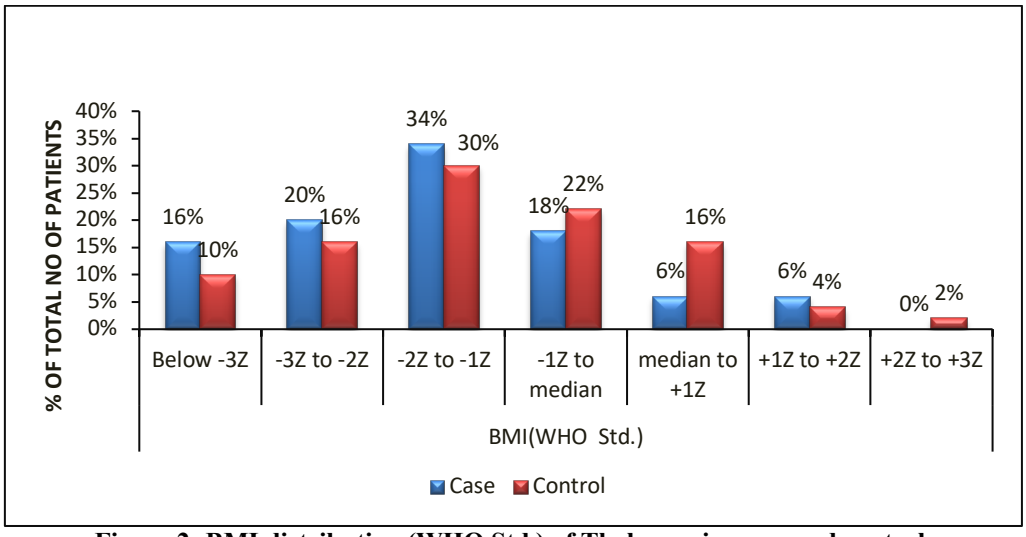

Figure 2: BMI distribution (WHO Std.) of Thalassemia cases and controls

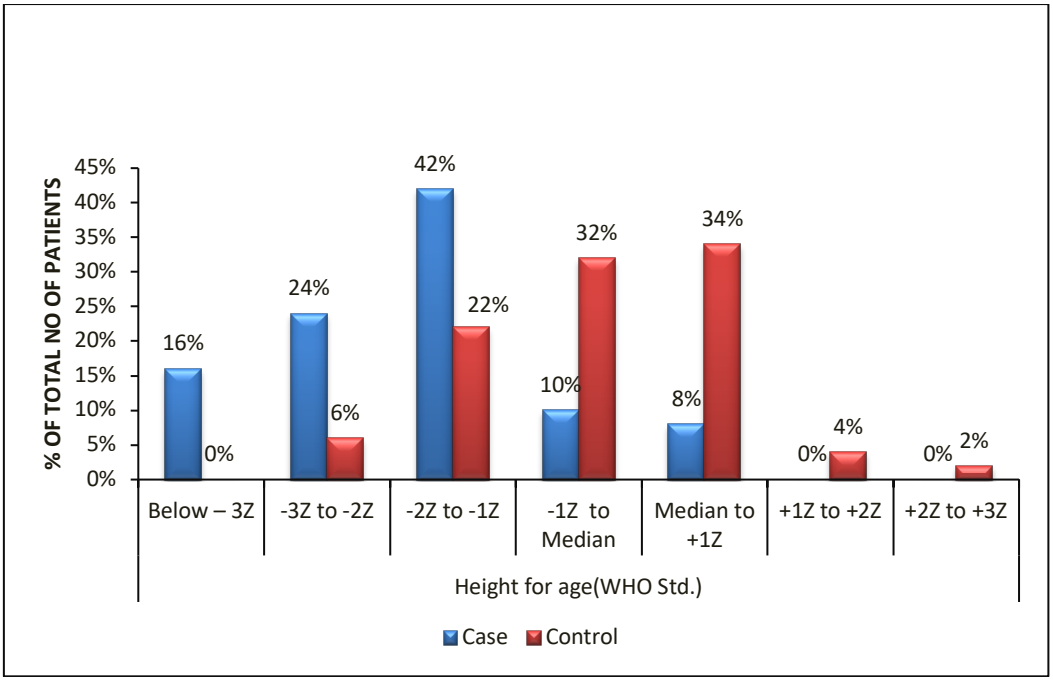

Figure 3: Height for age distribution (WHO Std.) of Thalassemia cases and controls

Discussion

Thalassemia is a heterogeneous inherited disorder of hemoglobin synthesis caused by mutations in globin genes, leading to varying degrees of quantitative defects in globin chain production and resulting in ineffective erythropoiesis and anemia [2]. Beta-thalassemia major (BTM) typically presents between 4–6 months of age due to the protective effect of high fetal hemoglobin levels at birth, which gradually decline during the first year of life [2]. Advances in regular blood transfusions and iron chelation therapy have significantly improved survival in thalassemia patients [6]. However, in the absence of a physiological pathway for iron excretion, repeated transfusions and increased intestinal iron absorption result in iron overload [7]. Despite increased longevity, this iron burden predisposes patients to multiple endocrine abnormalities, including hypogonadism, diabetes mellitus, hypothyroidism, and hypoparathyroidism [8].

A high prevalence of endocrine dysfunction in thalassemia has been documented by several studies [12,13]. Carmen Barbu et al. reported that 80% of patients with beta-thalassemia major had at least one endocrinopathy [14], while Sharma R et al. found a prevalence of 44.9% among adolescents (mean age 13.6 years) [15]. Iron overload remains the principal therapeutic complication, with hemosiderosis of endocrine glands, including the thyroid, well documented histologically in chronically transfused patients [7]. In the present study, we evaluated thyroid function in children with beta-thalassemia major and iron overload, comparing them with healthy controls. The mean age of cases was 11.45±3.65 years, while that of controls was 10.78±2.42 years, with no statistically significant difference (p=0.798). Females constituted 34% of cases and 48% of controls. Anthropometric assessment revealed that the mean weight of thalassemia patients (26.12±8.62 kg) was significantly lower than controls (30.56±9.47 kg; p=0.002). Additionally, most thalassemia patients up to 10 years of age had weights below the median when compared with WHO growth charts, consistent with findings by Tienboon P et al. [16]. However, no significant difference in weight was observed among thalassemia patients when stratified by thyroid status (p=0.253). Similarly, mean height was significantly lower in thalassemia patients (131.24±13.79 cm) compared to controls (139.11±12.32 cm; p=0.003), in agreement with Gulati R et al. [17].

A majority of cases (92%) were below the median height, and 16% were below -3 Z scores, indicating significant growth retardation. Comparable findings have been reported by Sharma R et al. [15] and Pignetti et al. [18]. However, height differences among thyroid function groups were not statistically significant (p=0.341), consistent with observations by Agarwal et al. [19] and Jain M et al. [20]. Body mass index (BMI) was comparable between cases and controls, with no statistically significant difference (p=0.461), and BMI-for-age distribution also did not differ significantly (p=0.581).

In our cohort, 26% of thalassemia patients exhibited some form of hypothyroidism, with 20% having subclinical hypothyroidism and 6% having primary hypothyroidism. No cases of secondary

hypothyroidism were identified. In the control group, 12% had subclinical hypothyroidism, while none had primary or secondary hypothyroidism. Although thalassemia patients showed lower mean T4 and higher mean TSH levels compared to controls, these differences were not statistically significant (p=0.242 and p=0.074, respectively). However, TSH levels were significantly elevated in patients with thyroid dysfunction (p<0.0001).Previous studies have reported variable prevalence of subclinical hypothyroidism in thalassemia, ranging from 8.9% to 32% [20-22], and primary hypothyroidism ranging from 3.2% to 12% [20,23]. While no cases of overt clinical hypothyroidism were observed in our study, similar findings have been reported by Gathwala et al. [21] and Gulati R et al. [17], although Agrawal et al. reported a prevalence of 6.9% [19].

No significant association was found between thyroid function and age at diagnosis, frequency of transfusion, or annual transfusion requirement, consistent with findings by Gathwala et al. [21]. However, Jain M et al. [20] reported a higher transfusion burden in hypothyroid patients. Similarly, no significant correlation was observed between thyroid function and current age, aligning with studies by Agarwal MB et al. [19] and Jain M et al. [20]. Pre-transfusion hemoglobin levels also did not show any significant association with thyroid dysfunction, as supported by previous studies [19,20].

Overall, our findings indicate that although growth retardation is common in thalassemia patients, it could not be statistically correlated with hypothyroidism alone. Other contributing factors such as malnutrition, growth hormone deficiency, chronic hypoxia, or additional endocrinopathies may play a role.

The strengths of our study include the inclusion of a healthy control group, selection of controls from siblings to minimize confounding factors, and assessment of organomegaly (liver and spleen size). However, limitations include the cross-sectional design, relatively small sample size, restriction of WHO growth standard comparisons to children up to 10 years, variability in chelation regimens, and the absence of hemoglobin and serum ferritin measurements in the control group.

Conclusion

This cross-sectional study of 50 transfusion-dependent beta-thalassemia major patients reveals that thyroid dysfunction represents a significant though often subclinical endocrine complication occurring in 26% of patients despite modern iron chelation therapy. The predominant form is subclinical hypothyroidism (20%), characterized by elevated TSH with preserved T4 levels, though primary hypothyroidism occurs in 6% with both T4 and TSH derangement.

Study Implications:

The persistent occurrence of thyroid dysfunction despite modern chelation therapy emphasizes need for:

1. Next-generation chelating agents with improved organ-specific iron removal
2. Longitudinal studies documenting disease progression and identifying intervention windows
3. Mechanistic investigations elucidating sex-specific and organ-specific iron toxicity
4. Prospective trials evaluating thyroid-protective strategies in iron overload

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