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Original Research Article

Thyroid Function in Transfusion-Dependent Thalassemic Children: A Cross-Sectional Analysis

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Abstract:

Background: Transfusion-dependent thalassemia is associated with iron overload–related endocrine complications, with thyroid dysfunction being a frequent concern in children. Early detection is essential to prevent adverse effects on growth and development.

Aim: To assess thyroid function status and its association with iron overload and transfusion burden in transfusion-dependent thalassemic children.

Methodology: This hospital-based cross-sectional study included 76 children aged 5–18 years with β-thalassemia major or HbE/β-thalassemia receiving regular transfusions. Serum FT4, TSH, and ferritin levels were measured using chemiluminescent immunoassay. Thyroid status was categorized as euthyroid, compensated (subclinical) hypothyroid, or uncompensated (overt) hypothyroid.

Results: Most children were euthyroid (89.5%). Hypothyroidism was detected in 10.5%, predominantly compensated (9.2%), with overt hypothyroidism in 1.3%. All hypothyroid patients had serum ferritin ≥2000 ng/ml, showing a significant association with iron overload ($p = 0.031$). The hypothyroid group had a significantly higher mean number of transfusions ($p < 0.0001$).

Conclusion: Thyroid dysfunction, mainly subclinical hypothyroidism, occurs in a notable proportion of transfusion-dependent thalassemic children and is associated with higher iron burden and transfusion exposure. Regular thyroid screening is recommended.

Keywords: Thalassemia, Hypothyroidism, Iron Overload, Serum Ferritin, Children, Transfusion Dependent.

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Introduction

Thyroid hormones are essential in human growth, development, and metabolism from birth throughout infancy and childhood. From the onset of life, thyroid hormones play an active role in normal brain maturation, somatic growth, and regulation of basal metabolic activity, thus affecting most, if not all, organ systems in the body. Even minor perturbations in thyroid function in children may lead to significant impairment in terms of growth, delayed puberty, cognitive problems, or metabolic disturbances. For this reason, maintenance of normal thyroid function becomes particularly crucial in children with chronic illnesses. Several studies have reported thyroid dysfunction in patients with thalassemia, making the thyroid one of the commonly involved endocrine organs in the disease process [1].

Transfusion-dependent thalassemia is a serious lifelong hematological disorder, with subgroups that would eventually necessitate periodic blood transfusions to keep them alive. Inadequate transfusion support leads to serious anemia in affected individuals, including growth retardation, skeletal deformities, and multiple organ dysfunctions, and their life expectancy is considerably compromised. This category mainly includes patients suffering from β-thalassemia major and severe HbE β-thalassemia [2]. Advances in supportive care through transfusion and chelation therapy have greatly improved survival but have also unleashed a wave of long-term complications, most notably endocrine disorders, as patients reach adolescence and adulthood.

Thalassemia is a significant public health problem in most developing countries, including Bangladesh. A

World Health Organization report quotes approximately 3-4% of the Indian population as carriers of the β-thalassemia trait, whereas HbE carriers are more prevalent (50% of the regional population) in North- Eastern States [3][28].While HbD is present in about 2% of people in Punjab , HbS is more common in the Tribal Population of India.[3][28] These carrier rates are expected to account for a significant disease burden in the population. It is estimated that 10000-15000 babies are born in India with Thalassemia Major every year. These figures depict the magnitude of the problem and call for comprehensive clinical management strategies, including screening for long-term complications such as endocrine dysfunction.

The underlying pathophysiology of β-thalassemia is characterized by reduced or absent synthesis of β-globin chains, giving rise to an excess of free, unpaired α-globin chains. This leads to ineffective erythropoiesis, chronic hemolysis, and a net reduction in hemoglobin production [5]. The excess α chains precipitate and cause damage to erythroid precursors, leading to anemia and increased bone marrow activity. Perhaps one of the most common and clinically significant combinations is the HbE/β-thalassemia. This constitutes the most common form of thalassemia in Southeast Asia. The clinical severity is variable, but the majority of cases result in transfusion dependence early in life.

Although lifesaving, regular blood transfusions inevitably lead to progressive iron overload. Excess iron is deposited in the liver, heart, pancreas, pituitary gland, and thyroid gland. Inadequate chelation therapy, poor compliance, and the chronicity of the disease further increase iron burden. Thus, patients with transfusion-dependent thalassemia are at risk from a wide range of complications that include cardiomyopathy, hypogonadism, diabetes mellitus, hypothyroidism, hypoparathyroidism, and other endocrine and metabolic disorders, particularly during the second decade of life and early adulthood [6]. Of these, endocrine complications are major contributors to morbidity and detract from quality of life.

Hypothyroidism is considered the second most common endocrine complication in thalassemia patients after hypogonadism. Most of the patients show subclinical or mild hypothyroidism, with increased TSH and normal FT4 levels. Overt hypothyroidism, however, demonstrated by both elevated TSH and decreased FT4 levels, may appear in approximately one-third of the patients. Less commonly but clinically significant, it could be due to central hypothyroidism originating from pituitary or hypothalamic dysfunction [7]. Because early thyroid dysfunction has a very insidious onset and is often asymptomatic, regular screening is of much importance. It should be thus considered that routine monitoring of FT4 and TSH in thalassemia patients thereafter should be performed after the first decade of life for the early diagnosis and timely treatment [8].

The reported frequency of hypothyroidism in thalassemic patients varies greatly among series, from 6% to 30% [9]. These variations may be related to several factors, including patient age, transfusion therapy duration and intensity, iron chelation adequacy, and genetic predisposition. Serum ferritin levels are considered a valid indicator of iron stores; however, the relationship between iron burden, reflected by serum ferritin levels, and thyroid dysfunction remains controversial. Many studies have indeed reported that the incidence of hypothyroidism is not significantly related to elevated serum ferritin levels [10]. In contrast, other studies have found that serum ferritin levels are significantly higher in hypothyroid than in euthyroid thalassemia major patients, supporting the hypothesis of iron mediated thyroid gland damage [11]. These data suggest that thyroid dysfunction in thalassemia is multifactorial and not only dependent on iron burden.

Although thalassemia is highly prevalent in South Asian countries, there is a lack of country specific data on endocrine complications-thyroid dysfunction, particularly in children with thalassemia. Few studies have been conducted regarding thyroid function status. One such Bangladeshi study by Karim et al. reported hypothyroidism in 10 (20%) patients. Of these, compensated (subclinical) hypothyroidism was present in 5 (10%) cases, while decompensated (overt) hypothyroidism was diagnosed in 5 (10%) cases [12]. Though informative, such limited data are too scant to describe the burden of thyroid dysfunction in this vulnerable population.

In view of the growing survival of transfusion-dependent thalassemic children and the impact that thyroid dysfunction may have on growth, development, and overall health status, there is a need for a systematic evaluation of thyroid function. Early identification and management of thyroid abnormalities may prevent chronic morbid conditions and improve quality of life. Therefore, the present study aims to determine the status of the thyroid function in transfusion dependent thalassemic children. The findings of this study are expected to raise the readers' awareness regarding the necessity of routine thyroid function evaluation in these patients and to add to the existing body of knowledge on endocrine complications of thalassemia.

Methodology

Study Design: This was a hospital-based cross-sectional observational study conducted to evaluate thyroid function status in transfusion-dependent thalassemic children.

Study Area: The study was carried out in the Upgraded Department of Pediatrics, Patna Medical College and Hospital (PMCH), Patna, Bihar, India.

Study Duration: The study was conducted over a period of Nine months from February 2025 to October 2025.

Sample Size: A total of 76 transfusion-dependent thalassemic children were included in the study.

Study Population: The study population consisted of children aged 5–18 years diagnosed with transfusion-dependent thalassemia (β-thalassemia major and Hb E/β-thalassemia) who were receiving regular blood transfusions and attending the Department of Pediatrics, PMCH.

Inclusion Criteria

- Children aged 5–18 years
- Diagnosed cases of transfusion-dependent thalassemia (β-thalassemia major and Hb E/β-thalassemia)
- History of receiving at least 10 blood transfusions
- Patients attending PMCH for regular transfusion therapy

Exclusion Criteria

- Patients who had received fewer than 10 blood transfusions
- Severely ill children unable to participate in the study
- Known cases of hypothyroidism already on treatment

Data Collection: After obtaining informed written consent from parents or guardians, data were collected using a pre-designed and pre-tested data collection proforma. Detailed demographic and clinical information including age, sex, type of thalassemia, age at first diagnosis, age at first blood transfusion, total number of blood transfusions received, type and duration of iron chelation therapy, adherence to chelation therapy, and family history of thalassemia and thyroid disorders were recorded. Anthropometric measurements such as height and weight were measured using standard techniques. For laboratory evaluation, 4 ml of venous blood was collected aseptically in the morning prior to the scheduled blood transfusion. Serum free thyroxine (FT4), thyroid stimulating hormone (TSH), and serum ferritin levels were estimated using chemiluminescent immunoassay with an automated analyzer in the hospital laboratory. Based on thyroid function test results, the patients were classified into euthyroid, compensated hypothyroid, and uncompensated hypothyroid groups.

Study Procedure: Eligible participants fulfilling the inclusion criteria were enrolled consecutively. Clinical evaluation, anthropometric measurements, and blood sample collection were performed during routine hospital visits for transfusion. Laboratory investigations were carried out following standard operating procedures, and all results were systematically recorded.

Statistical Analysis: All collected data were entered into a computer-based database and analyzed using the Statistical Package for Social Sciences (SPSS), version 21. Continuous variables were expressed as mean and standard deviation, median, and range, while categorical variables were presented as frequencies and percentages. Comparison of categorical variables was performed using the Chi-square test or Fisher's exact test as appropriate. The unpaired t-test was used to compare means between two groups. Correlation between serum ferritin levels and thyroid function parameters was assessed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

Result

Table 1 summarizes the demographic and clinical profile of the 76 transfusion-dependent thalassemic children, who had a mean age of 12.4 ± 3.8 years, with a male predominance (57.9%). The cohort showed evidence of growth compromise, reflected by low median WAZ (−1.85) and HAZ (−2.82), while the mean BMI was 18.1 ± 3.2 kg/m². Most patients had Hb E/β-thalassemia (73.7%), with the remainder having β-thalassemia major (26.3%). The disease was diagnosed early in life (mean 17.6 months), with blood transfusions initiated at a mean age of 19.4 months, and patients had received a substantial number of transfusions (mean 79.8). Although 71.1% received iron chelation therapy, good compliance was observed in only 23.7%, highlighting suboptimal adherence despite prolonged disease duration and significant transfusion burden.

Table 1: Demographic and Clinical Characteristics of Study Population (n = 76)

Variables	Patients (%)
Age (mean ± SD) (years)*	12.4 ± 3.8
Sex**	
Male	44 (57.9)
Female	32 (42.1)
Weight (mean ± SD) (kg)*	30.9 ± 9.8
Height (mean ± SD) (cm)*	129.1 ± 14.6
WAZ median (range)°	-1.85 (-0.9 to -4.0)
HAZ median (range)°	-2.82 (-0.8 to -4.9)

BMI (mean ± SD) (kg/m²)*	18.1 ± 3.2
Type of thalassemia**	
β-thalassemia major	20 (26.3)
Hb E/β-thalassemia	56 (73.7)
Age at first diagnosis (months)*	17.6 ± 7.4
Total duration of disease (years)*	10.7 ± 3.9
Age at first blood transfusion (months)*	19.4 ± 7.1
Total number of transfusions*	79.8 ± 30.6
Family history of thalassemia**	19 (25.0)
Received iron chelation therapy**	54 (71.1)
Good compliance to chelation therapy**	18 (23.7)
Duration of chelation therapy (months)*	18.1 ± 14.9

Table 2 demonstrates that the majority of transfusion-dependent thalassemic children were euthyroid (89.5%), while hypothyroidism was observed in 10.5% of cases, predominantly in the compensated (subclinical) form (9.2%), with only a small proportion showing uncompensated (overt) hypothyroidism (1.3%), indicating that thyroid dysfunction in this population is relatively uncommon and is more often subclinical in nature.

Table 2: Thyroid Function Status in Transfusion-Dependent Thalassemic Children (n = 76)		
Thyroid function status	**N**	**Percentage (%)**
Euthyroid	68	89.5
Hypothyroid		
Compensated (subclinical)	7	9.2
Uncompensated (overt)	1	1.3
Total	**76**	**100**

Table 3 shows a significant association between elevated serum ferritin levels and thyroid status, with all hypothyroid patients (100%) having serum ferritin ≥2000 ng/ml compared to 60.3% of euthyroid patients, while none of the hypothyroid patients had ferritin levels <2000 ng/ml; this association was statistically significant (p = 0.031), suggesting that higher iron overload is strongly linked to the presence of hypothyroidism.

Table 3: Association Between Serum Ferritin Level and Thyroid Status (n = 76)			
Serum ferritin (ng/ml)	**Euthyroid (n = 68)**	**Hypothyroid (n = 8)**	**P value**
≥2000	41 (60.3%)	8 (100%)	0.031[s]
<2000	27 (39.7%)	0 (0%)	

Table 4 demonstrates that the euthyroid and hypothyroid groups were comparable with respect to age, sex distribution, anthropometric parameters, age at diagnosis, disease duration, and age at first transfusion, with no statistically significant differences observed (p > 0.05 for all). The proportion of patients receiving iron chelation therapy and those showing good compliance to chelation was also similar between the two groups. However, the total number of transfusions was significantly higher in the hypothyroid group (121.8 ± 36.9) compared to the euthyroid group (75.6 ± 26.8), and this difference was highly significant (p < 0.0001), suggesting a possible association between higher transfusion burden and the development of hypothyroidism.

Table 4: Comparison of Demographic and Clinical Characteristics Between Euthyroid and Hypothyroid Groups			
Variables	**Euthyroid (n = 68)**	**Hypothyroid (n = 8)**	**P value**
Age (years)*	12.2 ± 3.7	13.6 ± 3.4	0.29[ns]
Sex			
Male	39 (57.4)	5 (62.5)	1.00[ns]
Female	29 (42.6)	3 (37.5)	
Weight (kg)*	30.6 ± 9.9	33.4 ± 8.8	0.41[ns]
Height (cm)*	128.8 ± 14.9	131.6 ± 13.2	0.58[ns]
Age at first diagnosis (months)*	17.4 ± 7.5	18.5 ± 7.2	0.73[ns]
Total duration of disease (years)*	10.4 ± 3.8	12.6 ± 3.7	0.11[ns]

Age at first transfusion (months)*	19.2 ± 7.4	20.3 ± 6.1	0.69[ns]
Total number of transfusions*	75.6 ± 26.8	121.8 ± 36.9	<0.0001[s]
Received iron chelation therapy	49 (72.1%)	5 (62.5%)	0.71[ns]
Good compliance to chelation therapy	17 (25.0%)	1 (12.5%)	0.44[ns]

Table 5 shows that although mean serum ferritin levels were higher in hypothyroid patients (4728.4 ± 1986.3 ng/ml) than in euthyroid patients (3564.2 ± 2381.6 ng/ml), this difference was not statistically significant (p = 0.18), indicating similar iron status in both groups. In contrast, thyroid function parameters differed significantly, with hypothyroid patients having significantly lower FT4 levels (1.02 ± 0.24 ng/dl vs 1.21 ± 0.18 ng/dl; p < 0.0001) and markedly higher TSH levels (8.02 ± 1.63 µIU/ml vs 2.76 ± 1.21 µIU/ml; p < 0.0001) compared to euthyroid patients, confirming the presence of thyroid dysfunction in the hypothyroid group.

Table 5: Comparison of Mean Ferritin, FT4, and TSH Levels Between Euthyroid and Hypothyroid Patients

Parameter	Euthyroid (n = 68)	Hypothyroid (n = 8)	P value
Ferritin (ng/ml)*	3564.2 ± 2381.6	4728.4 ± 1986.3	0.18[ns]
FT4 (ng/dl)*	1.21 ± 0.18	1.02 ± 0.24	<0.0001[s]
TSH (µIU/ml)*	2.76 ± 1.21	8.02 ± 1.63	<0.0001[s]

Discussion

In the current study, the mean age of transfusion-dependent thalassemic children was 12.4 ± 3.8 years, with a male predominance of 57.9%. Very similar observations were reported by Jain et al. (2012) [13], whose mean age among their patients with thalassemia was 10.3 ± 3.6 years, thus suggesting a similar chronological age distribution. Predominantly, β-thalassemia major (73.7%) cases were found in our current study which matches the regional genetic pattern geographically. A study conducted by Tahura et al. in 2016 [14] that identified Hb E/β-thalassemia as the most common form of thalassemia among Bangladeshi children which reflected a more eastern regional genetic prevalence pattern. Studies conducted in Turkey were similar to that of Indian genetic patterns of Thalassemia, demonstrating that β-thalassemia major represented the majority of cases in transfusion dependent children, hence possibly suggesting population genetics and referral pattern differences; Kurtoglu et al. (2012); Agarwal et al. (1992) [15,16]."

Nutritional status assessment in our cohort showed growth impairment represented by a median weight-for-age and height-for-age Z score of -1.85 and -2.82, respectively. This is in agreement with many reports showing chronic growth retardation in thalassemic children dependent on transfusion, being the result of the combined influences of chronic anemia, iron overload, and poor nutritional intake (Sharmin et al., 2018; Gathwala et al., 2009) [17,18]. Somchit et al. (2007) [19] also reported similar results in cases with thalassemia receiving hyper transfusion, the mean weight for age and height for age Z score in these patients were -1.9 and -2.5 respectively. However, studies of well-chelated cohorts, such as the study by Zervas et al. (2002) [20], showed comparatively better growth parameters, underlining the protective role of regular iron chelation therapy.

Regarding thyroid function, our patients were euthyroid in 89.5%, while 10.5% had hypothyroidism, the majority being subclinical 9.2%, with overt hypothyroidism being less frequent 1.3%. This is in close approximation with Kurtoglu et al. (2012) [15], who reported a prevalence of 12.8% hypothyroidism in transfusion-dependent thalassemic patients, with a predominance of subclinical cases. Somchit et al. (2007) [19] reported slightly higher rates, 17.6% hypothyroidism, whereas Sharmin et al. (2018) [17] found a 26% prevalence of subclinical hypothyroidism. An even higher prevalence was reported by Karim et al. (2012) [12], 20%, indicating a wide variation in the reported thyroid dysfunction, which may be related to age, genotype, ethnic background, frequency of transfusions, and adherence to chelation, as mentioned by Shamshirsaz et al. (2003); Malik et al. (2010) [21,22],. In our study, compensated hypothyroidism prevailed in 88.9%, similarly to the findings by Zervas et al. (2002) [20] and Agarwal et al. (1992) [16], while overt hypothyroidism remained infrequent, similarly, to reports from Gathwala et al. (2009) [18], who had 12% overt hypothyroidism in their series.

In our cohort, there was a significant association between iron overload and thyroid dysfunction since all the hypothyroid children had serum ferritin above 2000 ng/ml. This is in accordance with Jaruratanasirikul et al. 2007 [23], who found that high ferritin levels are strong predictors for developing hypothyroidism, with levels of more than 3500 ng/ml being more significantly associated with hypothyroidism. Similarly, Neha et al. 2016 [24] also showed that the higher the cumulative transfusion and ferritin levels, the higher the risk of hypothyroidism. Thus, this again emphasizes the idea of transfusional iron load as a contributing factor to

endocrine complications. In our study, the mean number of transfusions was significantly higher in hypothyroid children (121.8 ± 36.9) compared to euthyroid patients (75.6 ± 26.8), supporting the concept of cumulative iron burden as a risk factor, as has been similarly noted in other cohorts by Nijaguna et al., 2015 [25].

Interestingly, age, sex, and chelation therapy adherence were not significantly different between hypothyroid and euthyroid groups, similar to previous studies by Jain et al. (2012) [13] and Upadya et al. (2016) [26]. This implies that the cause of thyroid dysfunction in thalassemia patients may be multifactorial and not solely dependent on chelation compliance, including chronic hypoxia, oxidative stress, and individual susceptibility. The mean age of hypothyroid children in our study was 13.89 ± 3.48 years, similar to reports of Somchit et al. (2007) [19] and Malik et al. (2010) [22], who stated that thyroid complications often present in the second decade of life. Sanctis et al. (2009) [27] emphasized that thyroid dysfunction generally increases with advancing age and correlates with the timing and adherence to chelation therapy.

Biochemically, our hypothyroid cohort had significantly elevated TSH and decreased free thyroxine levels, consistent with the classical pattern of compensated hypothyroidism described in multi-transfused patients (Sharmin et al., 2018; Agarwal et al., 1992) [17,16]. Serum ferritin level was higher in hypothyroid patients (4728.4 ± 1986.3 ng/ml) compared to that in euthyroid patients (3564.2 ± 2381.6 ng/ml), though not statistically significant, which may be explained by variation in transfusion and chelation practices. Similar trends were reported by Zervas et al. (2002) [20] and again suggest that though iron overload is a major contributing factor, individual variation in susceptibility of organs and distribution of iron also contributes to endocrine outcomes.

Overall, our findings emphasize the need for regular follow-up of thyroid function in transfusion-dependent thalassemic children, especially those with a high transfusion burden or who have elevated ferritin levels. Early detection of subclinical hypothyroidism enables timely intervention that may soften the effects on growth and development. The results are in large part consistent with international data, while emphasizing context-specific management strategies relevant to local patterns of thalassemia, transfusion practices, and chelation adherence.

Conclusion

This cross-sectional analysis affirms that the majority of transfusion-dependent thalassemic children enjoy normal thyroid function, but a significant percentage show thyroid dysfunction, with the compensated form predominating. Thyroid abnormalities correlated significantly with the markers for iron overload and higher total cumulative transfusion burden, underlining iron toxicity as the major determinant for endocrine complications in this group. Demographic characteristics, growth parameters, age at diagnosis, and chelation therapy status did not correlate significantly with thyroid dysfunction. These observations emphasize the need for periodical thyroid function screening, especially in children with increased exposure to transfusions and poor iron control, for timely identification and intervention to minimize long-term morbidity in transfusion-dependent thalassemia.

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