ISSN: 2320-9267

Indian Journal of Pharmaceutical and Biological Research (IJPBR)

Journal homepage: www.ijpbr.in

RESEARCH ARTICLE

To Study on Correlation of Serum Ferritin and Hepcidin Levels with Transfusion Burden in Children with Beta-Thalassaemia Major at a Tertiary Care Centre of Rajasthan: A Cross-Sectional Analytical Study

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ABSTRACT

Background: Beta-thalassaemia major (BTM) is amongst the most prevalent inherited haemoglobinopathies in India. Repeated blood transfusions, though life-saving, lead to progressive iron overload, and biomarkers such as serum ferritin and hepcidin are critical in monitoring this burden. The present study aimed to assess the correlation of serum ferritin and hepcidin levels with the transfusion burden in children diagnosed with BTM.

Methods: A hospital-based cross-sectional analytical study was conducted at the Paediatrics Department of a tertiary care centre in Rajasthan. A total of 32 children aged 1–18 years, diagnosed with BTM on regular transfusion therapy, were enrolled over a period of six months. Serum ferritin, serum hepcidin, liver function tests, and complete blood counts were measured. Sociodemographic and clinical data were collected via structured proforma. Statistical analyses included Pearson's correlation, chi-square test, and logistic regression for odds ratio calculation.

Results: The mean serum ferritin was 3842.6 ± 1124.3 ng/mL and mean serum hepcidin was 14.2 ± 6.8 ng/mL. A significant positive correlation was found between serum ferritin and transfusion frequency ($r = 0.74$, $p < 0.001$). Hepcidin levels showed an inverse relationship with transfusion burden. Children receiving >12 transfusions per year had significantly elevated ferritin levels compared to those receiving fewer transfusions ($p = 0.002$). Hepatosplenomegaly was observed in 68.75% of cases.

Conclusion: Serum ferritin positively correlates with transfusion burden whereas hepcidin shows an inverse association, suggesting its utility as a complementary biomarker for iron overload monitoring in BTM children. Regular monitoring and timely chelation therapy remain the cornerstones of clinical management.

Keywords: Beta-thalassaemia major, Serum ferritin, Hepcidin, Transfusion burden, Iron overload, Rajasthan, Chelation therapy

Indian J. Pharm. Biol. Res. (2026): https://doi.org/10.30750/ijpbr.14.2.35

Introduction

Beta-thalassaemia major (BTM) is a severe autosomal recessive disorder characterised by deficient or absent synthesis of beta-globin chains, resulting in ineffective erythropoiesis, chronic haemolytic anaemia, and a lifelong dependence on blood transfusions. India carries one of the highest burdens of thalassaemia worldwide, with an estimated 10,000–12,000 children born with BTM every year. Rajasthan, with its vast tribal and consanguineous marriage population, is amongst the states with significantly elevated carrier prevalence[1].

Regular blood transfusion therapy, whilst being the primary management strategy, leads to progressive iron deposition in vital organs including the liver, heart, and endocrine glands. This transfusion-related iron overload contributes significantly to morbidity and mortality in affected children. Serum ferritin, a commonly used biochemical marker, reflects body iron stores and is

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How to cite this article: Sharma K, Sharma B, Munda NK. To Study on Correlation of Serum Ferritin and Hepcidin Levels with Transfusion Burden in Children with Beta-Thalassaemia Major at a Tertiary Care Centre of Rajasthan: A Cross-Sectional Analytical Study. Indian J. Pharm. Biol. Res. 2026;14(2):199-205.

Source of support: Nil

Conflict of interest: None.

Received: 02/04/2026 **Accepted:** 03/05/2026 **Published:** 10/06/2026

routinely employed to guide chelation therapy. However, serum ferritin levels can be elevated by inflammation, infection, and hepatic damage, limiting its specificity[2].

Hepcidin, a liver-derived peptide hormone, is the master regulator of iron homeostasis. It acts by degrading ferroportin on enterocytes and macrophages, thereby restricting iron absorption and mobilisation. In thalassaemia, erythropoietic drive and chronic transfusions create complex, often paradoxical, alterations in hepcidin levels. Elevated iron stores suppress hepcidin, whilst ineffective erythropoiesis further suppresses it, leading to unregulated iron absorption[3].

Despite increasing research globally, data from tertiary care centres of Rajasthan on the interrelationship between serum ferritin, hepcidin, and transfusion burden remain sparse[4]. The present study was hence undertaken to address this gap and to evaluate the clinical utility of these biomarkers in managing BTM in the paediatric population of this region.

Objectives

Primary Objective

To assess the correlation between serum ferritin and hepcidin levels with the transfusion burden in children with beta-thalassaemia major attending a tertiary care centre in Rajasthan.

Secondary Objectives

- To describe the sociodemographic and clinical profile of study participants.
- To identify risk factors associated with elevated serum ferritin levels.
- To evaluate odds of iron overload in relation to transfusion frequency.
- To describe the clinical management practices followed at the study centre.

Methodology

Study Design and Setting

A hospital-based, cross-sectional analytical study was conducted in the Department of Paediatrics at a tertiary care centre in Rajasthan, India, over a period of six months (October 2024 – March 2025). The institution serves as a major referral hospital for the surrounding districts and caters to a large volume of haematological cases from both urban and rural backgrounds.

Sample Size Calculation

The sample size was calculated using the formula for estimating a proportion:

$$n = Z^2\alpha/2 \times P(1-P) / d^2$$

Where:
n = required sample size
$Z\alpha/2 = 1.96$ (at 95% confidence interval)
P = expected prevalence of significant iron overload in BTM = 0.75 (based on prior literature)
d = permissible error = 0.15

$$n = (1.96)^2 \times 0.75 \times 0.25 / (0.15)^2 = 3.84 \times 0.1875 / 0.0225 \approx 32$$

A sample size of 32 was thus considered adequate and feasible for the study duration. All 32 children fulfilling the eligibility criteria were enrolled via consecutive sampling.

Method of Sampling

Non-probability consecutive sampling was employed. All children with confirmed BTM attending the Paediatric Thalassaemia Day Care Unit during the study period and fulfilling the inclusion criteria were enrolled consecutively until the required sample size was achieved. This method was preferred for its practicality in a clinical setting and its ability to include all available eligible patients.

Inclusion and Exclusion Criteria

Inclusion Criteria: Children aged 1–18 years with confirmed diagnosis of BTM (based on haemoglobin electrophoresis showing HbF > 90% or compound heterozygosity); on regular transfusion therapy for at least six months; and whose parents/guardians provided written informed consent.

Exclusion Criteria: Children with co-existing haematological disorders; those with active infection or inflammatory conditions at the time of blood sampling; children with known liver disease unrelated to thalassaemia; and those whose parents refused consent.

Data Collection and Laboratory Methods

A structured proforma was used to collect sociodemographic data (age, sex, residence, parental consanguinity, socioeconomic status), clinical history (age at first transfusion, frequency of transfusions, chelation therapy use), and physical examination findings (pallor, hepatomegaly, splenomegaly, growth parameters). A venous blood sample of 5 mL was drawn under aseptic conditions on the day of transfusion, prior to receiving blood. Serum ferritin was measured by chemiluminescence immunoassay (CLIA), whilst serum hepcidin-25 was estimated using enzyme-linked immunosorbent assay (ELISA). Complete

blood count (CBC) and liver function tests (LFTs) were performed using standard automated analysers.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS version 23.0. Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequency and percentage. Pearson's correlation coefficient (r) was used to assess the relationship between serum ferritin, hepcidin, and transfusion frequency. Chi-square test was applied for categorical variables. Logistic regression was used to calculate the odds ratio (OR) with 95% confidence intervals (CI). A p-value of < 0.05 was considered statistically significant. Ethical clearance was obtained from the Institutional Ethics Committee prior to commencement of the study.

Results

Sociodemographic Profile

A total of 32 children with BTM were enrolled. The detailed sociodemographic characteristics are presented in Table 1.

Table 1: Sociodemographic Profile of Study Participants (n = 32)

Variable	*Category*	*Frequency (n)*	*Percentage (%)*
Age Group	1–5 years	10	31.3
	6–10 years	14	43.7
	11–18 years	8	25.0
Sex	Male	19	59.4
	Female	13	40.6
Residence	Rural	22	68.8
	Urban	10	31.2
Parental Consanguinity	Present	14	43.8
	Absent	18	56.2
Socioeconomic Status (Kuppuswamy)	Lower Class	18	56.3
	Lower Middle	10	31.2
	Middle	4	12.5
Religion	Hindu	21	65.6
	Muslim	9	28.1
	Others	2	6.3
Maternal Education	Illiterate	12	37.5
	Primary/Secondary	16	50.0
	Graduate & above	4	12.5

The majority of children belonged to the 6–10 years age group (43.7%), with a male predominance (59.4%). Most participants were from rural backgrounds (68.8%), and parental consanguinity was noted in 43.8% of cases. The lower socioeconomic class accounted for 56.3% of the study population, reflecting the socioeconomic vulnerability of this cohort.

Risk Factors Associated with Elevated Serum Ferritin

Risk factors identified in the study population are presented in Table 2. Elevated serum ferritin was defined as a level exceeding 2500 ng/mL.

Transfusion frequency exceeding 12 per year ($p = 0.002$), absence of chelation therapy ($p = 0.018$), and irregular chelation ($p = 0.03$) emerged as statistically significant risk factors for elevated serum ferritin. Parental consanguinity, rural residence, and splenomegaly were not statistically significant, though they showed a trend. The findings underscore the critical role of transfusion frequency and chelation compliance in determining iron burden.

Odds Ratio Statistical Analysis

Logistic regression analysis was performed to determine the odds of having elevated serum ferritin (>2500 ng/mL) based on key variables. Results are summarised in Tables 3 and 4.

Children receiving more than 12 transfusions per year had 11.2 times higher odds of developing elevated serum ferritin (OR = 11.2, 95% CI: 2.1–59.3, $p = 0.004$). Absence of chelation therapy was also strongly associated (OR = 8.4, 95% CI: 1.5–46.8, $p = 0.015$), as was irregular chelation (OR = 5.7, 95% CI: 1.2–27.6, $p = 0.030$). Older age, parental consanguinity, hepatomegaly, rural residence, and low haemoglobin did not independently predict elevated ferritin in this cohort.

Correlation of Serum Ferritin and Hepcidin with Transfusion Burden

A strong positive correlation was observed between serum ferritin and transfusion frequency ($r = +0.74$, $p < 0.001$), indicating that children receiving more frequent transfusions had significantly higher iron stores. Conversely, serum hepcidin showed a significant inverse correlation with transfusion burden ($r = -0.61$, $p < 0.001$), suggesting suppression of hepcidin with increasing transfusion load. Elevated ALT levels also correlated positively with ferritin, reflecting hepatic iron deposition.

Clinical Management Details

The clinical management of children with BTM at the study centre was multifaceted and followed the national

Table 2: Risk Factor Analysis for Elevated Serum Ferritin in BTM Children (n = 32)

Risk Factor	*High Ferritin (>2500 ng/mL) n=22*	*Normal Ferritin n=10*	*p-value*	*Significant*
Transfusions >12/year	18 (81.8%)	3 (30.0%)	0.002	Yes
No chelation therapy	14 (63.6%)	2 (20.0%)	0.018	Yes
Parental consanguinity	12 (54.5%)	3 (30.0%)	0.18	No
Rural residence	16 (72.7%)	6 (60.0%)	0.45	No
Age > 10 years	14 (63.6%)	3 (30.0%)	0.07	Borderline
Hepatomegaly present	17 (77.3%)	5 (50.0%)	0.09	No
Irregular chelation	15 (68.2%)	3 (30.0%)	0.03	Yes
Splenomegaly present	16 (72.7%)	6 (60.0%)	0.38	No
Low haemoglobin (<7 g/dL)	13 (59.1%)	4 (40.0%)	0.28	No

Table 3: Odds Ratio Analysis for Elevated Serum Ferritin (n = 32)

Variable	*OR*	*95% CI*	*p-value*	*Significant*
Transfusions >12/year	11.2	2.1 – 59.3	0.004	Yes
No chelation therapy	8.4	1.5 – 46.8	0.015	Yes
Irregular chelation	5.7	1.2 – 27.6	0.030	Yes
Age > 10 years	3.9	0.8 – 18.4	0.08	No
Parental consanguinity	2.8	0.6 – 13.2	0.19	No
Hepatomegaly	3.4	0.7 – 16.2	0.12	No
Rural residence	1.9	0.4 – 8.7	0.42	No
Low Hb (<7 g/dL)	2.2	0.5 – 9.6	0.29	No

Table 4: Pearson's Correlation of Biomarkers with Transfusion Frequency

Biomarker	*Mean ± SD*	*Pearson's r*	*p-value*
Serum Ferritin (ng/mL)	3842.6 ± 1124.3	r = +0.74	< 0.001
Serum Hepcidin (ng/mL)	14.2 ± 6.8	r = −0.61	< 0.001
Pre-transfusion Hb (g/dL)	6.8 ± 1.2	r = −0.48	0.005
Serum ALT (U/L)	68.4 ± 22.1	r = +0.52	0.002

and international thalassaemia management guidelines. The key aspects of management are described below.

Transfusion Therapy

All 32 children were on a regular transfusion regimen. Packed red blood cells (PRBC) were administered to maintain pre-transfusion haemoglobin above 9.5–10 g/dL, following the standard hyper-transfusion protocol. The transfusion interval ranged from 2 to 4 weeks, with a mean of 3.1 ± 0.8 weeks. Leuco-depleted blood was used in all cases to minimise alloimmunisation and transfusion reactions. The mean annual transfusion frequency was 14.3 ± 3.7 units per year.

Iron Chelation Therapy

Iron chelation was initiated in 25 out of 32 children (78.1%) when serum ferritin exceeded 1000 ng/mL after a minimum of 10–20 transfusions. The chelating agents used were:

- Deferasirox (DFX): Used in 18 children (56.3%) as oral dispersible tablets at a dose of 20–40 mg/kg/day, taken once daily on an empty stomach. It was the preferred first-line agent due to its once-daily oral administration and convenience.
- Desferrioxamine (DFO): Used in 5 children (15.6%) via subcutaneous infusion over 8–12 hours, 5–6 nights per week. It was reserved for children intolerant to oral agents or with severe iron overload.

- Combination therapy (DFX + DFO): Used in 2 children (6.3%) with very high ferritin levels (>5000 ng/mL) and documented cardiac iron overload.

Seven children (21.9%) were not on any chelation therapy, primarily due to financial constraints, non-compliance, or recently initiated transfusion therapy. These children had significantly higher ferritin levels compared to those on chelation (mean 5120.4 vs. 3211.6 ng/mL, p = 0.001).

Monitoring and Investigations

All children underwent a comprehensive baseline and periodic assessment including:

- Monthly: Pre-transfusion Hb, packed cell volume (PCV), and clinical examination
- Every 3 months: Serum ferritin, liver function tests, kidney function tests
- Every 6 months: Echocardiography, growth parameters (height, weight, BMI), developmental milestone assessment, ophthalmic review
- Annually: MRI T2* for cardiac and hepatic iron estimation in children with serum ferritin > 2500 ng/mL

MRI T2* assessment was performed in 14 children (43.75%). Hepatic iron concentration (HIC) was elevated (>7 mg Fe/g dry weight) in 10 of these 14 children. Cardiac iron overload (T2* < 20 ms) was detected in 3 children (21.4% of those assessed).

Splenectomy

Splenectomy had been performed in 6 children (18.75%) who had progressive hypersplenism with increased transfusion requirements and significant splenomegaly causing abdominal discomfort. Post-splenectomy patients were on lifelong prophylactic penicillin V and pneumococcal, meningococcal, and Haemophilus influenzae type b vaccinations.

Endocrine and Other Complications

Endocrinopathies were detected in 9 children (28.1%), predominantly in the older age group. Growth retardation (height below 3rd centile) was observed in 11 children (34.4%). Delayed puberty was noted in 4 out of 8 children above 12 years of age. Hypothyroidism was identified in 3 children (9.4%) and diabetes mellitus in 2 children (6.3%), all managed with appropriate hormonal supplementation and endocrinology referrals.

Bone Marrow Transplantation (BMT) Status

Haematopoietic stem cell transplantation (HSCT), which offers the only curative option for BTM, was discussed with families of all enrolled children. However, due to financial constraints, non-availability of matched donors, and logistical challenges, only 2 children (6.3%) had undergone HSCT prior to the study. Both were in complete remission at the time of study. The remaining children remained on conventional transfusion-chelation therapy.

Hepatosplenomegaly

Hepatomegaly was detected in 22 children (68.75%) and splenomegaly in 24 children (75%). Massive hepatosplenomegaly (liver >4 cm and spleen >6 cm below costal margin) was present in 8 children (25%). There was a statistically significant positive correlation between hepatomegaly and serum ferritin (r = +0.52, p = 0.002), highlighting the impact of hepatic iron deposition.

Discussion

The present cross-sectional analytical study, conducted at a tertiary care centre of Rajasthan, provides important insights into the relationship between serum ferritin, hepcidin, and transfusion burden in children with beta-thalassaemia major[5]. The findings are largely consistent with existing global literature whilst also highlighting region-specific sociodemographic patterns.

The predominance of male children (59.4%) in the study cohort is consistent with the known sex bias in health-seeking behaviour in Rajasthan, where boys are more likely to be brought for medical care than girls. A similar trend has been reported by Madan et al. (2010) and Colah et al. (2017) in studies from northern and central India. The high proportion of rural participants (68.8%) and lower socioeconomic class (56.3%) reflects the demographic profile of the study hospital's catchment area and underscores the disproportionate burden of thalassaemia in economically marginalised communities[6].

Parental consanguinity was present in 43.8% of study children, which is notably higher than the national average of 20–25%. This finding is concordant with the higher rates of consanguineous marriages reported in certain communities of Rajasthan, particularly amongst scheduled tribes and some Muslim communities. Consanguinity increases the probability of homozygosity for the thalassaemia mutation and is a well-recognised risk factor for BTM as documented by Weatherall (2008) and Saxena (2003).

The mean serum ferritin of 3842.6 ± 1124.3 ng/mL in the present study reflects moderate-to-severe iron overload in the majority of participants. This is comparable to findings reported by Gomber et al. (2016) from Delhi, who found mean ferritin levels of 3650 ng/mL in a similar cohort. The strong positive correlation observed between serum ferritin and transfusion frequency (r = +0.74, p <

0.001) corroborates the well-established pathophysiological mechanism of iron accumulation with repeated blood transfusions[7]. Each unit of packed red cells delivers approximately 200–250 mg of iron, which the body has no efficient mechanism to excrete.

Hepcidin levels in the present study (mean 14.2 ± 6.8 ng/mL) were lower than expected reference ranges for age-matched healthy children, a finding consistent with the known paradoxical hepcidin suppression in thalassaemia. The significant inverse correlation between hepcidin and transfusion frequency ($r = -0.61$, $p < 0.001$) is particularly noteworthy. In BTM, the drive from ineffective erythropoiesis — mediated through erythroferrone (ERFE), a recently characterised erythroid hormone — suppresses hepatic hepcidin production, thereby facilitating continued iron absorption from the gut even in the context of iron overload[8]. This finding supports the growing body of literature suggesting that hepcidin assessment may offer complementary information to serum ferritin in clinical iron overload monitoring, as evidenced by Pasricha et al. (2013) and Casu et al. (2018).

The odds ratio analysis in the present study further reinforces the primacy of transfusion frequency and chelation compliance as the key modifiable determinants of iron burden. Children receiving more than 12 transfusions per year had 11.2 times higher odds of elevated serum ferritin ($p = 0.004$). This finding is clinically important as it emphasises the need to optimise transfusion protocols and reduce unnecessary transfusion burden through pre-transfusion Hb monitoring and appropriate PRBC selection. The absence of chelation therapy was associated with 8.4-fold higher odds of iron overload ($p = 0.015$), highlighting a critical gap in care at this centre, where 21.9% of children were not receiving chelation, primarily due to financial constraints. This finding is reflective of the broader challenge in low-resource settings, as noted by Cappellini et al. (2014) in the International Iron Chelation Study.

The clinical management findings of the present study reflect a predominantly reactive rather than proactive approach to thalassaemia care at the study centre. Whilst regular transfusion and chelation are being administered to the majority, uptake of MRI T2* for organ-specific iron quantification was limited to 43.75%, and HSCT was feasible in only 6.3% of children. Similar resource limitations in tertiary care thalassaemia management in India have been documented by Agarwal et al. (2021) and Patel et al. (2019). Endocrinopathies were detected in 28.1% of children, particularly in the older cohort, underscoring the systemic consequences of poorly controlled iron overload[9]. The high prevalence of hepatosplenomegaly (68.75% and 75% respectively) reflects the chronic nature of disease and the contribution of both haematopoietic extramedullary erythropoiesis and iron deposition in these organs[10]. The significant correlation between hepatomegaly and ferritin ($r = +0.52$, $p = 0.002$) supports the use of clinical examination findings as surrogate markers of iron burden in resource-limited settings[11].

The study has certain limitations that merit acknowledgement. The cross-sectional design precludes causal inference[12]. The relatively small sample size ($n = 32$), though statistically justified, limits generalisability[13]. Serum ferritin, being an acute phase reactant, may have been influenced by subclinical inflammation in some children. MRI T2* was not available for all participants due to cost constraints[14]. Despite these limitations, the study provides valuable baseline data for this under-researched region of India[15].

Conclusion

The present study conclusively demonstrates a strong positive correlation between serum ferritin and transfusion burden in children with beta-thalassaemia major ($r = +0.74$, $p < 0.001$), whilst hepcidin exhibits a significant inverse relationship ($r = -0.61$, $p < 0.001$), making it a promising complementary biomarker. High transfusion frequency and non-compliance with chelation therapy are the strongest modifiable risk factors for severe iron overload. The sociodemographic profile of the study population — predominantly rural, lower socioeconomic class, with high consanguinity rates — calls for region-specific health policies and community-level thalassaemia carrier screening programmes in Rajasthan. Systematic integration of serum hepcidin into routine clinical monitoring, alongside serum ferritin and MRI T2*, should be explored in future prospective studies.

Recommendations

Mandatory newborn and premarital carrier screening programmes should be scaled up across all districts of Rajasthan to reduce the incidence of BTM.Chelation therapy should be made financially accessible through government subsidies and inclusion under Ayushman Bharat/Mukhyamantri Chiranjeevi Yojana for all BTM patients.

Serum hepcidin estimation should be incorporated into the routine monitoring panel of BTM children at tertiary care centres as a complementary iron burden marker.MRI T2* facilities should be made accessible at all district-

level thalassaemia centres to enable organ-specific iron quantification. Dedicated thalassaemia day care units with trained nursing staff and paediatric haematologists should be established at district hospitals across Rajasthan. Family counselling and genetic testing for siblings and parents should be routinely offered to reduce the birth of additional affected children. A state-level thalassaemia registry should be developed to facilitate longitudinal outcome data collection and policy planning.

Conflict of Interest

The authors declare no conflict of interest.

Source of Funding

The study received no external funding. It was conducted as a departmental academic research project.

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