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Original Research Article

Relationship between serum ferritin levels and hepatic and renal function parameters in patients with transfusion-dependent β-thalassemia major: a hospital-based cross-sectional study

Rahul Garg[1], Mandayal Jamatia[2]*, Ritesh Vishwakarma[1]

[1]Department of Medical Biochemistry, Index Medical College Hospital and Research Centre, Malwanchal University, Indore, Madhya Pradesh, India
[2]Department of Biochemistry, Jaipur National University Institute of Medical Sciences and Research Centre, Jaipur, Rajasthan, India

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***Correspondence:**
Dr. Mandayal Jamatia,
E-mail: jamatiamandayal@gmail.com

ABSTRACT

Background: Transfusion-dependent β-thalassemia major requires lifelong blood transfusions, resulting in iron overload and hepatic and renal complications. Serum ferritin is widely used as a surrogate marker of body iron stores. This study evaluated serum ferritin levels and their association with hepatic and renal function parameters in patients with transfusion-dependent β-thalassemia major.
Methods: This hospital-based cross-sectional study included 100 patients with transfusion-dependent β-thalassemia major. Demographic data were recorded, and serum ferritin, liver function tests, and renal function parameters were analyzed using laboratory methods. Data were analyzed using descriptive statistics and Pearson's correlation analysis.
Results: The mean serum ferritin level was 2701.48±1393.83 ng/ml, with 46% of patients exhibiting ferritin concentrations >2500 ng/ml. Hepatic abnormalities included elevated AST (72%), ALT (60%), bilirubin (55%), and ALP (33%). Renal assessment revealed reduced estimated glomerular filtration rate (eGFR) in 35% of patients, while elevated serum creatinine and blood urea nitrogen were observed in 17% and 13%, respectively. Serum ferritin showed weak but statistically significant negative correlations with ALT ($r=-0.227$, $p=0.023$) and total bilirubin ($r=-0.273$, $p=0.006$), whereas no significant correlation was found with serum creatinine.
Conclusions: Patients with transfusion-dependent β-thalassemia major demonstrated substantial iron overload with frequent hepatic and renal biochemical abnormalities. Regular monitoring of serum ferritin together with liver and renal function tests may facilitate early detection of organ involvement and optimization of iron-chelation therapy.

Keywords: Beta-thalassemia major, Blood transfusion, Iron overload; Kidney function tests, Liver function tests, Serum ferritin

INTRODUCTION

β-thalassemia major is one of the most common and severe inherited hemoglobin disorders worldwide and remains a major public health challenge, particularly in South Asia, the Middle East, and the Mediterranean region, where carrier frequencies are high. The disorder results from mutations in the HBB gene, leading to absent or markedly reduced synthesis of β-globin chains. Consequently, ineffective erythropoiesis, chronic hemolytic anemia, and severe tissue hypoxia develop, necessitating lifelong regular red blood cell transfusions beginning in early childhood. Despite considerable advances in diagnosis and management, transfusion-dependent β-thalassemia (TDT)

continues to be associated with substantial morbidity, reduced quality of life, and significant healthcare utilization.[1-4] Global epidemiologic studies have highlighted substantial geographic variation in the burden of clinically significant thalassemia, with important evidence gaps across different populations.[5,6] Studies from the Middle East have also documented the prevalence of hemoglobinopathies and the molecular heterogeneity of transfusion-dependent thalassemia.[7,8]

The introduction of regular transfusion therapy together with effective iron chelation has markedly improved survival, transforming β-thalassemia major from a fatal childhood disorder into a chronic disease with increasing life expectancy. However, prolonged survival has shifted the clinical focus from anemia-related mortality to the prevention and management of chronic transfusion-related complications, particularly iron overload. Each unit of transfused blood contains approximately 200-250 mg of iron, and because humans lack a physiological mechanism for active iron excretion, repeated transfusions result in progressive iron accumulation. Excess iron eventually exceeds the binding capacity of transferrin and ferritin, leading to the generation of non-transferrin-bound iron, oxidative stress, and tissue injury.[9-13] Persistent iron deposition in vital organs, including the liver, heart, endocrine glands, and kidneys, contributes substantially to long-term morbidity and mortality in patients with TDT. Recent advances in the therapeutic landscape, including disease-modifying treatment approaches such as luspatercept, have further expanded the management options for thalassemia while transfusion and iron-overload control remain central to care.[14]

The liver is the principal site of iron storage and is usually the earliest organ affected by transfusional iron overload. Excess hepatic iron promotes oxidative stress, lipid peroxidation, inflammatory responses, hepatocellular injury, and progressive fibrosis, which may eventually culminate in cirrhosis or hepatic failure if inadequately controlled.[15,16] Serum aminotransferases and other liver function parameters are frequently altered in transfusion-dependent patients and provide useful biochemical indicators of early hepatic involvement. Consequently, periodic assessment of hepatic function forms an integral component of routine surveillance in contemporary thalassemia management guidelines.

In addition to hepatic complications, renal involvement has increasingly been recognized as an important but often underappreciated consequence of transfusion-dependent β-thalassemia. Renal injury is multifactorial and may result from chronic anemia, persistent hypoxia, iron-mediated oxidative damage, hemosiderin deposition, repeated transfusions, and the potential nephrotoxic effects of iron chelation therapy. These mechanisms may lead to glomerular and tubular dysfunction that frequently remains clinically silent until significant renal impairment has occurred. Recent evidence indicates that early detection of renal dysfunction is essential for preserving long-term renal function and improving overall clinical outcomes in patients with TDT.[17,18]

Accurate assessment of body iron burden is fundamental for optimizing iron chelation therapy and preventing irreversible organ damage. Although magnetic resonance imaging (MRI)-based techniques, particularly T2* MRI and liver iron concentration assessment, are considered the gold standard for evaluating tissue iron deposition, their widespread use remains limited in many resource-constrained settings because of high cost, limited accessibility, and technical requirements. In contrast, serum ferritin estimation is inexpensive, readily available, minimally invasive, and remains the most widely used surrogate biomarker of total body iron stores in routine clinical practice. Although serum ferritin may be influenced by inflammation, infection, and liver disease, persistently elevated concentrations have consistently been associated with increased iron burden, progressive organ dysfunction, and adverse clinical outcomes in patients with transfusion-dependent β-thalassemia.[19-23]

Recent international guidelines and standards emphasize the importance of comprehensive monitoring for iron overload, along with serial evaluation of hepatic, cardiac, endocrine, and renal function, to facilitate early recognition of organ damage and individualized optimization of chelation therapy.[24,25] Nevertheless, data evaluating the relationship between serum ferritin and hepatic and renal biochemical parameters among transfusion-dependent β-thalassemia patients in Central India remain limited. Regional studies are particularly important because genetic heterogeneity, transfusion practices, chelation regimens, and healthcare accessibility may substantially influence iron burden and the pattern of organ involvement.

Therefore, the present study was undertaken to evaluate serum ferritin levels and investigate their association with hepatic and renal function parameters in patients with transfusion-dependent β-thalassemia major attending a tertiary care teaching hospital. The findings are expected to enhance understanding of iron overload-related organ dysfunction and provide clinically relevant evidence to support improved monitoring strategies, timely therapeutic interventions, and long-term management of this vulnerable patient population.

METHODS

Study design

This hospital-based cross-sectional observational study was conducted to assess serum ferritin levels and evaluate hepatic and renal function parameters among transfusion-dependent β-thalassemia major patients.

The study aimed to assess the extent of transfusion-related iron overload and its impact on hepatic and renal

biochemical profiles among transfusion-dependent thalassemia patients.

Sample size

Sample size was determined based on the prevalence reported in previous studies and feasibility.

Study setting and duration

The study was conducted in the Department of Medical Biochemistry in collaboration with the department of pediatrics, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India. The study was conducted between February 2025 and February 2026. The institution is a tertiary care teaching hospital catering to a large population of pediatric and hematological patients from Madhya Pradesh and neighboring states.

Study population

A total of 100 clinically and hematologically confirmed cases of β-thalassemia major receiving regular blood transfusion therapy were included in the study. Patients attending the pediatric hematology clinic and transfusion unit during the study period were enrolled consecutively after fulfilling the eligibility criteria. Demographic and clinical details, including age, sex, duration of disease, frequency of blood transfusions, and history of iron chelation therapy, were recorded using a predesigned proforma.

Inclusion Criteria

The following participants were included in the study: patients with a confirmed diagnosis of β-thalassemia major based on clinical and hematological findings. Patients receiving regular blood transfusion therapy. Patients aged 5 years and above. Patients attending follow-up visits during the study period. Patients or guardians willing to provide written informed consent.

Exclusion criteria

The following patients were excluded from the study: patients with acute or chronic infectious diseases. Patients with chronic inflammatory disorders. Patients with chronic liver disease unrelated to transfusion-associated iron overload. Patients with pre-existing renal disorders unrelated to β-thalassemia. Patients with malignancy or severe systemic illness. Patients unwilling to participate in the study.

Sample collection

Under strict aseptic precautions, approximately 5 ml of venous blood was collected from each participant using sterile disposable syringes. Blood samples were transferred into plain sterile vacutainer tubes and allowed to clot at room temperature. Serum was separated by centrifugation at 3000 rpm for 10 minutes and subsequently used for biochemical investigations. Hemolyzed samples were excluded from analysis to avoid analytical interference.

Biochemical analysis

Serum ferritin levels were estimated using the chemiluminescence immunoassay (CLIA) method, a highly sensitive and specific technique for assessing body iron stores.

Liver function parameters, including alanine aminotransferase (ALT/SGPT), aspartate aminotransferase (AST/SGOT), total bilirubin, total protein, and serum albumin, were measured using standard enzymatic and colorimetric methods on a fully automated biochemistry analyser.

Renal function assessment included estimation of serum creatinine and blood urea using commercially available diagnostic reagent kits according to the manufacturers' instructions and standard laboratory protocols. Internal quality control procedures were performed throughout the study period to ensure analytical accuracy, precision, and reliability of biochemical measurements.

Ethical approval

The study was conducted as part of the Ph.D. research work registered with Malwanchal University, Indore, Madhya Pradesh, vide Registration Ref. No. MU/Acm/Ph.D./Med BioChem/2024/002 dated 21 February 2025. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and institutional ethical guidelines. Written informed consent was obtained from all adult participants and from parents or legal guardians of pediatric participants before enrolment in the study.

Statistical analysis

Data were compiled, coded, and analyzed using the Statistical Package for the Social Sciences (SPSS) software version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Pearson's correlation coefficient was used to evaluate relationships among selected biochemical parameters. Student's t-test was applied to compare continuous variables wherever appropriate. A p value of less than 0.05 was considered statistically significant.

RESULTS

A total of 100 patients with transfusion-dependent β-thalassemia major were enrolled in the present study. The age distribution of the participants is presented in Table 1. The majority of patients belonged to the 30-39-year age

group (29.0%), followed by the 10-19-year (25.0%) and 20-29-year (18.0%) age groups. The demographic and clinical characteristics are summarized in Table 1. The study population comprised 51 males (51.0%) and 49 females (49.0%). More than half of the participants (52.0%) had a disease duration of less than 10 years, while all patients were receiving regular blood transfusions at intervals of 3-4 weeks.

Table 1: Demographic and clinical characteristics of transfusion-dependent β-thalassemia major patients (n=100).

Variables	Category	Frequency (%)
Age group (years)	0-9	12 (12.0)
	10-19	25 (25.0)
	20-29	18 (18.0)
	30-39	29 (29.0)
	≥40	16 (16.0)
Gender	Male	51 (51.0)
	Female	49 (49.0)
Duration of disease	<10 years	52 (52.0)
	10-19 years	24 (24.0)
	20-29 years	17 (17.0)
	30-39 years	7 (7.0)
Transfusion frequency	Every 3-4 weeks	100 (100.0)

Table 2: Distribution of iron chelation therapy among transfusion-dependent β-thalassemia major patients (n=100).

Chelation regimen	Frequency (%)
Deferasirox	39 (39.0)
Deferiprone	5 (5.0)
Deferoxamine	8 (8.0)
Deferasirox + deferiprone	7 (7.0)
Deferiprone + deferoxamine	13 (13.0)
Deferasirox + deferoxamine	14 (14.0)
No chelation therapy	14 (14.0)
Total	100 (100.0)

The pattern of iron chelation therapy is shown in Table 2. Deferasirox monotherapy was the most frequently prescribed regimen (39.0%), followed by combination therapy with deferasirox plus deferoxamine (14.0%) and deferiprone plus deferoxamine (13.0%). Deferoxamine and deferiprone monotherapy were administered to 8.0% and 5.0% of patients, respectively, whereas 7.0% received a combination of deferasirox and deferiprone. Fourteen patients (14.0%) were not receiving iron chelation therapy at the time of enrolment.

Serum ferritin analysis demonstrated a substantial burden of iron overload among the study participants (Table 3). The mean serum ferritin concentration was 2701.48±1393.83 ng/ml, with values ranging from 601.4 to 4945.9 ng/ml. Nearly half of the patients (46.0%) had serum ferritin levels exceeding 2500 ng/ml, while 40.0% had concentrations between 1000 and 2500 ng/ml. Only 14.0% of participants-maintained serum ferritin levels below 1000 ng/ml.

Table 3: Serum ferritin concentration and distribution among transfusion-dependent β-thalassemia major patients (n=100).

Parameters	Value
Mean serum ferritin (ng/ml)	2701.48±1393.83
Range (ng/ml)	601.4-4945.9
<1000 ng/ml	14 (14.0)
1000-2500 ng/ml	40 (40.0)
>2500 ng/ml	46 (46.0)

Table 4: Hepatic and renal function parameters among transfusion-dependent β-thalassemia major patients (n=100).

Parameters	Mean±SD	Abnormal (%)
ALT (U/l)	60.55±20.62	60 (60.0)
AST (U/l)	59.90±32.54	72 (72.0)
ALP (U/l)	134.08±35.43	33 (33.0)
Total bilirubin (mg/dl)	1.34±0.42	55 (55.0)
Albumin (gm/dl)	4.35±0.58	0 (0.0)
Serum creatinine (mg/dl)	1.018±0.285	17 (17.0)
Blood urea nitrogen (mg/dl)	29.56±11.23	13 (13.0)
eGFR (ml/minute/1.73 m^2)	86.84±12.38	35 (35.0)

The hepatic biochemical profile is presented in Table 4. The mean serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin, and albumin levels were 60.55±20.62 U/l, 59.90±32.54 U/l, 134.08±35.43 U/l, 1.34±0.42 mg/dl, and 4.35±0.58 gm/dl, respectively. Elevated AST levels were observed in 72.0% of patients, followed by elevated ALT (60.0%), total bilirubin (55.0%), and ALP (33.0%). Serum albumin concentrations remained within the normal reference range in all participants.

Renal function parameters are summarized in Table 4. The mean serum creatinine concentration was 1.018±0.285 mg/dl, the mean blood urea nitrogen concentration was 29.56±11.23 mg/dl, and the mean estimated glomerular filtration rate (eGFR) was 86.84±12.38 ml/minute/1.73 m^2. Elevated serum creatinine and blood urea nitrogen levels were identified in 17.0% and 13.0% of patients, respectively, while reduced eGFR was observed in 35.0% of the study population, indicating the presence of renal function abnormalities in a considerable proportion of patients.

Correlation analysis between serum ferritin and selected biochemical parameters is presented in Table 5. Serum ferritin demonstrated a weak but statistically significant negative correlation with serum ALT (r=−0.227, p=0.023) and total bilirubin (r=−0.273, p=0.006). No statistically significant correlation was observed between serum ferritin and serum creatinine (r=−0.113, p=0.261).

Table 5: Correlation of serum ferritin with hepatic and renal biochemical parameters.

Parameters	Pearson's r	P value
Ferritin versus ALT	−0.227	0.023*
Ferritin versus total bilirubin	−0.273	0.006*
Ferritin versus serum creatinine	−0.113	0.261

Pearson's correlation coefficient was used. *P<0.05 was considered statistically significant. ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate.

Overall, the findings demonstrate a high burden of transfusional iron overload in patients with transfusion-dependent β-thalassemia major, accompanied by frequent hepatic biochemical abnormalities and evidence of early renal dysfunction.

DISCUSSION

The present study evaluated the association between serum ferritin levels and hepatic and renal function parameters among patients with transfusion-dependent β-thalassemia major. Despite remarkable improvements in transfusion practices and iron chelation therapy, transfusional iron overload continues to represent a major determinant of long-term morbidity in these patients. The mean serum ferritin concentration in the present study was 2701.48±1393.83 ng/ml, with nearly half of the participants (46%) demonstrating serum ferritin levels above 2500 ng/ml (Table 3). These findings indicate persistent iron overload despite regular chelation therapy and are consistent with the Thalassaemia International Federation (TIF) guidelines, which identify sustained elevation of serum ferritin as an important marker of iron burden and risk of progressive organ injury in transfusion-dependent β-thalassemia.[1,2]

The demographic profile of the study cohort reflects the long-term survival of patients with β-thalassemia major. Most participants (88%) were aged ≥10 years, with an almost equal distribution between males and females (Table 1). This age distribution is consistent with the increasing recognition of β-thalassemia as a chronic condition requiring lifelong monitoring, reflecting advances in transfusion support, iron chelation, and multidisciplinary care.[3,4,14] The increasing availability of effective long-term management has contributed to improved survival, while epidemiological studies continue to demonstrate substantial variation in the prevalence and clinical burden of thalassemia across different geographic regions.[5,6]

Iron chelation remains the cornerstone of preventing transfusion-related iron toxicity. In the present study, deferasirox monotherapy was the most commonly prescribed regimen, followed by combination therapy with deferasirox and deferoxamine (Table 2). Comparative evidence supports the efficacy of deferasirox and its role in the management of transfusional iron overload.[26,27] Long-term oral deferiprone therapy has also demonstrated efficacy and acceptable safety in selected patients with transfusion-dependent thalassemia.[28] The broader management of thalassemia requires individualized long-term monitoring and treatment of disease-related complications.[29] Long-term safety and efficacy data in pediatric patients further support the role of deferasirox in transfusion-dependent β-thalassemia.[30] Combination chelation therapy may be considered in patients with substantial iron overload or inadequate response to individual chelators. Studies evaluating deferasirox-deferoxamine combination therapy have demonstrated potential benefits in patients with significant iron burden, including effects on cardiac iron overload.[31,32]

Iron-induced hepatic injury remains one of the earliest and most frequent complications of transfusion-dependent β-thalassemia. In the present study, elevated liver enzymes and bilirubin were observed in a substantial proportion of patients (Table 4), indicating biochemical evidence of hepatic involvement. Chronic iron deposition within hepatocytes promotes oxidative stress, cellular injury, inflammatory responses, and progressive fibrosis, ultimately increasing the risk of cirrhosis and hepatic failure if iron burden is inadequately controlled.[15,16] Similar evidence from studies evaluating hepatic iron burden and serum ferritin supports the importance of monitoring hepatic involvement in patients with transfusion-dependent thalassemia.[19,23]

An important observation in the present study was the preservation of serum albumin concentrations despite abnormalities in liver enzymes and bilirubin. This finding suggests that hepatic synthetic function remained largely preserved, although biochemical evidence of hepatocellular injury was already present. The coexistence of biochemical abnormalities with preserved albumin concentrations may indicate an earlier stage of hepatic involvement rather than advanced hepatic synthetic dysfunction. Therefore, regular biochemical surveillance may help identify hepatic abnormalities before clinically significant hepatic failure develops.

Renal dysfunction is increasingly recognized as an important complication of transfusion-dependent β-thalassemia. In the present study, abnormalities in serum creatinine, blood urea nitrogen, and estimated glomerular filtration rate were identified in a considerable proportion of patients (Table 4). Renal injury in β-thalassemia is multifactorial and may result from chronic anemia, recurrent hypoxia, oxidative stress, iron deposition, and the cumulative effects of prolonged chelation therapy. Multicenter evidence describing complications among

patients with transfusion-dependent thalassemia supports the importance of recognizing renal and other organ involvement during long-term follow-up.[22,34]

Correlation analysis demonstrated weak but statistically significant negative associations between serum ferritin and serum ALT as well as total bilirubin, whereas no significant relationship was observed with serum creatinine (Table 5). Although serum ferritin remains a widely used surrogate marker of body iron stores, organ dysfunction in transfusion-dependent β-thalassemia is influenced by several additional factors, including transfusion intensity, duration of disease, inflammation, liver disease, adherence to chelation therapy, and individual susceptibility to iron-mediated injury.[17,18] Consequently, serum ferritin should be interpreted in conjunction with clinical findings and biochemical assessment rather than as an isolated indicator of tissue iron burden.[19,20]

Evidence from other chronically transfused populations also suggests that serum ferritin may not always show a linear relationship with tissue iron burden or hepatic injury. In children with sickle cell disease receiving chronic transfusion therapy, changes in serum ferritin were reported to be nonlinear and associated with iron load and liver injury.[33] Although these findings arise from a different disease population, they further support cautious interpretation of serum ferritin as a surrogate marker and the need for complementary assessment of organ involvement.

From a clinical perspective, the present findings support the continued use of serum ferritin as an accessible and cost-effective marker for monitoring iron overload, particularly in resource-limited settings where MRI-based assessment of liver and cardiac iron concentration may not be routinely available. Although MRI-based techniques provide important information regarding tissue-specific iron deposition, serial serum ferritin estimation combined with periodic evaluation of hepatic and renal function offers a practical approach to routine monitoring and identification of patients who may require optimization of iron-chelation therapy.[19-21,24,25]

The long-term clinical significance of iron overload is also supported by cohort data demonstrating overall and complication-free survival patterns among patients with β-thalassemia major followed over extended periods.[35] These findings further emphasize the importance of sustained monitoring and prevention of transfusion-related complications.

The present study provides a comprehensive evaluation of serum ferritin together with hepatic and renal function parameters in a well-characterized cohort of transfusion-dependent β-thalassemia major patients. Simultaneous assessment of iron burden and organ function enhances the clinical relevance of the findings, particularly in resource-constrained settings where serum ferritin remains a practical biomarker for routine monitoring. However, the cross-sectional design limits causal inference; the study was conducted at a single tertiary care center; and MRI-based quantification of tissue iron was not performed. In addition, serum ferritin may be influenced by inflammatory conditions and may not always accurately reflect total body iron stores. Evidence from other chronically transfused populations also indicates that ferritin changes may not directly correspond to iron burden or hepatic injury.[33] Future multicenter prospective studies incorporating MRI-derived liver and cardiac iron measurements and long-term follow-up are warranted to validate these findings and better define the relationship between iron overload and organ dysfunction.

CONCLUSION

The present study demonstrates a considerable burden of iron overload among patients with transfusion-dependent β-thalassemia major, accompanied by frequent hepatic and renal biochemical abnormalities despite regular transfusion support and iron-chelation therapy. Nearly half of the participants had serum ferritin concentrations >2500 ng/ml. Hepatic abnormalities, particularly elevated AST, ALT, alkaline phosphatase, and total bilirubin, were common, while reduced eGFR and elevated serum creatinine and blood urea nitrogen indicated renal involvement in a substantial proportion of patients. Serum albumin remained within the normal range, suggesting preserved hepatic synthetic function. The weak associations between serum ferritin and selected hepatic parameters, together with the absence of a significant correlation with serum creatinine, indicate that ferritin alone may not adequately reflect organ involvement. Therefore, serum ferritin should be interpreted alongside hepatic and renal biochemical parameters, with regular surveillance and individualized optimization of iron-chelation therapy. Multicenter prospective studies incorporating MRI-based tissue iron assessment and longitudinal follow-up are warranted.

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