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Association of Serum Ferritin Levels with Hepatic and Renal Function Parameters in Transfusion-Dependent β-Thalassemia Major Patients: A Cross-Sectional Study

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ABSTRACT

Background

β-thalassemia major is a severe inherited hemoglobinopathy characterized by lifelong transfusion dependence. Repeated blood transfusions lead to progressive iron overload, which contributes to hepatic, renal, endocrine, and cardiovascular complications. Serum ferritin is widely used as an accessible surrogate marker of body iron stores and remains an important tool for monitoring iron burden in clinical practice.

Objective

To evaluate serum ferritin levels and assess hepatic and renal function parameters in transfusion-dependent β-thalassemia major patients.

Materials and Methods

A hospital-based cross-sectional observational study was conducted among 100 transfusion-dependent β-thalassemia major patients attending a tertiary care center. Clinical and demographic data were recorded. Serum ferritin levels, liver function parameters [alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), bilirubin, and albumin], and renal function parameters [serum creatinine, blood urea nitrogen (BUN), and estimated glomerular filtration rate (eGFR)] were evaluated using standard laboratory methods. Data were analyzed using descriptive statistics and Pearson's correlation analysis.

Results

The mean serum ferritin concentration was 2701.48 ± 1393.83 ng/mL, and 46% of patients exhibited ferritin levels greater than 2500 ng/mL, indicating significant iron overload. Elevated AST, ALT, bilirubin, and ALP levels were observed in 72%, 60%, 55%, and 33% of patients, respectively. Reduced eGFR was identified in 35% of participants, while abnormal serum creatinine and BUN levels were observed in 17% and 13% of patients, respectively. Correlation analysis demonstrated a strong positive association between serum creatinine and BUN (r = 0.817) and strong negative correlations between eGFR and both serum creatinine (r = −0.730) and BUN (r = −0.741).

Conclusion

Transfusion-dependent β-thalassemia major patients exhibit a substantial burden of iron overload accompanied by significant hepatic and renal abnormalities. Regular monitoring of serum ferritin together with liver and renal function parameters may facilitate early detection of organ dysfunction and support appropriate long-term management strategies.

Keywords

β-thalassemia major; serum ferritin; iron overload; liver function tests; renal function; organ dysfunction; transfusion-dependent thalassemia.

INTRODUCTION

β-thalassemia major is one of the most severe and prevalent inherited hemoglobinopathies worldwide, posing a substantial public health burden, particularly in regions with high carrier frequencies and limited access to specialized healthcare services. Characterized by mutations in the β-globin gene, the disorder results in absent or markedly reduced β-globin chain synthesis, leading to ineffective erythropoiesis, chronic hemolytic anemia, and lifelong transfusion dependence beginning in early childhood. Despite significant advances in supportive care, β-thalassemia major continues to be associated with considerable morbidity and impaired quality of life because of its chronic nature and multisystem complications.[10,21]

Over the past few decades, regular blood transfusion therapy combined with iron chelation has transformed β-thalassemia major from a rapidly fatal childhood disease into a chronic condition compatible with long-term survival.[9,27] While these therapeutic advances have substantially improved life expectancy, they have also shifted clinical attention toward the prevention and management of transfusion-related complications, particularly iron overload. Because the human body lacks an effective physiological mechanism for excreting excess iron, repeated transfusions result in progressive iron accumulation that eventually exceeds the storage capacity of physiological iron-binding proteins.[7,9] Excess iron is subsequently deposited in vital organs, including the liver, heart, endocrine glands, and kidneys, where it generates reactive oxygen species, leading to cellular injury, tissue fibrosis, and progressive organ dysfunction.[7,25]

Among the organs affected by iron overload, the liver serves as the primary reservoir for excess iron and is often the earliest organ to demonstrate evidence of iron-induced toxicity. Hepatic iron deposition promotes oxidative stress, inflammation, hepatocellular injury, and fibrosis, which may ultimately progress to cirrhosis and liver failure if inadequately controlled.[12,28] Elevated liver enzymes are frequently encountered in transfusion-dependent patients and may serve as early biochemical indicators of hepatic involvement. Given the central role of the liver in iron metabolism, assessment of hepatic function remains an essential component of long-term monitoring in β-thalassemia major.[12]

In recent years, increasing attention has also been directed toward renal involvement in β-thalassemia major. Historically considered less prominent than cardiac or hepatic complications, renal dysfunction is now recognized as an important contributor to disease

burden. Chronic anemia, persistent hypoxia, iron-mediated oxidative injury, hemosiderin deposition, and long-term iron chelation therapy have all been implicated in the development of glomerular and tubular abnormalities.[5,34] These pathophysiological mechanisms may result in subtle renal impairment that often remains clinically silent until significant functional deterioration has occurred. Consequently, regular evaluation of renal function has become increasingly important in the comprehensive management of transfusion-dependent thalassemia patients.

Accurate assessment of body iron burden is fundamental to preventing iron-related complications. Although liver biopsy and magnetic resonance imaging (MRI)-based techniques provide reliable quantification of tissue iron stores, their routine use is often limited by cost, availability, and technical requirements, particularly in low- and middle-income countries.[12,14] In contrast, serum ferritin measurement is inexpensive, readily accessible, and widely utilized in routine clinical practice as a surrogate marker of total body iron stores. Elevated serum ferritin concentrations have been associated with an increased risk of organ dysfunction, adverse clinical outcomes, and mortality among transfusion-dependent patients.[1,9,20] Despite its recognized limitations, serum ferritin remains the most practical and frequently employed biomarker for monitoring iron overload and guiding chelation therapy.

Given the lifelong transfusion requirements of patients with β-thalassemia major and the persistent risk of iron-induced organ damage, early identification of hepatic and renal dysfunction is essential for optimizing patient outcomes. However, data describing iron burden and organ function abnormalities among transfusion-dependent β-thalassemia patients in central India remain relatively limited. A better understanding of these clinical and biochemical alterations may facilitate earlier recognition of complications and contribute to more effective disease-monitoring strategies.

Therefore, the present study was undertaken to evaluate serum ferritin levels and assess hepatic and renal function parameters among transfusion-dependent β-thalassemia major patients. By examining the burden of iron overload and associated biochemical abnormalities, this study aims to provide clinically relevant evidence that may assist in improving the long-term monitoring and management of this vulnerable patient population.

MATERIALS AND 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.

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 & Research Centre, Indore, Madhya Pradesh, India. The study was carried out over a period of one year from 24 February 2025 to 22 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:

1. Patients with a confirmed diagnosis of β-thalassemia major based on clinical and hematological findings.
2. Patients receiving regular blood transfusion therapy.
3. Patients aged 5 years and above.
4. Patients attending follow-up visits during the study period.
5. Patients or guardians willing to provide written informed consent.

Exclusion Criteria

The following patients were excluded from the study:

1. Patients with acute or chronic infectious diseases.
2. Patients with chronic inflammatory disorders.
3. Patients with chronic liver disease unrelated to transfusion-associated iron overload.
4. Patients with pre-existing renal disorders unrelated to β-thalassemia.
5. Patients with malignancy or severe systemic illness.
6. 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. Hemolysed 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 under 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 analysed using 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 for comparison of continuous variables wherever appropriate. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 100 transfusion-dependent β-thalassemia major patients were included in the study. The age distribution of the participants is presented in Table 1 and Figure 1. The highest proportion of patients belonged to the 30–39 years age group (29%), followed by the 10–19 years (25%) and 20–29 years (18%) age groups. Demographic and clinical characteristics are summarized in Table 2 and Figure 2. The study cohort demonstrated a nearly equal gender distribution, comprising 51% males and 49% females. 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.

The pattern of iron chelation therapy among study participants is shown in Table 3 and Figure 3. Deferasirox monotherapy was the most frequently prescribed chelation regimen, accounting for 45.3% of patients. Combination therapies, including deferasirox plus deferoxamine (16.3%) and deferiprone plus deferoxamine (15.1%), were also commonly utilized, whereas deferiprone monotherapy was the least frequently prescribed regimen (5.8%).

Assessment of iron burden revealed persistently elevated serum ferritin levels in a substantial proportion of patients (Tables 4 and 5; Figure 4). The mean serum ferritin concentration was 2701.48 ± 1393.83 ng/mL, with values ranging from 601.4 ng/mL to 4945.9 ng/mL. Nearly half of the study population (46.0%) exhibited serum ferritin levels exceeding 2500 ng/mL, indicating significant iron overload, whereas only 14.0% maintained ferritin concentrations below 1000 ng/mL.

Liver function parameters are presented in Table 6 and Figure 5. The mean serum ALT, AST, 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 g/dL, respectively. Elevated AST levels were observed in 72.0% of patients, followed by elevated ALT (60.0%), bilirubin (55.0%), and ALP (33.0%). Serum albumin concentrations remained within the normal range in all patients.

Renal function assessment is summarized in Table 7 and Figure 6. The mean serum creatinine level was 1.018 ± 0.285 mg/dL, while the mean blood urea nitrogen (BUN) level was 29.56 ± 11.23 mg/dL. The mean estimated glomerular filtration rate (eGFR) was 86.84 ± 12.38 mL/min/1.73 m². Abnormal serum creatinine and BUN values were observed in 17.0% and 13.0% of patients, respectively, whereas reduced eGFR values were identified in 35.0% of participants.

The relationships between serum ferritin and selected biochemical parameters were evaluated using Pearson's correlation analysis (Table 8; Figure 7). Serum ferritin demonstrated a weak but statistically significant negative correlation with 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).

Overall, the findings demonstrate a substantial burden of iron overload (Tables 4 and 5; Figure 4) accompanied by frequent hepatic abnormalities (Table 6; Figure 5) and renal dysfunction (Table 7; Figure 6) among transfusion-dependent β-thalassemia major patients.

DISCUSSION

The present study evaluated the relationship between iron overload, as assessed by serum ferritin levels, and hepatic and renal function parameters in transfusion-dependent β-thalassemia major patients. Despite substantial advances in transfusion support and iron chelation therapy, iron overload remains a major contributor to morbidity and long-term complications in this population. In the present study, the mean serum ferritin concentration was 2701.48 ± 1393.83 ng/mL, and nearly half of the patients (46%) exhibited ferritin levels exceeding 2500 ng/mL (Tables 4 and 5; Figure 4), indicating a considerable burden of iron accumulation. These findings are consistent with previous studies by Cappellini et al., Cohen et al., and Borgna-Pignatti et al., which demonstrated that chronic transfusional iron overload continues to be highly prevalent despite regular chelation therapy and remains a major determinant of organ dysfunction and adverse clinical outcomes.[9,20,23,27]

The demographic profile of the study population revealed a predominance of adolescents and adults with a nearly equal distribution of males and females (Table 1; Figure 1). These findings reflect the significant improvement in survival achieved through advances in transfusion practices, iron chelation, and comprehensive supportive care. Similar demographic trends have been reported worldwide, where thalassemia has evolved from a predominantly pediatric disorder into a chronic disease requiring lifelong multidisciplinary management.[10,27]

Iron chelation therapy remains the cornerstone of preventing iron-mediated tissue injury. In the present study, deferasirox monotherapy was the most commonly prescribed chelation regimen, followed by combination therapies involving deferoxamine and deferiprone (Table 3; Figure 3). Similar treatment patterns have been reported in multicenter studies evaluating long-term chelation strategies, which have demonstrated the effectiveness of deferasirox-based regimens in improving iron control and clinical outcomes.[9,20,24]

The liver is the principal site of iron storage and is particularly susceptible to iron-mediated oxidative injury. In the current study, elevated ALT, AST, ALP, and bilirubin levels were observed in 60%, 72%, 33%, and 55% of patients, respectively (Table 6; Figure 5), suggesting substantial hepatic involvement. Excess iron promotes the generation of reactive oxygen species, leading to lipid peroxidation, hepatocellular injury, inflammation, and progressive fibrosis. Similar observations have been reported by Angelucci et al. and Brittenham et al., who demonstrated a strong association between iron burden and hepatic dysfunction in transfusion-dependent patients.12,28 The high prevalence of abnormal liver function tests observed in the present study underscores the importance of routine hepatic surveillance and timely optimization of chelation therapy.

An important observation was the preservation of serum albumin levels despite elevations in liver enzymes and bilirubin (Table 6). This finding suggests that hepatic synthetic function remained relatively preserved despite evidence of hepatocellular injury. Similar observations have been reported in previous studies, indicating that biochemical evidence of liver injury often precedes clinically significant impairment of hepatic synthetic capacity.[12,28]

Renal abnormalities were also frequently observed among the study participants. Elevated serum creatinine and BUN levels were identified in 17% and 13% of patients, respectively, while reduced eGFR was present in 35% of cases (Table 7; Figure 6). These findings suggest

that renal dysfunction represents an important yet often underrecognized complication of transfusion-dependent β-thalassemia major. The pathogenesis of renal injury is multifactorial and may involve chronic anemia, tissue hypoxia, iron deposition, oxidative stress, tubular dysfunction, and the potential nephrotoxic effects of prolonged chelation therapy. Similar findings have been reported by Al-Kuraishy and Al-Gareeb and by Mandal et al.[5,34]

Correlation analysis revealed weak but statistically significant associations between serum ferritin and selected hepatic biochemical parameters (Table 8; Figure 7). Serum ferritin demonstrated a significant negative correlation with ALT and total bilirubin, whereas no significant association was observed with serum creatinine. Although serum ferritin remains a practical surrogate marker of body iron stores, these findings suggest that biochemical evidence of hepatic and renal dysfunction may be influenced by multiple factors beyond iron burden alone, including chronic anemia, oxidative stress, transfusion history, and individual response to chelation therapy.

Serum ferritin remains one of the most widely utilized indicators of body iron stores because of its accessibility, affordability, and practicality in routine clinical practice. Although ferritin concentrations may be influenced by inflammatory states and hepatic disease, serum ferritin continues to serve as an important surrogate marker of iron burden, particularly in resource-limited settings where advanced techniques such as MRI T2* assessment may not be readily available.[7,12,14] The persistently elevated ferritin levels observed in the present study (Tables 4 and 5; Figure 4) reinforce the importance of regular ferritin monitoring for identifying patients at increased risk of iron-related complications and guiding individualized chelation therapy.

The clinical implications of the present findings are substantial. Routine assessment of serum ferritin levels together with liver function tests (Table 6; Figure 5) and renal function parameters (Table 7; Figure 6) may facilitate the early detection of organ dysfunction and enable timely therapeutic intervention. Such an approach may help minimize irreversible tissue damage, preserve organ function, and improve long-term survival among patients with transfusion-dependent β-thalassemia major.[9,20,25]

Nevertheless, several limitations should be acknowledged. The cross-sectional design limits the ability to establish causal relationships between iron overload and organ dysfunction. In addition, serum ferritin was used as the primary marker of iron burden without complementary assessment using MRI-derived liver or cardiac iron quantification. Furthermore, the study was conducted at a single tertiary care center, which may limit the generalizability of the findings. Future multicenter longitudinal studies incorporating advanced imaging modalities and serial assessment of organ function are warranted to further clarify the relationship between iron overload and organ-specific complications.[12,14]

Overall, the present findings reinforce the central role of iron overload in the development of hepatic and renal complications among transfusion-dependent β-thalassemia major patients. The substantial burden of iron accumulation observed in the present study (Tables 4–7; Figures 4–6) highlights the importance of vigilant long-term monitoring, individualized chelation strategies, and multidisciplinary care aimed at minimizing organ-related complications and improving patient outcomes.[9,20,25,27]

Strengths of the Study

The present study comprehensively evaluated serum ferritin levels together with hepatic and renal function parameters in transfusion-dependent β-thalassemia major patients. Simultaneous assessment of iron burden and organ function provides clinically relevant information regarding the spectrum of transfusion-related complications. The study findings are readily applicable to routine clinical practice, particularly in resource-limited settings where serum ferritin remains the most accessible marker of iron overload.

Limitations

The cross-sectional design precludes assessment of temporal relationships and causality. The study was conducted at a single tertiary care center, which may limit generalizability. Serum ferritin was used as the primary marker of iron burden and may be influenced by inflammatory conditions. Furthermore, advanced imaging techniques such as MRI T2* were not utilized, and longitudinal follow-up was not performed

CONCLUSION

Transfusion-dependent β-thalassemia major continues to pose a significant clinical challenge owing to the lifelong requirement for regular blood transfusions and the consequent risk of progressive iron overload. In the present study, a substantial proportion of patients exhibited persistently elevated serum ferritin levels, with nearly half demonstrating ferritin concentrations exceeding 2500 ng/mL (Tables 4 and 5; Figure 4), despite ongoing iron chelation therapy. These findings indicate that iron overload remains a prevalent concern and continues to contribute to long-term disease-related complications.

The study further revealed a high prevalence of hepatic and renal abnormalities among transfusion-dependent patients. Elevated liver enzymes and bilirubin levels (Table 6; Figure 5), together with abnormalities in renal function parameters, including reduced eGFR (Table 7; Figure 6), suggest ongoing organ involvement secondary to chronic iron accumulation.

An important finding of the present study was the persistence of biochemical evidence of organ dysfunction despite the widespread use of iron chelation therapy (Table 3; Figure 3). This highlights the complexity of iron overload management and suggests that iron-mediated tissue injury may continue to occur even in adequately treated patients. Consequently, individualized chelation strategies and closer therapeutic monitoring may be required to optimize long-term outcomes and minimize organ-related complications.

Serum ferritin was found to be a practical and clinically informative marker of iron burden, with elevated levels corresponding to the high prevalence of hepatic and renal abnormalities observed in the study population (Tables 4–7; Figures 4–6). Although advanced techniques such as MRI-based iron quantification provide more precise assessment of tissue iron stores, serum ferritin remains an accessible, cost-effective, and widely available tool for routine monitoring, particularly in resource-limited settings.

The findings of this study underscore the importance of a comprehensive and multidisciplinary approach to the long-term management of transfusion-dependent β-thalassemia major. Regular assessment of serum ferritin levels, liver function tests, and renal function parameters should be incorporated into standard follow-up protocols to facilitate the early detection of organ dysfunction and guide timely therapeutic interventions. Such measures may help reduce the burden of iron-related complications, preserve organ function, and improve overall patient outcomes.

In conclusion, persistent iron overload remains a major determinant of morbidity among patients with transfusion-dependent β-thalassemia major and is associated with significant hepatic and renal dysfunction. The substantial burden of iron overload observed in the present study (Tables 4 and 5; Figure 4) reinforces the need for vigilant monitoring and individualized management strategies. Strengthening surveillance protocols and optimizing chelation therapy according to patient-specific iron burden may contribute significantly to improved clinical outcomes, enhanced quality of life, and better long-term survival in this vulnerable population.

RECOMMENDATIONS

1. Periodic monitoring of serum ferritin levels.
2. Routine liver and renal function assessment.
3. Optimization of iron chelation therapy.
4. Early identification of organ dysfunction.
5. Multicenter longitudinal studies with MRI-based iron quantification.

Conflict of Interest

The authors declare that there are no conflicts of interest related to this study.

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Table 1. Age Distribution of Study Participants (n = 100)

Age Group (Years)	Frequency (n)	Percentage (%)
0–9	12	12.0
10–19	25	25.0
20–29	18	18.0
30–39	29	29.0
≥40	16	16.0
Total	**100**	**100.0**

Table 1
Age distribution of transfusion-dependent β-thalassemia major patients included in the study. The highest proportion of participants belonged to the 30–39 years age group (29%), followed by the 10–19 years (25%) and 20–29 years (18%) age groups, indicating improved survival and continued transfusion support into adulthood.

Table 2. Demographic and Clinical Characteristics of Study Participants (n = 100)

Variable	Frequency (%)
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
Demographic and clinical characteristics of transfusion-dependent β-thalassemia major patients included in the study. The table presents gender distribution, duration of disease, and blood transfusion frequency among study participants.

Table 3. Distribution of Chelation Therapy among Study Participants

Chelation Therapy	Frequency (%)
Deferasirox only	39 (45.3)
Deferiprone only	5 (5.8)
Deferoxamine only	8 (9.3)
Deferasirox + Deferiprone	7 (8.1)
Deferiprone + Deferoxamine	13 (15.1)
Deferasirox + Deferoxamine	14 (16.3)

Table 3: Distribution of iron chelation therapy regimens among transfusion-dependent β-thalassemia major patients. Deferasirox monotherapy was the most frequently prescribed regimen, followed by combination therapies involving deferasirox, deferiprone, and deferoxamine.

Table 4. Serum Ferritin Levels among Study Participants

Parameter	Value
Mean ± SD	2701.48 ± 1393.83 ng/mL
Minimum	601.4 ng/mL
Maximum	4945.9 ng/mL

Table 4: Serum ferritin levels among transfusion-dependent β-thalassemia major patients. The table summarizes the mean, minimum, and maximum serum ferritin concentrations, reflecting the extent of iron overload in the study population.

Table 5. Distribution of Patients According to Ferritin Levels

Ferritin Level (ng/mL)	Frequency (%)
<1000	14 (14.0)
1000–2500	40 (40.0)
>2500	46 (46.0)

Table 5: Distribution of patients according to serum ferritin categories. Serum ferritin levels were categorized to assess the severity of iron overload among transfusion-dependent β-thalassemia major patients.

Table 6. Liver Function Parameters among Study Participants

Parameter	Mean ± SD	Abnormal n (%)
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)
Bilirubin (mg/dL)	1.34 ± 0.42	55 (55.0)
Albumin (g/dL)	4.35 ± 0.58	0 (0.0)

Table 6: Liver function parameters in transfusion-dependent β-thalassemia major patients. The table presents mean biochemical values and the proportion of patients with abnormal results for alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), bilirubin, and albumin.

Table 7. Renal Function Parameters among Study Participants

Parameter	Mean ± SD	Abnormal n (%)
Creatinine (mg/dL)	1.018 ± 0.285	17 (17.0)
BUN (mg/dL)	29.56 ± 11.23	13 (13.0)
eGFR (mL/min/1.73 m²)	86.84 ± 12.38	35 (35.0)

Table 7: Renal function parameters in transfusion-dependent β-thalassemia major patients. The table summarizes the mean values and frequency of abnormal findings for serum creatinine, blood urea nitrogen (BUN), and estimated glomerular filtration rate (eGFR).

Table 8. Correlation Analysis of Biochemical Parameters

Parameters	Pearson's r
ALT vs AST	0.007
ALP vs Bilirubin	-0.038
Creatinine vs BUN	0.817
eGFR vs Creatinine	-0.730
eGFR vs BUN	-0.741

Table 8: Correlation analysis of selected biochemical parameters in transfusion-dependent β-thalassemia major patients. Pearson's correlation coefficients (r) were calculated to evaluate the relationships between liver and renal function markers, including ALT, AST, ALP, bilirubin, creatinine, BUN, and eGFR.

Abbreviations: ALT = alanine aminotransferase; AST = aspartate aminotransferase; ALP = alkaline phosphatase; BUN = blood urea nitrogen; eGFR = estimated glomerular filtration rate.

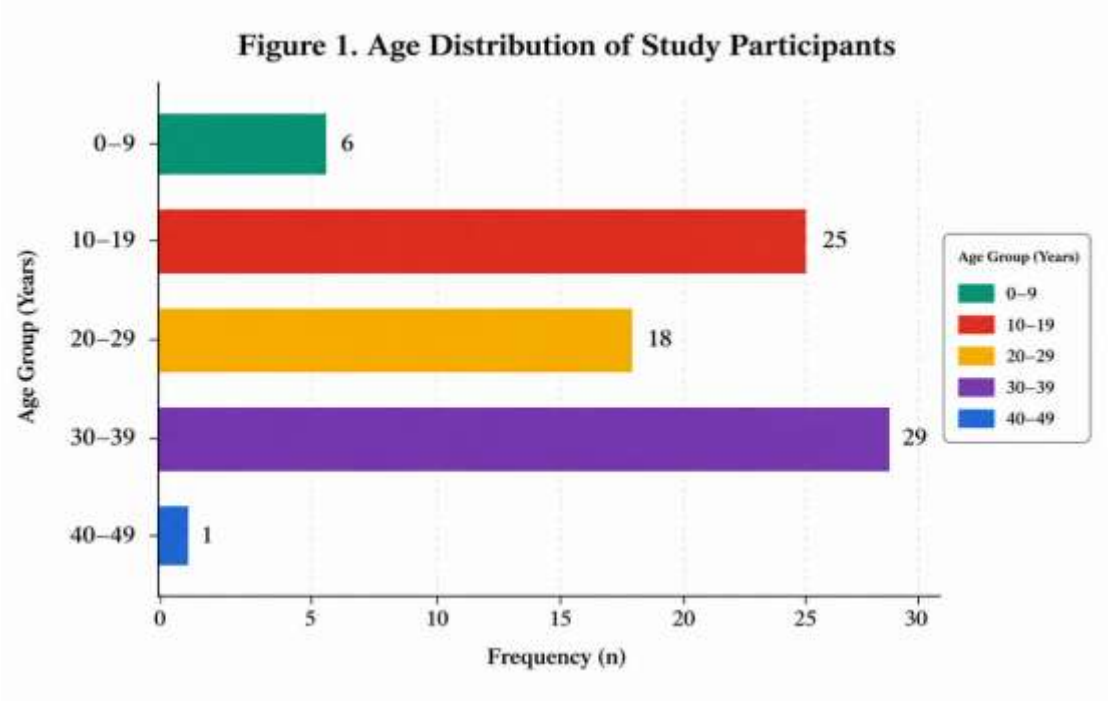

Figure 1: Distribution of study participants according to age group (N = 100). The highest proportion of participants was observed in the 30–39 years age group (29%), followed by 10–19 years (25%) and 20–29 years (18%)

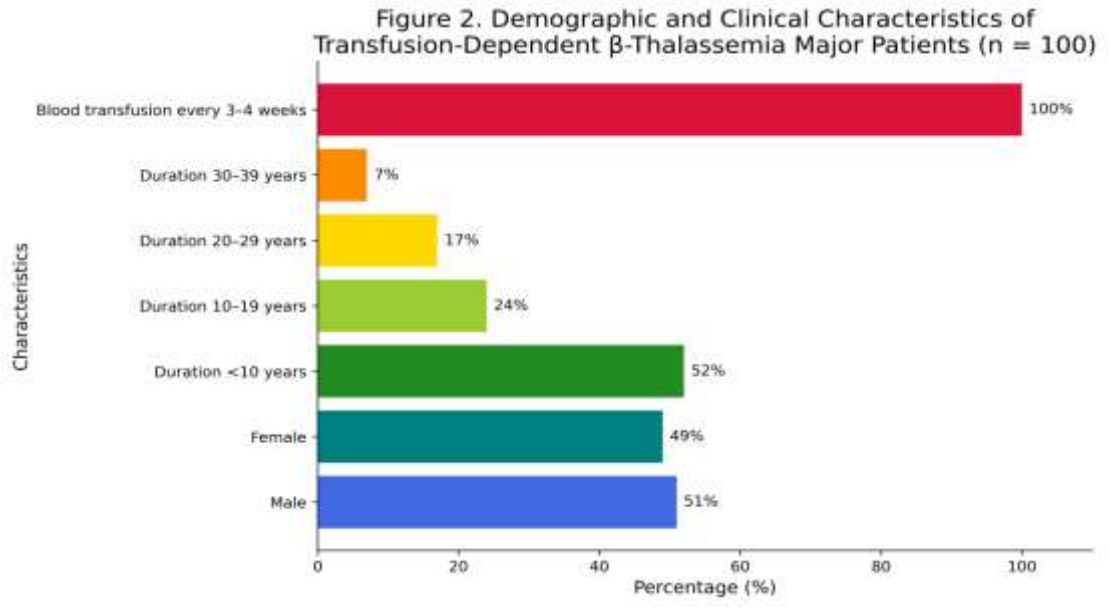

Figure 2. Demographic and clinical characteristics of transfusion-dependent β-thalassemia major patients (n = 100). The cohort showed a nearly equal gender distribution (51% males, 49% females). Most patients had a disease duration of <10 years (52%), and all were receiving regular blood transfusions every 3–4 weeks.

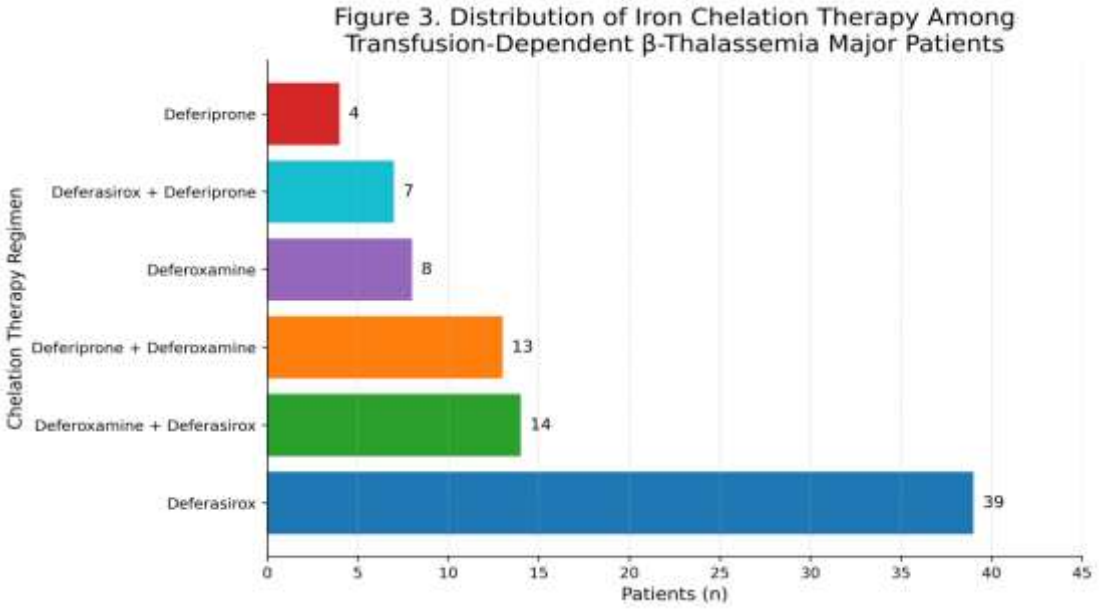

Figure 3. Distribution of iron chelation therapy among transfusion-dependent β-thalassemia major patients. Deferasirox monotherapy was the most frequently prescribed chelation regimen, followed by deferasirox plus deferoxamine and deferiprone plus deferoxamine combination therapies. Deferiprone monotherapy was the least commonly utilized treatment regimen.

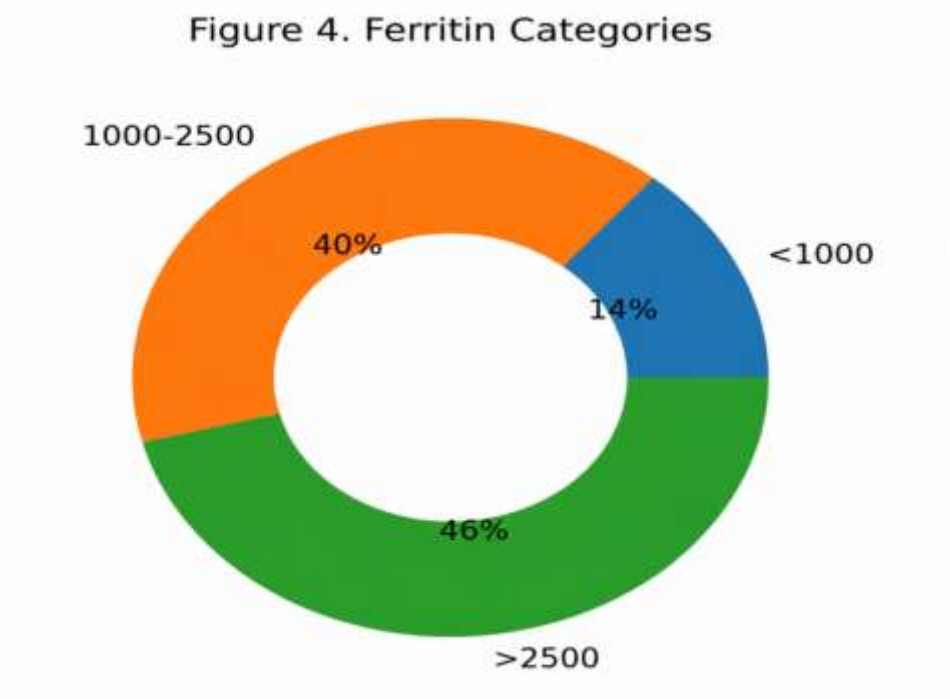

Figure 4. Distribution of serum ferritin categories among transfusion-dependent β-thalassemia major patients. Nearly half of the patients had serum ferritin levels >2500 ng/mL, indicating significant iron overload. Forty percent had ferritin levels between 1000 and 2500 ng/mL, while only 14% had ferritin levels below 1000 ng/mL.

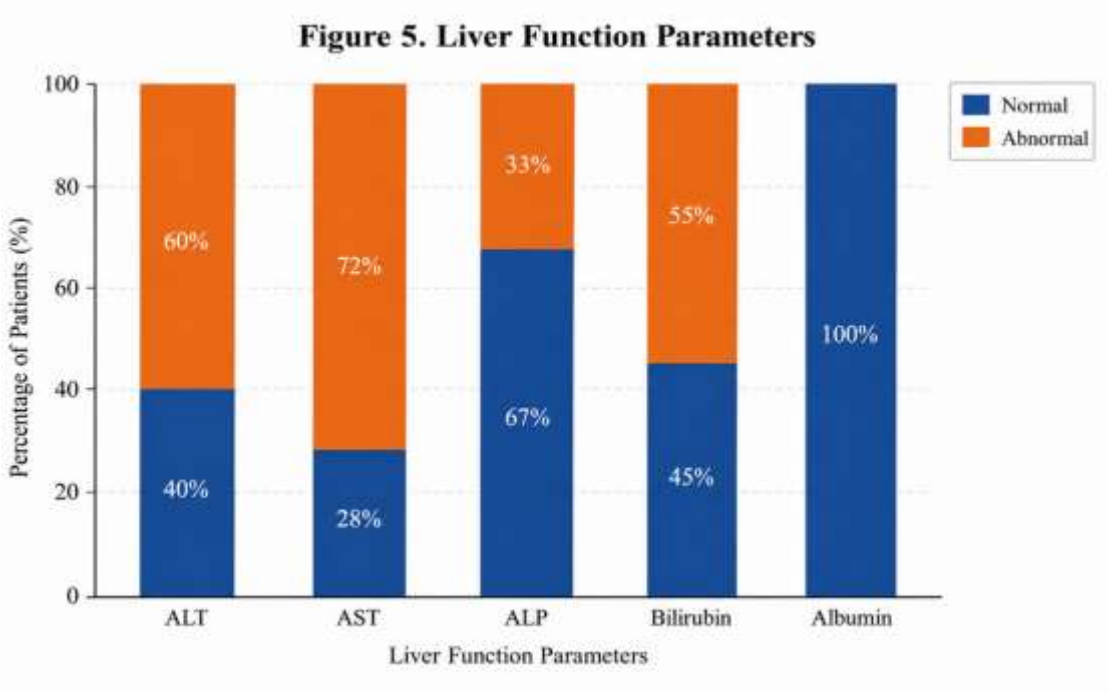

Figure: 5 Distribution of normal and abnormal liver function parameters among transfusion-dependent β-thalassemia major patients. Elevated AST levels were the most common abnormality, followed by increased ALT and bilirubin levels. Albumin levels remained within the normal range in all patients, indicating preserved hepatic synthetic function despite biochemical evidence of liver injury.

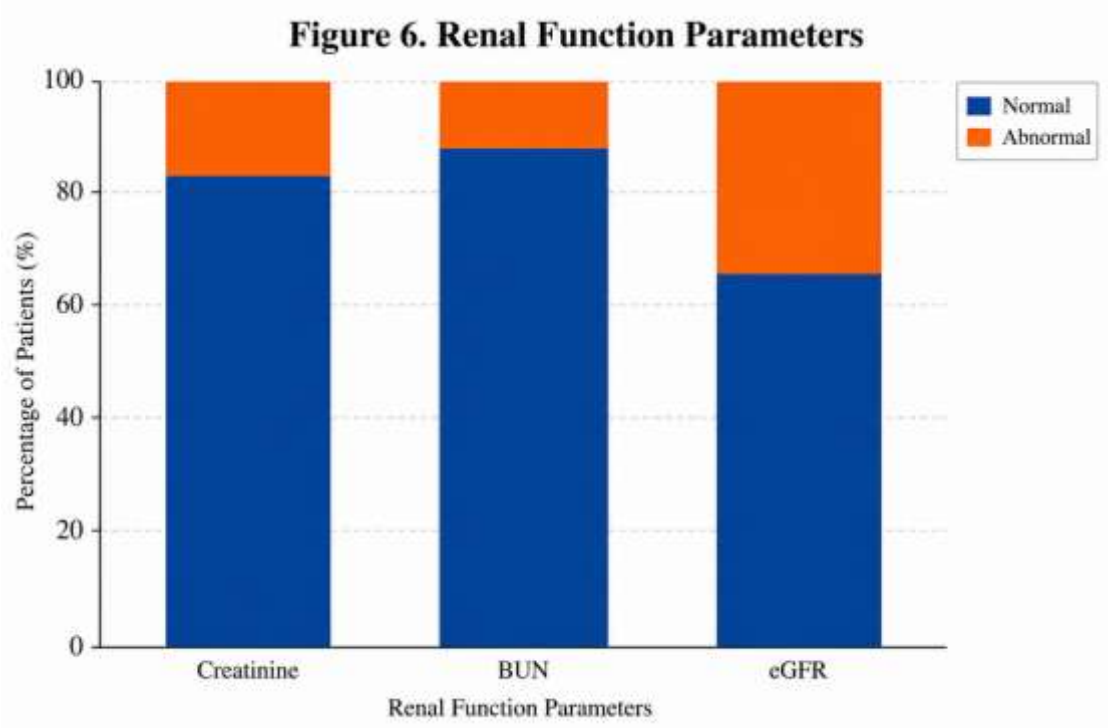

Figure: 6 Distribution of normal and abnormal renal function parameters among transfusion-dependent β-thalassemia major patients. **Reduced eGFR was observed more frequently than abnormal serum creatinine and blood urea nitrogen (BUN) levels, suggesting early renal impairment in a substantial proportion of patients.**

Abbreviations: BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate.

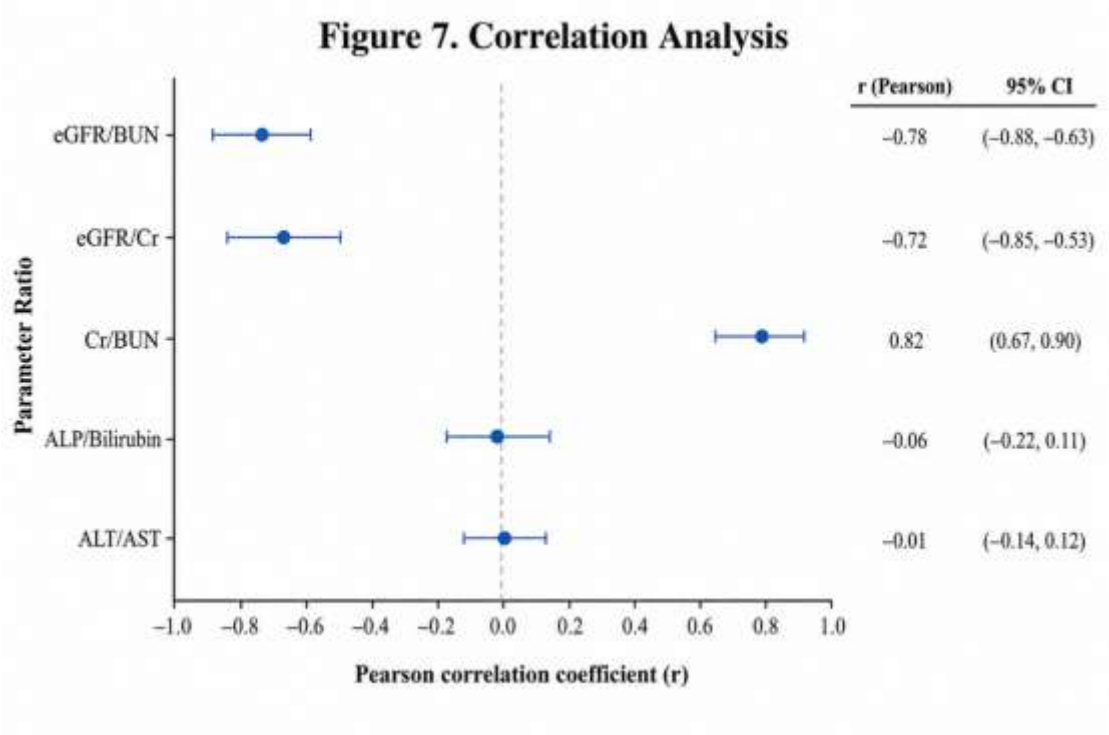

Figure 7. Pearson correlation analysis of selected biochemical parameters in transfusion-dependent β-thalassemia major patients. A strong positive correlation was observed between serum creatinine and BUN (r = 0.817), whereas eGFR demonstrated strong negative correlations with serum creatinine (r = −0.730) and BUN (r = −0.741). Minimal correlations were observed between ALT and AST (r = 0.007) and between ALP and bilirubin (r = −0.038), indicating weak associations between these liver function parameters.

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; Cr, serum creatinine.