Original Article

Assessment of Hepatic function and Fibrosis using Fibroscan in Transfusion-dependent Paediatric Thalassemia Major patients- A Cross Sectional Study

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Abstract

Background: Chronic blood transfusions in pediatric β-thalassemia major lead to progressive iron accumulation, with the liver serving as a primary storage site. Iron-induced oxidative stress and inflammation can precipitate hepatic steatosis, fibrosis, and ultimately cirrhosis if unrecognized. The objective is to characterize hepatic dysfunction in transfusion-dependent children with β-thalassemia major by integrating clinical examination, biochemical markers, serum ferritin levels, and transient elastography, and to explore associations with transfusion frequency and chelation adherence. **Material and Methods:** In this cross-sectional study, 150 children aged 2–18 years with transfusion-dependent β-thalassemia major were enrolled at a tertiary pediatric centre. Data collected included demographic and anthropometric measures, clinical assessment for hepatomegaly and splenomegaly, pre-transfusion hemoglobin, liver enzymes (AST, ALT), and serum ferritin. FibroScan provided controlled attenuation parameter (CAP) for steatosis and liver stiffness measurement (LSM) for fibrosis staging. Transfusion burden and chelation compliance were recorded. Statistical analyses employed Pearson's correlation and Chi-square tests, with $p < 0.05$ denoting significance. **Results:** The cohort's mean age was 9.5 ± 4.2 years; 59.3% were male. Hepatomegaly and splenomegaly were observed in 59.3% and 19.3%, respectively. Serum ferritin exceeded 3000 ng/mL in 36.0% of participants. AST and ALT were elevated (>2× ULN) in 46.7% and 20.0%. FibroScan revealed steatosis grades S1–S3 in 58.1% and fibrosis stages F1–F3 in 28.6%. Higher transfusion frequency (≥15/year) correlated with increased hepatomegaly ($p = 0.001$), splenomegaly ($p = 0.001$), ferritin >3000 ng/mL ($p = 0.001$), and advanced fibrosis ($p = 0.001$). Strong positive correlations were found between ferritin and AST ($r = 0.918$) and ALT ($r = 0.919$). Poor chelation adherence was associated with ferritin >3000 ng/mL in 92.9% of non-compliant patients ($p < 0.001$). **Conclusion:** Non-invasive integration of biochemical markers, ferritin quantification, and transient elastography effectively delineates hepatic injury in pediatric β-thalassemia major. Transfusion intensity and chelation adherence are critical determinants of iron-mediated liver dysfunction, underscoring the need for routine elastographic monitoring and targeted chelation strategies.

Keywords: β-Thalassemia Major; Hepatic Fibrosis; Transient Elastography; Iron Overload; Serum Ferritin.

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Introduction

Transfusion-dependent β-thalassemia major is a hereditary hemoglobinopathy characterized by absent or markedly reduced β-globin chain synthesis, resulting in life-threatening anemia that necessitates lifelong blood transfusions.[1] Chronic transfusions, while essential for survival, precipitate systemic iron overload due to the body's inability to excrete excess iron, leading to its deposition in organs such as the liver, heart, and endocrine glands.[2] The liver, as the principal iron-storage organ, is particularly susceptible to iron-induced oxidative damage, inflammation, and progressive fibrosis, which may culminate in cirrhosis or hepatocellular carcinoma if left undetected.[3,4] Although serum ferritin and liver function tests (LFTs) are routinely employed to monitor iron burden and hepatic injury, ferritin levels may not reliably predict histological damage, and standard biochemical markers often remain normal until advanced liver pathology develops.[5]

Non-invasive imaging modalities, notably transient elastography (FibroScan), enable quantitative assessment of hepatic stiffness and controlled attenuation, providing early detection of fibrosis and steatosis without the risks associated with biopsy. Despite growing recognition of FibroScan's utility in chronic liver diseases, its application in pediatric β-thalassemia major remains underexplored.

This study delivers an integrated evaluation of hepatic health in

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transfusion-dependent pediatric β-thalassemia major by combining detailed clinical profiling, conventional biochemical markers, serum ferritin quantification, and quantitative elastography (FibroScan) metrics of liver stiffness and steatosis. By examining how these diverse parameters relate to transfusion intensity and chelation adherence, the research establishes a unified, non-invasive framework for early detection and monitoring of iron-mediated liver injury in this vulnerable population.

Aims and Objectives

1. To evaluate hepatic dysfunction in multi-transfused pediatric patients with thalassemia major using clinical assessment, biochemical markers, and non-invasive elastography (FibroScan).
2. To analyze the relationship between the severity of hepatic dysfunction and serum ferritin levels in transfusion-dependent children with beta-thalassemia major.

Materials and Methods

The study employed a cross-sectional design conducted over one year in the Department of Pediatrics at M.G.M. Medical College and M.Y. Hospital, Indore, Madhya Pradesh Medical Science University, Jabalpur. Eligibility comprised pediatric patients aged 2–18 years with a confirmed diagnosis of transfusion-dependent β-thalassemia major, who had received regular blood transfusions for at least one year. Exclusion criteria included coexisting chronic liver disease (e.g., viral hepatitis), use of hepatotoxic medications, and lack of parental consent.

Sample size was calculated based on an expected 3.5% prevalence of transfusion-dependent thalassemia major using $n = 4pq/d^2$, yielding 150 subjects at 80% power and 5% significance. A convenience sampling method enrolled 150 patients.[6]

Demographic and clinical data—including age, sex, socioeconomic status, anthropometry (weight, height, BMI)—were recorded using IAP growth charts to determine centile positions. Hepatomegaly and splenomegaly were assessed by abdominal palpation and categorized as significant or non-significant based on standard physical findings.

Pre-transfusion hemoglobin was measured by automated cell counter and serum ferritin was quantified by immunoassay. Liver enzymes (AST, ALT) and bilirubin were analyzed biochemically; elevations were defined as >2× the upper limit of normal. Non-invasive hepatic assessment employed FibroScan MINI 450. Controlled attenuation parameter (CAP) scores quantified steatosis and were graded S0–S3 using thresholds. Liver stiffness measurements (LSM) staged fibrosis. Transfusion frequency was dichotomized at 15 transfusions per year. Chelation compliance was considered adequate if patients received recommended doses of deferasirox or deferoxamine consistently over the preceding year.Data were entered into Microsoft Excel and analyzed with IBM SPSS v.22. Categorical variables were summarized as counts and percentages; associations were tested by Pearson's Chi-square. Continuous variables were expressed as means with standard deviations. Correlations were assessed using the Pearson correlation coefficient. A p-value <0.05 denoted statistical significance.

Operational Definitions

- Transfusion-Dependent β-Thalassemia Major: Diagnosis confirmed by hemoglobin electrophoresis with requirement for regular blood transfusions over at least one year.[7]
- Significant Hepatomegaly: Liver palpable >2 cm below the right costal margin on clinical examination.[8]
- Significant Splenomegaly: Spleen palpable >1 cm below the left costal margin.[9]
- Pre-Transfusion Hemoglobin Categories: 3–6 g/dL; 7–9 g/dL; >9 g/dL, as measured immediately before transfusion.[10]
- Serum Ferritin Categories: <1000; 1000–2000; 2000–3000; >3000 ng/mL to reflect iron load.[11]
- Elevated Liver Enzymes: AST or ALT >2× the laboratory upper limit of normal.[12]
- FibroScan CAP Steatosis Grades- S0: <238 dB/m (≤ 5% fat); S1: 238–260 dB/m (11–33% fat); S2: 260–290 dB/m (34–66% fat); S3: >290 dB/m (≥ 67% fat).[13]
- FibroScan LSM Fibrosis Stages- F0: <2.5 kPa (no fibrosis); F0–F1: 2.5–7.0 kPa (normal–mild fibrosis); F1–F2: 7.0–9.5 kPa (mild–moderate fibrosis); F2–F3: 9.5–13.0 kPa (moderate–severe fibrosis).[14]
- Transfusion Frequency- Low: <15 transfusions/year; High: ≥15 transfusions/year.[10]
- Chelation Compliance: Adequate adherence to prescribed chelation dosing over the previous year; inadequate if dosing fell below recommended levels.[15]

Results

This cross-sectional analysis of 150 pediatric patients with transfusion-dependent β-thalassemia major reveals a substantial burden of hepatic involvement driven by chronic iron overload.

[Table 1] shows that participants were predominantly 6–12 years old, with a male predominance and nearly half from the lower-middle socioeconomic status. Growth impairment affected approximately one-quarter to one-third of the cohort.

[Table 2] depicts that over half of the children had significant hepatomegaly, while splenomegaly was less common. Most maintained pre-transfusion hemoglobin between 7–9 g/dL. Iron overload (ferritin >3000 ng/mL) and transaminase elevations—particularly AST—were frequent.

Most children exhibited no or mild steatosis (S0–S1) and normal to mild fibrosis (F0–F1); severe steatosis and moderate–severe fibrosis were uncommon as depicted in [Table 3].

Higher transfusion intensity (≥15/year) was significantly associated with increased hepatomegaly, splenomegaly, severe iron overload, and moderate–severe fibrosis (all $p < 0.01$) as shown in [Table 4].

[Table 5] depicts that AST and ALT levels rose in parallel with ferritin, indicating that iron overload is closely linked to hepatocellular injury. Bilirubin showed a weak association; albumin did not correlate.

Poor adherence to chelation therapy was strongly associated with extreme iron overload (ferritin >3000 ng/mL in 92.9% of non-compliant patients; $p < 0.001$) as shown in [Table 6].

Table 1: Baseline Socio-Demographic and Anthropometric Characteristics (n = 150)

Characteristic	Category	n (%)
Age (years)	2–6	48 (32.0)
	6–12	58 (38.7)
	12–18	44 (29.3)
Sex	Male	89 (59.3)
	Female	61 (40.7)
Socioeconomic Status	Upper	10 (6.7)
	Upper middle	43 (28.7)
	Lower middle	74 (49.3)
	Upper lower	23 (15.3)
Weight <3rd centile	Yes	44 (29.4)
Height <3rd centile	Yes	36 (24.0)
BMI <5th centile	Yes	21 (14.0)

Table 2: Hepatic, Splenic, Hematological, and Biochemical Parameters

Parameter	Category	n (%)
Hepatomegaly	Significant	89 (59.3)
	Non-significant	61 (40.7)
Splenomegaly	Significant	29 (19.3)
	Non-significant	121 (80.7)
Pre-transfusion Hemoglobin (g/dL)	3–6	9 (6.0)
	7–9	122 (81.3)
	>9	19 (12.6)
Serum Ferritin (ng/mL)	<1000	34 (22.6)
	1000–2000	25 (16.6)
	2000–3000	37 (24.6)
	>3000	54 (36.0)
ALT >2× ULN	Yes	30 (20.0)
AST >2× ULN	Yes	70 (46.7)

Table 3: FibroScan Steatosis (CAP) and Fibrosis (LSM) Grades

Measure	Grade	kPa or dB/m	n (%)
Steatosis (CAP)	S0	<238 dB/m	63 (42.0)
	S1	238–260 dB/m	43 (28.7)
	S2	260–290 dB/m	34 (22.7)
	S3	>290 dB/m	10 (6.7)
Fibrosis (LSM)	F0	<2.5 kPa	11 (7.3)
	F0–F1	2.5–7.0 kPa	96 (64.0)
	F1–F2	7.0–9.5 kPa	20 (13.3)
	F2–F3	9.5–13.0 kPa	23 (15.3)

Table 4: Transfusion Frequency and Hepatic Complications

Transfusions/Year	Hepatomegaly (%)	Splenomegaly (%)	Ferritin >3000 ng/mL (%)	Fibrosis ≥F2 (%)
<15	48.4	25.0	25.0	6.3
≥15	75.6	51.2	66.3	44.2

Table 5: Correlation Coefficients (r) Between Ferritin and Enzymes

Parameter Pair	r Value	p-Value	Interpretation
AST vs. Ferritin	0.918	<0.001	Very strong, significant positive
ALT vs. Ferritin	0.919	<0.001	Very strong, significant positive
Bilirubin vs. Ferritin	0.024	0.001	Weak but significant positive
Albumin vs. Ferritin	0.231	0.078	No significant correlation

Table 6: Chelation Adherence and Ferritin Levels

Compliance	<1000	1000–2000	2000–3000	>3000
Adequate (n = 132)	24 (18.2)	25 (18.9)	62 (47.0)	21 (15.9)
Inadequate (n = 14)	0 (0.0)	0 (0.0)	1 (7.1)	13 (92.9)

Bar chart is showing that 64.0% of patients had F0–F1 fibrosis, while 28.6% exhibited moderate–severe fibrosis (F1–F3) in [Figure 1].

Bar chart illustrates the predominance of no steatosis (S0, 42.0%) and mild steatosis (S1, 28.7%), with severe steatosis (S3) in 6.7% in [Figure 2].

Stacked bar chart depicts the marked increase in moderate–severe fibrosis (≥F2) among patients receiving ≥15 transfusions per year compared to those with fewer transfusions in [Figure 3].

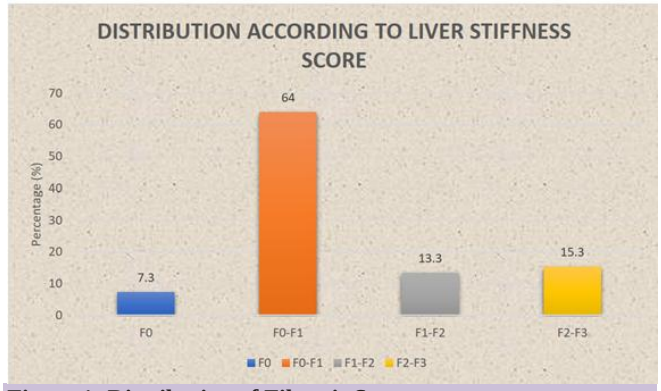

Figure 1: Distribution of Fibrosis Stages

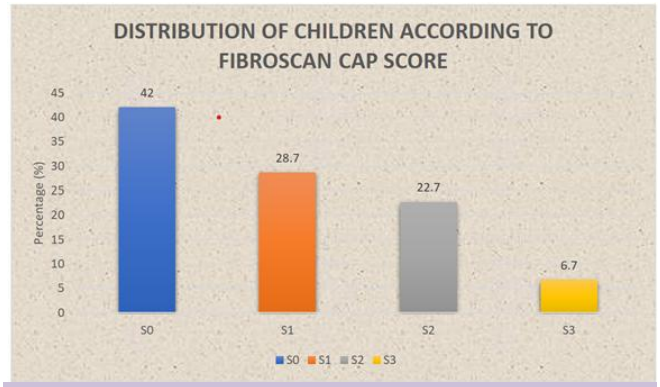

Figure 2: Steatosis Grading by CAP Score

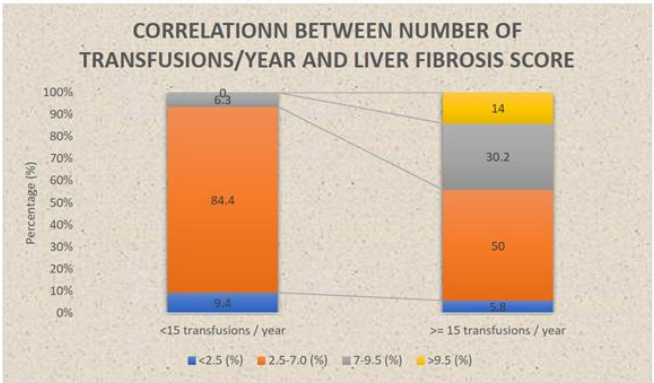

Figure 3: Correlation Between Transfusion Frequency and Fibrosis Severity

Discussion

The present study delineated a high burden of hepatic involvement in pediatric patients with transfusion-dependent β-thalassemia major, with 59.3% exhibiting significant hepatomegaly and 28.6% demonstrating moderate-to-severe fibrosis on transient elastography. These findings align closely with those of Sudhakar et al. (2023), who reported hepatomegaly in 65.3% of transfusion-dependent children and elevated transaminases in 38.9% of cases.[16] The incremental rise in fibrosis stages with age in our cohort mirrors the age-dependent increase in liver stiffness reported by Wijenayake et al. (2025), where 43.2% of adolescents had F2–F3 fibrosis compared to 4.2% of younger children.[17] The cohort's mean age of 9.5 ± 4.2 years and male predominance (59.3%) reflect established epidemiological trends. Age-dependent increases in hepatomegaly—from 29.1% in 2–6 years to 86.3% in 12–18 years—underscore cumulative iron deposition over time. Harish et al. (2019) examined 41 transfusion-dependent children (median age 8 years), reporting growth retardation in 60% despite chelation, with more severe stunting in those harboring higher ferritin levels.[18] This suggests that early-life nutritional deficits and iron burden critically influence both growth and hepatic outcomes. Furthermore, Mohammed et al. (2023) assessed 106 homozygous β-thalassemia major patients (mean ferritin 3,799 ± 2,344 ng/mL in males, 4,101 ± 1,977 ng/mL in females) and found hepatomegaly in 71.7%—notably higher than our 59.3%.[19] Their elevated prevalence likely reflects differences in chelation adherence or genetic modifiers, highlighting the necessity of tailoring monitoring strategies to demographic and treatment variables.

Biochemical correlations demonstrated very strong positive associations between serum ferritin and both SGOT (r = 0.918) and SGPT (r = 0.919), indicating that iron overload underpins hepatocellular injury in this population. Bhalodiya et al. (2023) similarly found that mean ferritin levels rose from 1262 µg/L in younger children to 2387 µg/L in older cohorts, with a parallel increase in transaminase elevations.20 Our observation of SGOT being disproportionately elevated relative to SGPT further suggests mitochondrial involvement in iron-mediated oxidative stress, a pattern also noted by Al-Moshary et al. (2020) in multi-transfused patients.[21]

Transient elastography revealed that while 64.0% of patients had F0–F1 fibrosis, 15.3% exhibited moderate–severe fibrosis. These rates are modestly lower than the 23.5% cirrhosis (TE >12.5 kPa) and 35% variable fibrosis reported by Elalfy et al. (2013) in Egyptian adolescents with HCV co-infection, likely reflecting the exclusion of viral hepatitis and diligent chelation in our cohort.[22] However, our fibrosis prevalence is comparable to the 28.6% F1–F3 fibrosis found in Parakh et al. (2022), who demonstrated significant TE correlations with both MRI R2* and serum ferritin.[23]

The impact of transfusion frequency on hepatic outcomes was pronounced: children receiving ≥15 transfusions/year had significantly higher rates of hepatomegaly (75.6% vs. 48.4%), splenomegaly (51.2% vs. 25.0%), serum ferritin >3000 ng/mL (66.3% vs. 25.0%), and moderate–severe fibrosis (44.2% vs. 6.3%). These associations corroborate the findings of Khan et al. (2023), who observed that TE measurements correlated strongly with transfusion burden and LIC by MRI (rS = 0.39, p = 0.0001).[24] The linear relationship between transfusion load, iron overload, and hepatic injury emphasizes the critical need for balanced transfusion protocols and aggressive chelation.

Chelation compliance emerged as a pivotal modifiable factor: 92.9% of non-compliant patients had ferritin >3000 ng/mL compared to 15.9% of compliant patients (p <0.001). This stark contrast echoes the observations of Shehata et al. (2019), who reported that poor chelation adherence significantly exacerbates hepatic siderosis and fibrosis in pediatric thalassemia patients.[25] Collectively, these comparisons illustrate that the severity of hepatic dysfunction in pediatric thalassemia major is driven by cumulative transfusion burden and iron overload, mitigated partially by effective chelation. The concordance of our results with recent international cohorts reinforces the global

applicability of these findings and highlights the necessity for early, routine, and non-invasive monitoring of liver health in this high-risk population.

Conclusion

Chronic transfusions in pediatric β-thalassemia major culminate in progressive hepatic injury characterized by hepatomegaly, steatosis, and fibrosis, all of which correlate strongly with iron overload and transfusion intensity. Transient elastography, coupled with biochemical markers, offers a comprehensive, non-invasive framework for early detection and monitoring of liver dysfunction. Effective chelation adherence significantly attenuates iron burden and hepatic injury, underscoring its central role in patient management.

Recommendations: To optimize hepatic health in pediatric patients with transfusion-dependent β-thalassemia major, it is advisable to integrate annual transient elastography alongside serum ferritin and standard liver function tests to enable earlier detection of fibrosis. Transfusion protocols should be carefully tailored to maintain pre-transfusion hemoglobin levels around 9 g/dL, balancing the need to suppress ineffective erythropoiesis with the goal of minimizing cumulative iron loading. Patient and family education initiatives must be strengthened to promote consistent adherence to chelation regimens, and multidisciplinary collaboration with hepatology specialists should be considered for individuals demonstrating persistent fibrosis despite optimized chelation.

Strengths and Limitations: This investigation's principal strength lies in its comprehensive, multimodal assessment of liver health—combining clinical evaluation, biochemical markers, and non-invasive elastography—in a sizeable cohort of pediatric thalassemia major patients. By excluding coexistent viral hepatitis and hepatotoxic medications, the study isolated iron-mediated hepatic injury, enhancing the specificity of its findings. However, its cross-sectional design precluded evaluation of fibrosis progression over time, and reliance on transient elastography without histological confirmation limited the granularity of fibrosis staging. Additionally, the single-center setting may constrain the generalizability of results, and the absence of inflammatory markers such as C-reactive protein prevented adjustment for potential confounders affecting serum ferritin levels.

Relevance of the Study: This investigation provides contemporary, region-specific evidence on the hepatic sequelae of transfusion-dependent thalassemia major, informing guidelines for non-invasive monitoring and reinforcing the imperative of chelation adherence to preserve liver health.

Authors' Contributions: Dr. Sanskriti Jain conceptualized the study, managed data collection, and drafted the manuscript. Dr. Shachi Jain Taran and Dr. Atul Shende oversaw methodological development, guided data interpretation, and contributed to manuscript revisions. Dr. Preeti Malpani provided overall project supervision, critically reviewed all components of the work, and approved the final manuscript.

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Conflicts of interest

There are no conflicts of interest.

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