MRI Estimation of Liver Iron Concentration in Patients with Hemoglobinopathy in Relation to Serum Ferritin in Basrah

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Article Info

E-ISSN: 2582-8940
Volume: 07
Issue: 02
Received: 20-03-2026
Accepted: 18-04-2026
Published: 16-05-2026
Page No: 192-205

Abstract

Background: liver iron overload in hemoglobinopathy population with repeated blood transfusion is an important morbidity warranting the evaluation of a non-invasive method of liver iron quantification. MR T2* / R2* relaxometry technique has shown promising results in this domain.

Aim of Study: this prospective cross sectional study aim to show the value of MR T2*/R2* relaxometry technique, in assessment of hepatic iron overload in a sample of hemoglobinopathy population at our hospital on repeated blood transfusion therapy in South of Iraq (Basra city).

Patients and Methods: a total of 70 anaemic patients, mean age 21 years (range : 1055 years old) patients with hemoglobinopathy who had received repeated blood transfusions and on chelation therapy at time of examination, underwent MRI T2* / R2* relaxometry study. The corresponding iron overload was calculated for each patient, then correlation between MRI relaxometry study and serum ferritin level using correlation coefficient was done.

Results: at the time of the study, serum ferritin levels ranged between 297 and 7324 ng/l (mean 1958 ng/l).based on the calculated liver iron concentration (LIC) values show that 37.1% of the patients had moderate level of LIC, the highest prevalence of severe LIC was seen significantly in patients using DFO drug(25%) and in those diagnosed with thalassemia major (16.2%), no significant association between LIC and splenectomy. Strong negative correlation was detected between LIC and T2*, Perfect positive correlation between LIC and R2* and moderate positive correlation between LIC and S. ferritin. No significant correlation between LIC and age. In conclusion, MRI R2*/T2* provides a non-invasive, precise, and repeatable method for measuring tissue iron content. The treatment of tissue iron excess should be improved by the widespread use of this approach, enabling early chelation intensification & monitoring of iron level & response to treatment

Conclusion: In conclusion, MRI R2* provides a non-invasive, precise, and repeatable method for measuring hepatic tissue iron content

Recommendation: that. MRI is effective in management of hemoglobinopathy so,repeat liver MRI for all patients who have LIC > 7mg/g DW liver (every 6 months,or 1year depending on guidelines and Cardiac MRI is required for all patient to exclude cardiac iron deposition.

DOI: https://doi.org/10.54660/IJMBHR.2026.7.2.192-205

Keywords: Hemoglobinopathy, liver iron concentration, MRI R2*/T2* relaxometry, serum ferritin, iron overload, thalassemia, sickle cell disease

Introduction

1. Iron Overload: Overview and Clinical Significance

Iron overload is a systemic disorder characterised by elevated plasma iron levels and progressive accumulation of iron in parenchymal cells, primarily in the form of ferritin and hemosiderin. The liver serves as the principal iron storage organ, and liver iron concentration (LIC) is linearly correlated with total body iron stores, rendering it the most reliable index of systemic

iron burden [1]. When the body's capacity to store iron safely is exceeded, free iron generates reactive oxygen species through the Fenton reaction, causing peroxidative damage to cell membranes, organelles, and DNA [2]. The resulting target-organ toxicity manifests as hepatic fibrosis and cirrhosis, cardiomyopathy with arrhythmias and heart failure, endocrinopathies (growth retardation, hypogonadism, diabetes mellitus), and ultimately premature death if left untreated [3].

Iron overload disorders are broadly classified as primary (hereditary) or secondary (acquired). Primary iron overload is exemplified by hereditary haemochromatosis, most commonly attributable to homozygous mutations in the HFE gene (C282Y), which impairs hepcidin-mediated regulation of intestinal iron absorption [4]. Secondary iron overload arises predominantly from chronic red blood cell transfusion, a cornerstone of management in haemoglobinopathies such as beta-thalassaemia major and sickle cell disease (SCD), as well as from myelodysplastic syndromes and other haematological disorders [5].

2. Epidemiology of Haemoglobinopathies

Haemoglobinopathies are among the most prevalent inherited disorders worldwide, particularly in the Middle East, sub-Saharan Africa, and Southeast Asia. In Iraq, these conditions constitute a major public health burden. A recent 10-year epidemiological study conducted in Basrah Governorate (2013–2022), encompassing 6,081 registered patients, reported an overall prevalence of 238 per 100,000 population, with sickle cell disease accounting for 68.58% of cases, followed by thalassaemia major (16.04%) and thalassaemia intermedia (10.93%) [6]. Notably, Basrah contains the largest cohort of haemoglobinopathy patients in Iraq, a fact attributed partly to the high rate of consanguineous marriages (60.52% of affected families) [6]. Clinically significant complications were documented in 14.21% of patients, with sickle crisis being the most frequent (26.83%), followed by cardiac disease (12.18%), osteonecrosis (10.81%), delayed growth (9.43%), and diabetes mellitus (7.78%) [6]. These data underscore the pressing need for systematic, non-invasive monitoring strategies in this region.

3. Pathophysiology of Transfusional Iron Overload

Each unit of packed red blood cells delivers approximately 200–250 mg of iron, which cannot be physiologically excreted by the human body. In chronically transfused patients, the transferrin iron-binding capacity is rapidly saturated; when transferrin saturation exceeds 45%, non-transferrin-bound iron (NTBI) appears in plasma, and when saturation surpasses 75%, the catalytically active labile plasma iron (LPI) fraction predominates [7]. NTBI is avidly taken up by hepatocytes, cardiomyocytes, and endocrine cells through divalent metal transporter-1 (DMT-1) and other unregulated pathways, driving progressive parenchymal iron accumulation [8]. In beta-thalassaemia, ineffective erythropoiesis further suppresses hepcidin synthesis, augmenting intestinal iron absorption and compounding the transfusional iron burden [9]. The net result is a dual-mechanism overload that can rapidly overwhelm chelation capacity if not closely monitored.

4. Diagnostic Modalities for Iron Overload

Several methods are available for assessing iron overload, each with distinct advantages and limitations. Serum ferritin (SF) is the most widely used surrogate marker owing to its low cost and ready availability. However, it is an acute-phase reactant and may be elevated by inflammation, infection, hepatitis, or malignancy independently of iron stores, limiting its specificity [10]. Moreover, SF correlates poorly with cardiac iron loading, and reliance on SF alone can underestimate or overestimate true iron burden, leading to suboptimal chelation decisions [11]. Liver biopsy, historically considered the reference standard for LIC measurement, provides direct histological and biochemical quantification but carries procedural risks (bleeding, infection, pneumothorax), sampling variability attributable to non-uniform hepatic iron distribution, and poor patient acceptability for serial monitoring [12].

Magnetic resonance imaging (MRI) has emerged as the non-invasive standard of care for quantifying organ iron in patients with haemoglobinopathies [11]. MRI exploits the paramagnetic properties of iron, which accelerate transverse relaxation and shorten T2 and T2* relaxation times in proportion to iron concentration. Specific LIC and cardiac T2* thresholds have been prospectively validated against clinical outcomes: LIC >7 mg/g dry weight is associated with significant clinical morbidity; cardiac T2* <20 ms predicts arrhythmias, and cardiac T2* <10 ms predicts heart failure and death [11]. Given the high prevalence of transfusion-dependent thalassaemia (TDT) in resource-limited settings, international expert panels have proposed evidence-based algorithms to guide optimal MRI utilisation for both liver and cardiac iron monitoring, stratifying access according to MRI availability [11].

5. MRI Techniques for Liver Iron Concentration Quantification

Three principal MRI-based approaches are employed for LIC quantification. The Signal Intensity Ratio (SIR) method compares liver signal to a reference tissue (paraspinal muscle or subcutaneous fat) on multiple spin-echo sequences; it is widely available but is insensitive at high LIC values and requires strict technical standardisation [13]. R2 relaxometry (FerriScan®), based on multi-spin-echo acquisitions, provides a validated, FDA-cleared measurement with a reported dynamic range of approximately 0.3–43 mg/g dry weight, though it requires dedicated post-processing software and prolonged scan times [14]. R2* relaxometry, derived from multi-echo gradient echo (GRE) sequences, is currently the preferred technique due to its speed (acquisition in a single breath-hold), wide dynamic range, excellent correlation with biopsy-derived LIC, and capacity for simultaneous cardiac iron assessment [15]. Iron shortens T2* (T2* = 1/R2*); as LIC increases, T2* decreases and R2* increases proportionally. The relationship between R2* (Hz) and LIC (mg/g dry weight) follows a well-characterised linear calibration: LIC (mg Fe/g dw) ≈ R2* × 0.0254 [16].

Liver iron burden is graded according to established thresholds derived from biopsy-validated MRI calibration studies (Table 1) [17].

Table 1: Grading of liver iron overload by LIC, T2*, and R2* values [17].

Grade	LIC (mg/g dry weight)	T2* (ms)	R2* (Hz)
Normal	< 1.8	> 14.3	< 70
Borderline	1.8 – 3.2	7.9 – 14.3	70 – 126
Mild	3.2 – 7.0	3.6 – 7.9	126 – 278
Moderate	7.0 – 15.0	1.7 – 3.6	278 – 595
Severe	> 15.0	< 1.7	> 595

6. Rationale of the Study

Despite the established superiority of MRI over serum ferritin for iron overload assessment, MRI-based LIC quantification remains under-utilised in Iraq and other resource-limited settings, where clinical decisions continue to rely predominantly on serum ferritin measurements. Basrah, which carries one of the highest burdens of haemoglobinopathy in the country, represents a particularly relevant setting in which to evaluate the diagnostic performance and practical utility of MRI R2*/T2* relaxometry for LIC estimation. Furthermore, the relationship between MRI-derived LIC and serum ferritin in this population has not been systematically characterised, leaving a significant knowledge gap regarding the reliability of ferritin-guided chelation in local clinical practice.

Aims of the study

This study therefore aimed to: (1) evaluate MRI T2*/R2* relaxometry as a non-invasive tool for quantifying liver iron concentration in patients with haemoglobinopathy attending Al-Sadr Teaching Hospital, Basrah; (2) determine the distribution of iron overload severity by MRI grading in this cohort; and (3) investigate the correlation between MRI-derived LIC and serum ferritin levels, with the objective of establishing an evidence-based guideline for iron monitoring and chelation management in the local clinical context.

Results

Table 2: Mean LIC in relation to type of the disease

Distribution by Anemia Type			
Anemia Type	**Count**	**Percentage**	**Mean LIC**
Thalassemia major	40	51.28%	8.03
Thalassemia intermedia	10	12.82%	9.04
Sickle cell disease	12	15.38%	3.06
Sickle thalassemia	15	19.23%	5.37
others	1	1.28%	6.5
Total	78	100.00%	

The study cohort comprised 78 patients with various chronic hemolytic anemias. Thalassemia major represented the predominant disease category, accounting for 51.28% of the total cohort (n=40), followed by sickle-thalassemia (19.23%, n=15), sickle cell disease (15.38%, n=12), and thalassemia intermedia (12.82%, n=10). Notably, thalassemia intermedia exhibited the highest mean liver iron concentration (LIC) of 9.04 mg Fe/g dry weight, marginally exceeding that of thalassemia major (8.03 mg Fe/g). This finding may reflect the greater contribution of ineffective erythropoiesis and enhanced gastrointestinal iron absorption in thalassemia intermedia patients, who are often inadequately chelated or not regularly transfused. Conversely, sickle cell disease was associated with the lowest mean LIC (3.06 mg Fe/g), consistent with a comparatively lower cumulative transfusion burden relative to thalassemic syndromes.

Table 3: LIC Grades Distribution with the corresponding S ferritin

LIC Grading Distribution				
LIC Grade	**LIC Range (mg Fe/g)**	**Count**	**Percentage**	**Mean S.F**
Normal	0.17-1.8	13	16.88%	801.62
Borderline	1.8-3.2	4	5.19%	1,240.25
Mild	3.2-7	26	33.77%	2,002.69
Moderate	7-15	27	35.06%	2,285.33
Severe	>15	7	9.09%	3,234.71
Total		77	100.00%	

The distribution of LIC grades revealed that a substantial majority of patients (77.92%) exhibited clinically significant hepatic iron overload, with 33.77% classified as mild (3.2–7 mg Fe/g), 35.06% as moderate (7–15 mg Fe/g), and 9.09% as severe (>15 mg Fe/g). Only 16.88% of patients demonstrated normal LIC values (0.17–1.8 mg Fe/g). A stepwise and progressive increase in mean serum ferritin (S.F) was observed across ascending LIC grade categories, ranging from 801.62 µg/L in the normal LIC group to 3,234.71 µg/L in the severe group. This dose-response relationship provides supporting evidence for the utility of serum ferritin as a surrogate marker of hepatic iron burden, albeit acknowledging its well-known limitations in the setting of chronic inflammation and hepatocellular damage.

Table 4: patients distribution regarding demographic and relevant characteristics

Sex	**Count**	**Percentage**
Male	42	53.85%
Female	36	46.15%
Total	78	100.00%
Age Group	Count	Percentage
0-10 years	7	8.97%
11-20 years	43	55.13%
21-35 years	21	26.92%
36-60 years	7	8.97%
centre	26	33.33%
periphery	52	66.67%
illiterate	22	28.21%
primary	39	50.00%
secondary	13	16.67%
higher education	5	6.41%
yes	7	8.97%
no	71	91.03%
yes	2	2.56%
no	76	97.44%
Meical disease associated		
no medical disease	61	78.21%
cardiac morbidity	9	11.54%
endocrinopathies (growth failure/ DM)	8	10.26%
Blood Transfusion Frequency		
null	12	15.38%
> monthly	9	11.54%
revery 1 month	22	28.21%
every 2 week -29 days	18	23.08%
every 1 week-13 days	4	5.13%
> weekly	3	3.85%
exchange transfusion every 3 m	4	5.13%
on demand	6	7.69%
Surgical Profile		
no surgery	39	50.00%
splenectomy	32	41.03%
splenectomy + cholecystectomy	6	7.69%
Cholecystectomy + others	2	2.56%
annual transfused blood	no	%
<2000cc/year	34	43.59%
2001-4000	15	19.23%
4001-6000	9	11.54%
>6000	21	26.92%

Table 3 presents a comprehensive demographic and clinical profile of the study population. The cohort included a slightly higher proportion of males (53.85%) compared to females (46.15%), with the majority of patients falling within the 11–20-year age group (55.13%), reflecting the chronic and lifelong nature of transfusion-dependent hemolytic anemias and the importance of early-onset monitoring. Most patients resided in peripheral areas (66.67%), and educational attainment was predominantly at the primary level (50.00%). Regarding medical comorbidities, 78.21% of patients had no documented co-existing conditions; however, cardiac morbidity (11.54%) and endocrinopathies (10.26%) were present in a clinically relevant minority, most likely as direct sequelae of chronic untreated or inadequately chelated iron overload. Monthly transfusion (28.21%) represented the most prevalent blood transfusion schedule, with an average annual transfused blood volume of 3,238.97 cc/patient/year (range: 0–7,800 cc). Splenectomy had been performed in 41.03% of patients, reflecting the high prevalence of hypersplenism as a disease complication.

Average annual transfused blood 3238.97 min 0 max 7800 cc/patient/year Complications were two with SCA and sequestration crises before, two with acute chest syndrome and aplastic crises in three patients existed medical comorbidities exist in 21.79% with an average LIC of 7.34

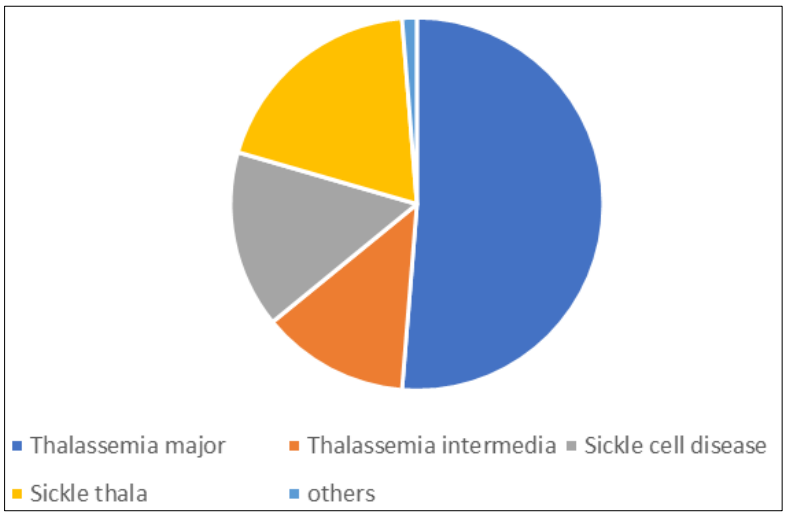

Fig 1: case distribution by the diseases

Graph 1 provides a visual representation of the case distribution across disease categories, confirming the numerical data presented in Table 1. The chart clearly illustrates the dominance of thalassemia major within the studied cohort, comprising more than half of the total patient population. The relative proportions of sickle-thalassemia and sickle cell disease are also evident, underscoring the heterogeneity of hemolytic anemias included in this study.

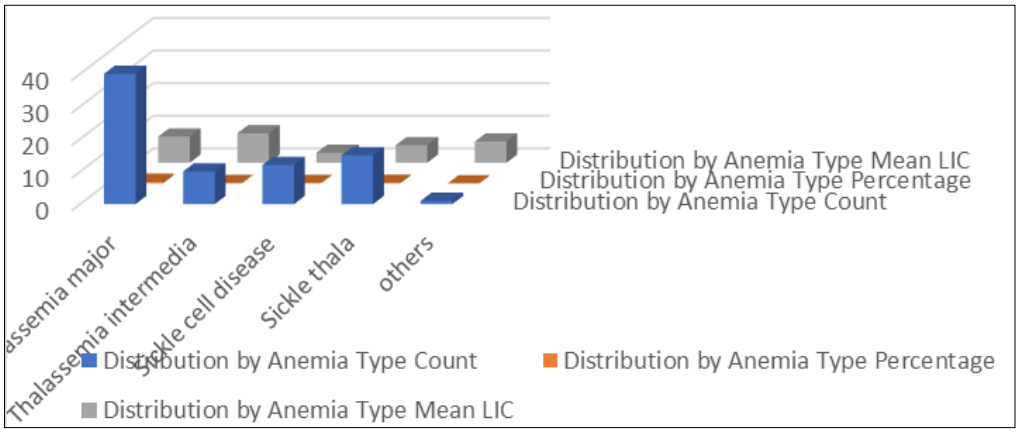

Fig 2: case distribution by disease and average LIC

Graph 2 complements Graph 1 by superimposing mean LIC values on the disease-type distribution, thereby highlighting the discordance between patient frequency and iron overload severity. Despite comprising only 12.82% of the cohort, patients with thalassemia intermedia demonstrated the highest mean LIC, further emphasizing the disproportionate iron accumulation in this subgroup. The comparatively low mean LIC in sickle cell disease patients reinforces the notion that disease-specific transfusion requirements are the primary determinants of hepatic iron loading.

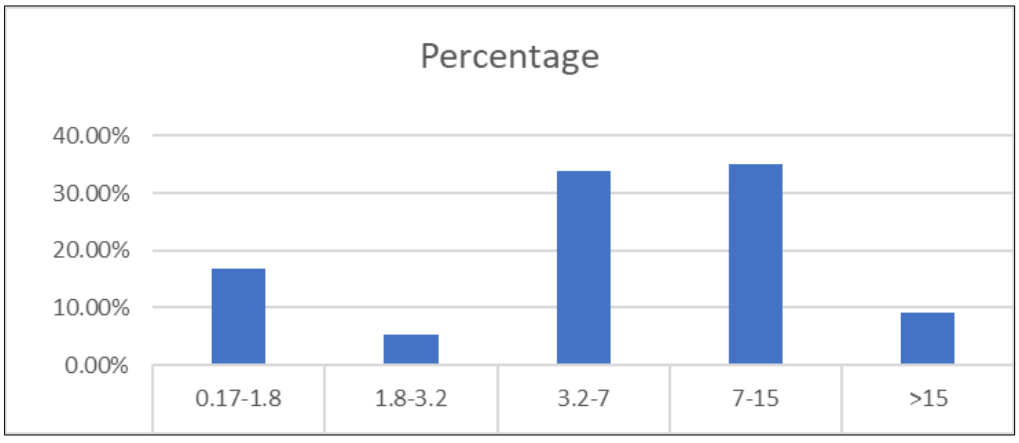

Fig 3: case distribution by age category

Graph 3 provides a visual illustration of the age distribution within the cohort, confirming the predominance of adolescents and young adults in the 11–20-year age bracket. This demographic pattern reflects the chronic yet survivable nature of these hemolytic conditions with appropriate medical management, as well as the increasing success of supportive care in extending patient survival into adulthood. The representation of older age groups (21–35 and 36–60 years), though smaller, underscores the growing population of adult patients with lifelong transfusion-dependent anemias who require sustained monitoring for iron-related end-organ damage.

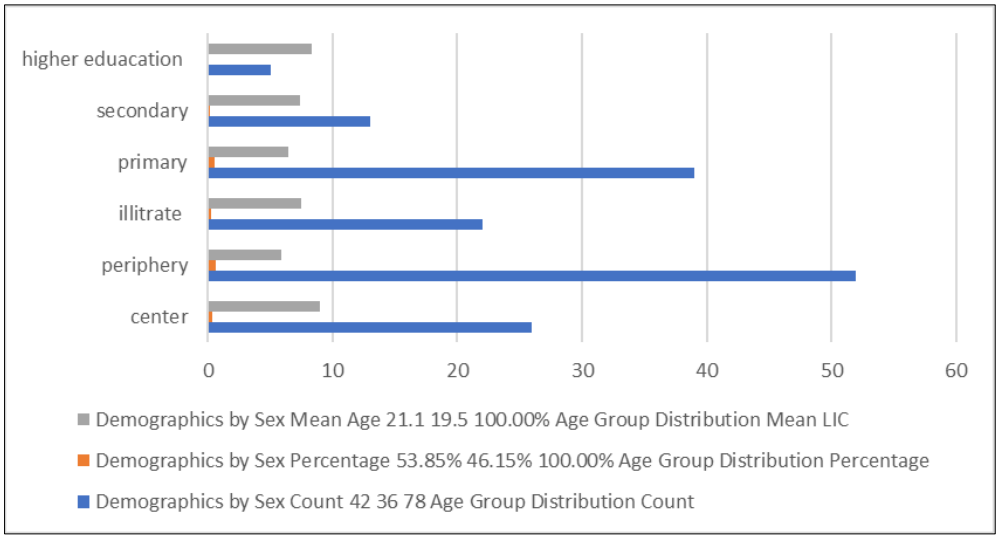

Fig 4: case distribution by residency and education toward LIC

Graph 4 visually compares mean LIC values stratified by residency and educational level. The consistently elevated LIC observed in centrally located patients relative to those from peripheral regions is clearly depicted, suggesting that urban-dwelling patients may carry heavier disease burdens, likely due to higher proportions of severely affected thalassemia major cases presenting to central referral centers. The variation in mean LIC across educational subgroups may reflect differences in health literacy, chelation compliance, and follow-up regularity, though caution is warranted in interpreting these associations given the small sample sizes within certain educational categories.

Table 5: LIC mean toward certain demographics

Age Group Distribution			
Age Group	**Count**	**Percentage**	**Mean LIC**
0-10 years	7	8.97%	8.27
11-20 years	43	55.13%	7.03
21-35 years	21	26.92%	5.51
36-60 years	7	8.97%	8.71
Residency			
centre	26	33.33%	8.99
periphery	52	66.67%	5.92
Education			
illiterate	22	28.21%	7.49
primary	39	50.00%	6.47
secondary	13	16.67%	7.34
higher education	5	6.41%	8.32
Complication Profile			
yes	7	8.97%	8.51
no	71	91.03%	6.94
Hepatitis			
yes	2	2.56%	5.5
no	76	97.44%	6.92
medical diseases associated			
no medical disease	61	78.21%	6.76
cardiac morbidity	9	11.54%	8.17
endocrinopathies (growth failure/ DM)	8	10.26%	6.4125
Surgical Profile			
no surgery	39	50.00%	6.61
splenectomy	32	41.03%	7.53
splenectomy + cholecystectomy	6	7.69%	5.42
Cholecystectomy + others	2	2.56%	5.2

Note: Tables 4(a) and 4(b) are presented consecutively as they both address the distribution of mean LIC across demographic, clinical, and laboratory subgroups, providing a unified analysis of LIC determinants.

The distribution of mean LIC across demographic and clinical subgroups revealed several noteworthy patterns. Patients in the 36–60-year age group exhibited the highest mean LIC (8.71 mg Fe/g), likely reflecting the cumulative effect of decades of transfusion therapy and potential long-term inadequacy of chelation. Patients residing in central areas demonstrated a markedly higher mean LIC (8.99 mg Fe/g) compared to peripheral residents (5.92 mg Fe/g), possibly attributable to a higher concentration of severely affected thalassemia major patients at referral centers. Patients with co-existing medical complications had a higher mean LIC (8.51 mg Fe/g) compared to those without (6.94 mg Fe/g), consistent with iron overload-mediated organ damage. Among surgical subgroups, splenectomised patients demonstrated a higher mean LIC (7.53 mg Fe/g) than non-operated patients (6.61 mg Fe/g), in keeping with the known post-splenectomy increase in both transfusion requirements and gastrointestinal iron absorption.

Table 6: mean LIC distribution towards certain lab variables

Hepatitis			
yes	2	2.56%	5.5
no	76	97.44%	6.92
Hepatopathies			
abnormal liver profile	12	15.38%	7.17
normal liver function	66	84.62%	5.39
blood groups			
A+	26	33.33%	7.09
B+	17	21.79%	6.87
AB+	3	3.85%	6.53
O+	29	37.18%	6.77
B-	1	1.28%	9.4
O-	2	2.56%	5.5

Analysis of mean LIC in relation to laboratory variables demonstrated that patients with abnormal liver profiles (hepatopathies) exhibited a higher mean LIC (7.17 mg Fe/g) compared to those with normal hepatic function (5.39 mg Fe/g), supporting the causal relationship between hepatic iron deposition and hepatocellular injury. Hepatitis co-infection was associated with a mean LIC of 5.50 mg Fe/g. Regarding ABO blood group distribution, no clinically meaningful inter-group differences in mean LIC were apparent (range: 5.50–9.40 mg Fe/g), and the notably small sample sizes in certain blood groups preclude definitive conclusions regarding blood group as a determinant of iron loading. No appreciable difference in mean LIC was observed between Rh-positive and Rh-negative patients (6.89 vs. 7.00 mg Fe/g, respectively).

No great difference between positive and negative Rh groups 6.89, 7.00 respectively whoever O-ve blood group showed the highest LIC

Table 7: mean LIC toward S ferritin profile of the patients

serum ferritin profile			**mean LIC**	**min**	**max**	**average**	**SD**
<1000	20	25.64%	3.47	297	900	505.7	
1001-2000	29	37.18%	6.81	1109	2000	1599.655	
2001-3000	18	23.08%	8.53	2112	3000	2795.235	
>3000	12	15.38%	10.12	3200	7324	4023	
Total	78	100.00%		297	7324	1952.551	±1283.57

Table 5 demonstrates a clear and progressive increase in mean LIC with rising serum ferritin categories. Mean LIC values ascended from 3.47 mg Fe/g in patients with S.F <1,000 µg/L (n=20) to 6.81 mg Fe/g (S.F 1,001–2,000 µg/L, n=29), 8.53 mg Fe/g (S.F 2,001–3,000 µg/L, n=18), and 10.12 mg Fe/g in those with S.F >3,000 µg/L (n=12). This dose-response relationship strongly supports a significant positive correlation between serum ferritin concentration and hepatic iron burden as measured by LIC, reinforcing the clinical practice of using serial serum ferritin measurements as a first-line monitoring tool in chronically transfused patients. Nonetheless, the potential confounding effects of acute-phase inflammatory responses and hepatic dysfunction on serum ferritin levels must be acknowledged when interpreting these associations.

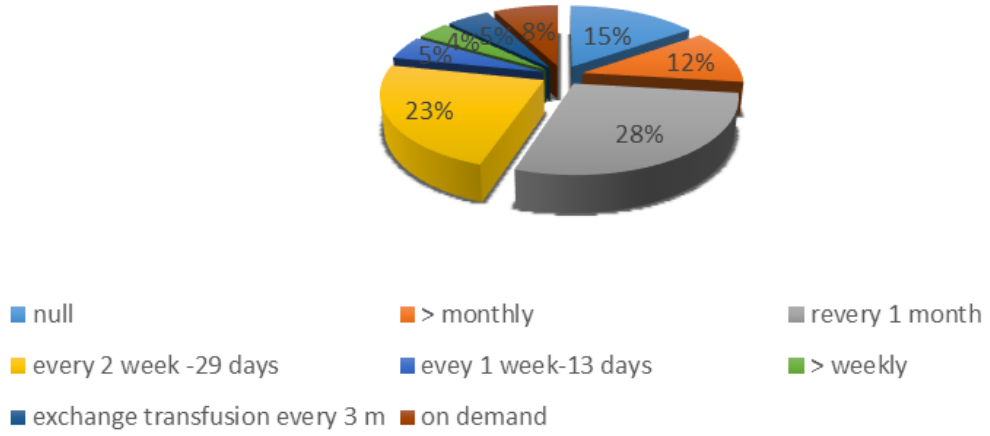

Fig 5: blood transfusion frequency for the studied patients

Graph 5 illustrates the heterogeneity of blood transfusion frequencies across the studied population. The predominance of monthly transfusion intervals (28.21%) is consistent with standard-of-care protocols for thalassemia major, with the goal of maintaining pre-transfusion haemoglobin levels above 9–10 g/dL. The subset of patients receiving transfusions every two weeks to 29 days (23.08%) likely includes more severely affected individuals, while the 'on-demand' group (7.69%) probably encompasses patients with milder phenotypes or sickle cell disease. The 15.38% classified as 'null' most likely represents patients who are transfusion-independent or who receive exchange transfusion only, highlighting the clinical diversity of the cohort.

Table 8: case distribution towards the annual blood transfusion required for the patients with mean LIC corelates

annual transfused blood	no	%	average Lic
<2000cc/year	34	43.59%	6.10
2001-4000	15	19.23%	7.16
4001-6000	9	11.54%	7.48
>6000	21	26.92%	7.65

Table 6 reveals a consistent positive trend between annual transfused blood volume and mean LIC. Patients receiving less than 2,000 cc/year had the lowest mean LIC (6.10 mg Fe/g), whereas those receiving the highest volumes (>6,000 cc/year) exhibited the greatest mean LIC (7.65 mg Fe/g). This incremental relationship underscores the pivotal role of cumulative transfusion burden as a primary determinant of hepatic iron loading and emphasizes the critical importance of tailoring chelation therapy intensity to each patient's individual transfusion requirements. The mean annual transfusion volume of 3,238.97 cc/patient/year (range: 0–7,800 cc) further illustrates the wide variability in transfusion exposure within this cohort.

Average of 3238.97 cc/patient/year min was 0 max was 7800 cc/patient/year

Table 9: Descriptive Analysis of Continuous Variables

Variable	Mean	SD	Median	Min	Max
Serum Ferritin (ng/l)	1971.42	1281.05	1800.00	297.00	7324.00
Haemoglobin (g/dl)	8.03	1.10	8.00	5.30	12.00
Platelets (×10³/µl)	474.09	481.20	378.00	150.00	4000.00
White Blood Cells (per mm³)	10680.52	4997.89	10000.00	290.00	29000.00
Body Weight (kg)	48.55	16.80	50.00	18.00	90.00
Body Height (m)	1.48	0.17	1.50	1.04	1.80
LIC (mg Fe/g dw)	6.88	4.57	6.50	1.14	24.00
T2* Relaxation Time (ms)	6.99	6.55	4.00	0.20	27.00
R2* Relaxometry (Hz)	274.56	198.53	250.00	46.00	950.00
Age (years)	20.43	11.28	16.00	9.00	57.00

SD = Standard Deviation. LIC categories (mg Fe/g dw): Normal <2, Mild 2–7, Moderate 7–14, Severe >14.

The mean serum ferritin level was 1971.42 ± 1281.05 ng/l, consistent with a chronically transfused population. Mean LIC was 6.88 ± 4.57 mg Fe/g dw, reflecting a predominance of mild-to-moderate hepatic iron burden. Mean T2* was 6.99 ± 6.55 ms and mean R2* was 274.56 ± 198.53 Hz, reflecting wide interindividual variability in iron deposition. A mean haemoglobin of 8.03 g/dl is consistent with the underlying haematological disorders managed on regular transfusion programmes.

Table 10: Association Between LIC Level and Categorical Patient Characteristics (Chi-Square Test) Iron Chelation Therapy

Group/Category	N	Normal (<2)	Mild (2–7)	Moderate (7–14)	Severe (>14)	p-value
DFX (Exjade)	42	1 (2%)	15 (36%)	21 (50%)	5 (12%)	<0.001 ***
DFO (Desferal)	8	0 (0%)	2 (25%)	4 (50%)	2 (25%)	
Hydroxyurea	11	6 (55%)	2 (18%)	3 (27%)	0 (0%)	
Combined	2	0 (0%)	1 (50%)	1 (50%)	0 (0%)	
No ICT	14	7 (50%)	6 (43%)	1 (7%)	0 (0%)	

*** Chi-square p-value

Table 11: Type of Anaemia and other variables

Group/Category	N	Normal (<2)	Mild (2–7)	Moderate (7–14)	Severe (>14)	p-value
Disease Category						
Thalassemia Major	40	2 (5%)	13 (32%)	20 (50%)	5 (12%)	
Thalassemia Intermedia	10	1 (10%)	3 (30%)	4 (40%)	2 (20%)	
Sickle Cell/Thalassemia	14	4 (29%)	4 (29%)	6 (43%)	0 (0%)	
Sickle Cell Disease	12	7 (58%)	5 (42%)	0 (0%)	0 (0%)	
Others	1	0 (0%)	1 (100%)	0 (0%)	0 (0%)	
Chi-square p-value						0.005**
Splenectomy Status						
Yes	38	6 (16%)	11 (29%)	18 (47%)	3 (8%)	0.526
No	39	8 (21%)	15 (38%)	12 (31%)	4 (10%)	
Sex						
Male	42	11 (26%)	15 (36%)	12 (29%)	4 (10%)	0.114
Female	35	3 (9%)	11 (31%)	18 (51%)	3 (9%)	
Comorbidities						
Present	17	1 (6%)	9 (53%)	6 (35%)	1 (6%)	0.211
Absent	60	13 (22%)	17 (28%)	24 (40%)	6 (10%)	

Chi-square test. LIC categories (mg Fe/g dw): Normal <2, Mild 2–7, Moderate 7–14, Severe >14. *p<0.05, **p<0.01, ***p<0.001, NS = Not Significan

The type of iron chelation therapy and type of haemoglobinopathy were both significantly associated with LIC category (p<0.001 and p=0.005, respectively). Patients on deferoxamine exhibited higher proportions of moderate-to-severe LIC, whereas those receiving hydroxyurea or no chelation predominantly had normal or mild iron loading, reflecting their lower transfusion burden. Sickle cell disease patients showed no moderate or severe LIC, in marked contrast to thalassaemia major. Sex, splenectomy status, and comorbidities showed no statistically significant association with LIC severity.

Table 12: Comparison of LIC (mg Fe/g dw) According to Different Patient Characteristics

Variable	Group	N	Mean ± SD	Median (IQR)	p-value	Statistical Test
Sex	Female	35	7.32 ± 3.76	7.10 (5.40–8.65)	0.104 NS	Mann–Whitney U
	Male	42	6.51 ± 5.16	5.75 (1.85–8.17)		
Type of Anaemia	Thalassemia Major	40	8.03 ± 4.04	7.80 (5.17–8.93)	<0.001 ***	Kruskal–Wallis
	Thalassemia Intermedia	10	9.04 ± 7.18	7.05 (6.35–7.73)		
	Sickle Cell/Thalassemia	14	5.37 ± 3.28	6.38 (1.77–7.47)		
	Sickle Cell Disease	12	3.06 ± 1.89	1.77 (1.58–5.22)		
	Others	1	6.50	6.50		
Iron Chelation Therapy	DFX (Exjade)	42	8.15 ± 3.92	7.30 (5.35–8.78)	<0.001 ***	Kruskal–Wallis
	DFO (Desferal)	8	10.66 ± 6.53	8.15 (6.92–10.52)		
	Hydroxyurea	11	4.08 ± 3.10	1.85 (1.65–7.10)		
	Combined	2	8.00 ± 2.40	8.00 (7.15–8.85)		
	No ICT	14	2.97 ± 2.35	1.85 (1.47–3.30)		
Splenectomy Status	Yes	38	7.20 ± 4.00	7.20 (5.00–9.15)	0.155 NS	Mann–Whitney U
	No	39	6.57 ± 5.09	6.30 (2.80–7.60)		
Comorbidities	Present	17	7.34 ± 3.30	6.50 (5.60–8.70)	0.350 NS	Mann–Whitney U
	Absent	60	6.75 ± 4.88	6.75 (2.55–8.20)		

Mann–Whitney U test (2-group comparisons) or Kruskal–Wallis H test (≥3 groups). IQR = interquartile range. *p<0.05, **p<0.01, ***p<0.001, NS = Not Significant.

The type of haemoglobinopathy and iron chelation regimen were the most influential determinants of LIC magnitude ($p<0.001$ for both). Thalassemia intermedia and thalassemia major carried the highest mean LIC (9.04 and 8.03 mg Fe/g dw, respectively), whereas sickle cell disease showed the lowest (3.06 mg Fe/g dw). Among chelation groups, patients on deferoxamine (DFO) demonstrated the highest LIC (10.66 ± 6.53), likely reflecting greater disease severity at the time of therapy initiation. Sex, splenectomy status, and comorbidities did not reach statistical significance, suggesting that transfusion burden and chelation adequacy are the primary drivers of hepatic iron loading in this cohort.

Table 13: Distribution of Studied Patients by Serum Ferritin Level (Dichotomous: < 3000 vs ≥ 3000 ng/l)

Serum Ferritin Category	Frequency (N)	Percentage (%)
< 3000 ng/l	57	72.7%
≥ 3000 ng/l	21	27.3%
Total	78	100.0%

Threshold of 3000 ng/l applied per study protocol; corresponds to clinically significant iron burden threshold.

Comment: The majority of patients (72.7%; n=56) maintained serum ferritin levels below 3000 ng/l, while 27.3% (n=21) presented with ferritin ≥3000 ng/l, identifying a subgroup with significant iron overload. This clinically relevant dichotomisation reflects the threshold above which end-organ iron toxicity risk is substantially elevated, particularly affecting cardiac and endocrine function.

Table 14: Comparison of MRI Parameters According to Serum Ferritin Level (< 3000 vs ≥ 3000 ng/l)

MRI Parameter	SF < 3000 ng/l (n=56) Mean ± SD	SF ≥ 3000 ng/l (n=21) Mean ± SD	p-value
LIC (mg Fe/g dw)	5.81 ± 3.49	9.74 ± 5.83	0.007
T2* Relaxation Time(ms)	7.99 ± 6.70	4.33 ± 5.41	0.011
R2* Relaxometry (Hz)	233.45 ± 166.92	384.19 ± 236.24	0.010

Mann–Whitney U test. *p<0.05, **p<0.01. SF = Serum Ferritin.

Comment: All three MRI-derived iron parameters differed significantly between ferritin groups. Patients with SF ≥3000 ng/l exhibited markedly higher LIC (9.74 ± 5.83 vs 5.81 ± 3.49 mg Fe/g dw; p=0.007) and R2* (384.19 ± 236.24 vs 233.45 ± 166.92 Hz; p=0.010), while T2* was correspondingly lower (4.33 ± 5.41 vs 7.99 ± 6.70 ms; p=0.011). These findings confirm that elevated serum ferritin is associated with greater hepatic iron deposition as quantified by MRI, supporting its utility as a screening surrogate while underscoring the need for direct MRI quantification in high-ferritin individuals.

Table 15: Comparison of MRI Parameters According to Serum Ferritin Categories (1000 ng/l Increments)

MRI Parameter	< 1000 (n=19)	1000–1999 (n=21)	2000–2999 (n=16)	3000–3999 (n=15)	4000–4999 (n=5)	≥ 5000 (n=1)	p-value (K-W)	Sig.
LIC (mg Fe/g dw)	3.47 ± 2.72 [1.80 (1.55–5.15)]	6.27 ± 2.56 [6.50 (5.00–7.80)]	7.98 ± 3.85 [7.15 (6.03–8.47)]	9.06 ± 5.09 [7.40 (6.75–9.40)]	12.10 ± 8.36 [8.80 (5.20–17.50)]	8.10	0.001	***
T2* (ms)	13.91 ± 7.18 [16.00 (7.20–19.00)]	5.81 ± 4.40 [4.00 (3.00–6.50)]	3.82 ± 2.76 [3.00 (3.00–4.25)]	4.79 ± 6.27 [3.00 (3.00–4.05)]	3.22 ± 2.42 [3.00 (1.50–5.50)]	3.00	<0.001	***
R2* (Hz)	123.75 ± 108.48 [63.00 (55.75–165.50)]	240.67 ± 101.14 [250.00 (200–300)]	354.25 ± 208.61 [285.00 (241–368.50)]	356.87 ± 208.71 [287.00 (244.50–367)]	480.60 ± 332.97 [357.00 (200–700)]	312.00	<0.001	***

Kruskal–Wallis H test across 6 ferritin strata. Values presented as Mean ± SD [Median (IQR)]. K-W = Kruskal–Wallis. *p<0.05, **p<0.01, ***p<0.001

A clear monotonic relationship was observed between serum ferritin strata and all three MRI iron parameters. LIC increased progressively from 3.47 ± 2.72 mg Fe/g dw in the <1000 ng/l group to 12.10 ± 8.36 mg Fe/g dw in the 4000–4999 ng/l group (Kruskal–Wallis p=0.001). Correspondingly, R2* increased from 123.75 ± 108.48 Hz to 480.60 ± 332.97 Hz, while T2* declined from 13.91 ± 7.18 ms to 3.22 ± 2.42 ms across the same range ($p<0.001$ for both). These trends demonstrate a robust gradient effect of ferritin level on hepatic iron burden, although the wide variability within each stratum — particularly at higher ferritin levels — underscores the limitation of ferritin as a precise quantitative surrogate for MRI-based iron measurement.

Table 16: Correlation Between Liver Iron Concentration (LIC) and Different Clinical/Laboratory Parameters

Parameter	Pearson r	p-value (Pearson)	Spearman ρ	p-value (Spearman)	Interpretation
Age (years)	0.018	0.879 NS	-0.132	0.252 NS	No significant correlation
T2* Relaxation Time (ms)	-0.705	<0.001 ***	-0.930	<0.001 ***	Strong negative correlation
R2* Relaxometry (Hz)	0.903	<0.001 ***	0.942	<0.001 ***	Very strong positive correlation
Serum Ferritin (ng/l)	0.485	<0.001 ***	0.497	<0.001 ***	Moderate positive correlation
Haemoglobin (g/dl)	-0.229	0.045 *	-0.189	0.100 NS	Weak negative (Pearson only)
Platelets (×10³/µl)	0.283	0.013 *	0.122	0.290 NS	Weak positive (Pearson only)
WBC (per mm³)	0.147	0.202 NS	0.154	0.182 NS	No significant correlation
Body Weight (kg)	-0.231	0.043 *	-0.193	0.093 NS	Weak negative (Pearson only)

Pearson (r) and Spearman (ρ) coefficients reported. *p<0.05, **p<0.01, ***p<0.001, NS = Not Significant.

R2* demonstrated the strongest correlation with LIC (Pearson r=0.903; Spearman ρ=0.942; p<0.001), confirming its primacy as the MRI biomarker for liver iron quantification. T2* showed a strong inverse correlation (r=−0.705; ρ=−0.930; p<0.001), consistent with the reciprocal T2*–iron relationship. Serum ferritin exhibited a moderate positive correlation with LIC (r=0.485; ρ=0.497; p<0.001), indicating clinical utility but imprecision as a standalone quantitative surrogate. Haemoglobin, platelet count, and body weight showed weak Pearson correlations that were not reproduced by the more robust Spearman test, suggesting these associations are influenced by outliers rather than reflecting true monotonic relationships. Age showed no significant correlation.

Table 17: Stratified Correlation Between Serum Ferritin and MRI Parameters by Ferritin Level

MRI Parameter	SF < 3000 ng/l (n=56) ρ (Spearman)	p-value	Sig.	SF ≥ 3000 ng/l (n=21) ρ (Spearman)	p-value	Sig.
T2* (ms)	-0.615	<0.001	***	-0.084	0.717	NS
R2* (Hz)	0.586	<0.001	***	0.114	0.623	NS
LIC (mg/g)	0.515	<0.001	***	0.122	0.599	NS

Spearman rank correlation (ρ). Stratified by binary ferritin threshold of 3000 ng/l. *p<0.05, **p<0.01, ***p<0.001, NS = Not Significant.

A striking divergence in correlation strength was observed between the two ferritin strata. In patients with SF <3000 ng/l, all three MRI parameters correlated significantly with serum ferritin: T2* (ρ=−0.615; p<0.001), R2* (ρ=+0.586; p<0.001), and LIC (ρ=+0.515; p<0.001). In marked contrast, none of these correlations reached statistical significance in the SF ≥3000 ng/l group (all p>0.59). This well-recognised 'plateau effect' at high ferritin levels reflects the saturation of ferritin as an iron-binding protein and the confounding influence of inflammation on ferritin synthesis. These findings underscore the fundamental limitation of serum ferritin as a quantitative biomarker in heavily iron-loaded patients, and reinforce the indispensability of MRI-based R2*/T2* quantification — particularly in patients with ferritin ≥3000 ng/l — for accurate assessment of hepatic iron burden and chelation therapy guidance.

he study enrolled a total of 78 patients with hemoglobinopathy, representing a cohort managed at a tertiary haematology centre in Basrah, 2024. The mean age of the study participants was 20.38 years (range: 9–57 years), reflecting a predominantly paediatric and young-adult population characteristic of transfusion-dependent haemoglobinopathies in this region. The mean haemoglobin concentration was 8.04 g/dl, consistent with the expected degree of chronic anaemia in patients receiving regular red cell transfusion therapy. Assessment of hepatic iron burden by MRI T2* relaxometry revealed a mean liver iron concentration (LIC) of 6.88 mg Fe/g dry weight, placing the cohort on average within the mild-to-moderate iron overload range (reference: mild 2–7; moderate 7–14 mg Fe/g dw). The mean serum ferritin level was 1,952.55 ng/l, corroborating the presence of significant systemic iron burden across the group, though with considerable interindividual variability. Collectively, these baseline characteristics confirm that this cohort represents a clinically relevant population with established transfusional iron overload, in whom accurate non-invasive quantification of hepatic iron by MRI carries direct implications for chelation therapy management and long-term organ protection.

Discussion

Since iron overload is a major side effect of repeated blood transfusions that used as a long-term treatment for patients with hemoglobinopathy, quantifying the amount of iron deposited in different organs (especially the liver) in patients receiving multiple transfusions is crucial for the efficient monitoring of iron chelation therapy. In the past, ferritin levels and biopsy were the only methods used to quantify the liver's iron concentration (18).

In order to identify clinically relevant changes in iron, it is necessary that iron levels be calculated with appropriate accuracy. Since it is commonly accepted to have adequately dependable accuracy and preciseness for clinical application, LIC computed from 1.5 T R2* is a widely employed approach (19).

This study is designed to assess the effectiveness of MRI in assessing iron deposition in the liver in patients with hemolysis. It studied 78 patients with β-thalassemia major (n=40), sickle-thalassemia (n=15), sickle cell disease (n=12), and thalassemia intermedia (n=10), with one additional patient in the 'other' category; 42 males and 36 females; age ranging from 2 to 60 years; serum ferritin ranging from 95 to >7,000 ng/mL; the majority of thalassemic patients were on regular blood transfusion and on iron chelation therapy.

The distribution of LIC grades in the present study demonstrated that only 16.88% of patients exhibited normal LIC values (0.17–1.8 mg Fe/g dry weight), while the large majority (77.92%) showed clinically significant hepatic iron overload: 33.77% mild (3.2–7 mg Fe/g), 35.06% moderate (7–15 mg Fe/g), and 9.09% severe (>15 mg Fe/g). This high prevalence of iron overload is consistent with findings in similar transfusion-dependent populations worldwide. Musallam *et al*. (21) and Cappellini *et al*. (22) both reported that the majority of chronically transfused patients accumulate at least mild-to-moderate hepatic iron regardless of ongoing chelation therapy, highlighting the inadequacy of current chelation practices in many real-world settings.

This study shows no significant correlation between the level of LIC with age and sex. This agrees with many previous studies such as Pakbaz *et al*. (20), Musallam *et al*. (21), and Cappellini *et al*. (22), who reported that there was no clear correlation between any hemoglobinopathy patient characteristics and LIC.

There was also no significant correlation between mean LIC and splenectomy, which simulates results in other research done by Papakonstantinou *et al*. (23), Angelucci *et al*. (24), and El Shanshory *et al*. (25), who reported that splenectomy and its timing had little insignificant effect on LIC and total body iron. This differs from Musallam *et al*. (21), who showed that mean LIC was higher in splenectomized patients than in non-splenectomized ones.

Notably, the present study identified a meaningful difference in mean LIC between patients residing in central urban areas and those from peripheral regions (8.99 vs. 5.92 mg Fe/g dry weight, respectively). This observation likely reflects a referral-pattern effect, whereby patients with more severe

disease phenotypes and higher cumulative transfusion requirements are disproportionately concentrated at tertiary and quaternary care centers in urban settings. Additionally, disparities in access to iron chelation agents, regularity of follow-up, and availability of MRI-based LIC monitoring in peripheral areas may compound the higher iron burden observed in centrally registered patients. These findings align with broader evidence documenting significant geographic and socioeconomic inequities in the management of iron overload among hemoglobinopathy populations globally (34).

This study shows a significant correlation between LIC and increased frequency of blood transfusion. This agrees with Musallam *et al*. (21), who showed that the mean LIC was higher in regularly transfused (9.7±6.7 mg Fe/g dw) or occasionally transfused (9.9±7.2 mg Fe/g dw) patients than in non-transfused patients (4.3±3.1 mg Fe/g dw) (P<0.001). This also explains why β-thalassemia major has a high level of LIC, because most patients have frequent blood transfusions every 2 weeks to 1 month.

Furthermore, when the cumulative annual transfused blood volume was examined, a graded positive association with mean LIC was observed: patients receiving <2,000 cc/year had the lowest mean LIC (6.10 mg Fe/g), increasing progressively to 7.16 mg Fe/g (2,001–4,000 cc/year), 7.48 mg Fe/g (4,001–6,000 cc/year), and 7.65 mg Fe/g (>6,000 cc/year). This dose-dependent relationship between cumulative iron input and hepatic loading is well recognized. Anderson (19) and Fahmy *et al*. (18) have both demonstrated that the rate of hepatic iron accumulation is directly proportional to total transfusion volume, and that chelation therapy intensity must therefore be individually tailored to the patient's annual transfusion burden in order to prevent progressive iron-mediated organ damage.

Additionally, this study revealed a positive association between LIC and R2* and a negative correlation between LIC and T2*. These results were in line with those of other studies by Fahmy *et al*. (18), Anderson (19), and El Shanshory *et al*. (25), which found that LIC is significantly correlated with T2* and R2* and that the greater the liver iron overload, the greater the decrease in Signal Intensity (SI) on MR images, which is represented by T2* as the liver parenchyma darkens progressively with increased TE.

This study shows a moderate correlation of serum ferritin and LIC. This correlation decreases with increasing serum ferritin levels, which is similar in comparison to Majd *et al*. (26), who showed that serum ferritin has a statistically significant positive correlation with LIC and a significant negative correlation with T2* of the liver. Similarly, Azarkeivan *et al*. (27), Kolnagou *et al*. (28), and Zamani *et al*. (29) showed a moderate correlation between LIC and serum ferritin. Assis *et al*. (30) demonstrated an excellent correlation between serum ferritin and T2* MRI liver.

On the other hand, in contrast to this study, a poor negative correlation between serum ferritin level and T2* MRI liver was observed by Eghbali *et al*. (31) and Fahmy *et al*. (18).

These results are further explained by the fact that serum ferritin is an acute phase reactant produced in response to inflammation. Interpreting serum ferritin values can be challenging due to a number of conditions that alter concentrations independently of changes in the body's iron burden, such as fever, hemolysis, ineffective erythropoiesis, and acute and chronic hepatic damage, all of which are common in patients with β-thalassemia major and other hemoglobinopathies (20).

However, this study noticed that in patients with serum ferritin <3,000 ng/L, a statistically significant moderate negative correlation was detected between serum ferritin and T2* ($r = -0.655$, $P = 0.001$), and moderate positive correlations were detected between serum ferritin and both R2* and LIC. In patients with serum ferritin ≥3,000 ng/L, no significant correlations were found ($P \geq 0.05$) (19 patients had high serum ferritin ≥3,000; 3 presented with mild LIC and 8 with moderate LIC). This is similar to studies by Azarkeivan *et al*. (27) and Neufeld (32), who reported that the correlation between serum ferritin and liver T2* and LIC is greatly weakened in patients with serum ferritin level >4,000 ng/mL (they submit that the maximum rate of synthesis and release of glycosylated ferritin by reticuloendothelial cells, once fully saturated, may be responsible for this phenomenon).

This variation in results can be explained by differences in clinical, genetic, and demographic characteristics of the study population such as age, sample size, serum ferritin levels, chelating protocols, iron kinetics of different organs, type of machine used in the study, and MR method used in LIC quantification.

The present study additionally revealed that patients with abnormal liver biochemical profiles (hepatopathies) exhibited a higher mean LIC (7.17 mg Fe/g) compared to those with normal hepatic function (5.39 mg Fe/g). This bidirectional relationship between hepatic iron deposition and hepatocellular injury is well-established: progressive iron accumulation in hepatocytes drives oxidative stress, lipid peroxidation, and inflammatory cascades that ultimately culminate in hepatic fibrosis and, in severe cases, cirrhosis. Furthermore, hepatitis co-infection, present in a subset of patients, was associated with a mean LIC of 5.50 mg Fe/g. These observations are consistent with those of Kanbour *et al*. (33), who demonstrated significant correlations between elevated LIC and elevated liver enzymes in patients with β-thalassemia major, underscoring the importance of early chelation intensification in patients showing biochemical evidence of hepatic injury. Regarding ABO blood group distribution, no clinically meaningful difference in mean LIC was observed across blood groups, and the small sample sizes within certain groups preclude definitive conclusions.

Also, this study noticed a high LIC level in patients who used the intravenous iron chelating agent deferoxamine (DFO). This is incompatible with TIF guidelines for the management of transfusion-dependent thalassemia (3), which suggest that in patients with LIC values >14 mg/g dry weight, a mean dose of 51 mg/kg resulted in LIC decreases of an average of 6.4 mg/g dry weight, and thus a dose of 50 mg/kg at least 5 days a week is recommended if a significant decrease to optimal LIC levels is required (3). This can be explained by the fact that the treatment is costly and inconvenient, requiring subcutaneous or intravenous infusion over at least 8 hours a day at least 5 days a week in regularly transfused patients. Adherence to therapy has been the main limiting factor to successful outcomes (3). Also, the drug is not always available and is irregularly taken by the patients.

This study shows no correlation between LIC and other comorbidities such as cardiomyopathy and heart failure, diabetes mellitus, and endocrinopathies. This disagrees with Musallam *et al*. (21), who showed that elevated liver iron concentration in patients with β-thalassemia intermedia is a marker of increased vascular, endocrine, and bone disease. It

also disagrees with Kanbour *et al*. (33), who found that a significant number of β-thalassemia major patients with high LIC and endocrine disorders still exist despite the recent development of new oral iron chelating agents.

This variation can be explained by the fact that this study involves four types of hemoglobinopathy (sickle cell disease, thalassemia major, thalassemia intermedia, and sickle-thalassemia). Furthermore, there was a large geographic variation in the study population. Most of the previous studies have been conducted either in the Middle East, Europe, or South Asia.

Conclusion

In conclusion, MRI R2* provides a non-invasive, precise, and repeatable method for measuring hepatic tissue iron content. The treatment of tissue iron excess should be improved by the widespread use of this approach, enabling early chelation intensification & monitoring of iron level in response to treatment

Recommendation

1. MRI is effective in management of hemoglobinopathy so,repeat liver MRI for all patients who have LIC > 7mg/g DW liver (every 6 months,or 1year depending on guidelines
2. Cardiac MRI is required for all patient to exclude cardiac iron deposition.
3. Increase sample size is needed, collect and analyse multiple data on multi-centre national or regional base

Abbreviation	Explanation
LIC	Liver Iron Concentration.
RES	Reticular Endothelial System.
SCD	Sickle Cell Disease
GI	Gastrointestinal
NTBI Iron	Non Transferrin Bond
SF	Serum Ferritin
TE	Echo Time
ROI	Region of Interest
ICT	Iron Chelating Treatment
DFX	Deferasirox (Exjade)
DFO	Deferoxamine (desferal)

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How to Cite This Article
Abdul Ameer HR, Mousa HJ, Badr FH, Alhijaj BA. MRI estimation of liver iron concentration in patients with hemoglobinopathy in relation to serum ferritin in Basrah. International Journal of Medical and All Body Health Research. 2026;7(2):192–205.
doi:10.54660/IJMBHR.2026.7.2.192-205.

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