Cardiac Complications in Iraqi Children with Beta-Thalassemia Major: Association with Serum Ferritin and Chelation Adequacy

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Abstract

Background: Beta thalassemia major (BTM), an inherited hemoglobinopathy, is characterized by absent or severely reduced β-globin chain synthesis, resulting in transfusion-dependent anemia that begins early in life. Cardiac complications, primarily iron-overload cardiomyopathy and congestive heart failure, remain the most common cause of mortality in these patients despite several decades of advances in supportive care.

Objective: To investigate the cardiac function in Iraqi children with BTM using ECG and Echocardiography, to determine whether abnormal findings of these tests are related to inadequate chelation therapy (assessed by urine drug levels) and body iron overload (estimated by serum ferritin), and to assess the correlation between CXR parameters versus echocardiographic measurements.

Methods: A cross-sectional study of 49 patients with beta-thalassemia major (BTM) disorder followed up at the outpatient clinic of AL-Zahraa Teaching Hospital, Al-Najaf, Iraq, from June to November 2025, comprising 29 males and 20 females aged between 10 months and 17 years.) ECG, transthoracic echocardiography, and CXR were performed in all the patients. We defined chelation adequacy as deferoxamine use≥ 4 days/week (optimal) or <4 days/week (suboptimal). Statistical analysis: Chi-square and Student's t-tests were applied using SPSS v20 (Significance level $P < 0.05$).

Results: Among the 42 chelated patients, ECG abnormalities were paradoxically more frequent in those receiving optimal chelation (62.5%) than in those on suboptimal regimens (30.8%; $P = 0.04$). Cardiomegaly on CXR followed a similar pattern (25% vs. 7.7%, $P > 0.05$), as did pericardial thickening on echocardiography (50% vs. 42.3%). In contrast, serum ferritin level showed a clear dose-response relationship with cardiac burden: patients with ferritin ≥1000 ng/mL had significantly higher rates of cardiomegaly (29.4% vs. 3.1%, $P < 0.05$) and ECG abnormalities (70.6% vs. 18.8%, $P < 0.001$) than those with lower ferritin. Six patients (12%) had clinically overt congestive heart failure.

Conclusions: Cardiac involvement is substantial in this Iraqi BTM cohort, and serum ferritin is a strong correlate of cardiac abnormalities. The paradoxically higher prevalence of ECG changes and cardiomegaly among optimally chelated patients most likely reflects confounding by indication — cardiac abnormalities prompting intensification of an already-interrupted regimen — rather than any direct effect of treatment. These findings underscore the need for early, uninterrupted chelation and regular cardiac surveillance in this population.

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Keywords: Beta Thalassemia Major, Cardiac Dysfunction, Chelation Therapy

Introduction

Beta-thalassemia major (BTM) is an autosomal-recessive hemoglobinopathy that results from reduced (β+) or absent (β0) synthesis of the β-globin chain. Imbalance in α- and β-globin chains causes ineffective erythropoiesis, severe hemolytic anemia, and transfusion dependence from late infancy [1–3]. β-Thalassemia is one of the most common genetic disorders worldwide, affecting an estimated 3% of the world's population, with the highest burden occurring in the Mediterranean basin, the Middle East, and south-eastern Asia [1, 2]. The introduction of routine transfusion programs with iron chelation markedly altered the

natural history of this disease, turning what once was a uniformly fatal illness of childhood into a chronic disease compatible with survival into adulthood [3].

Nevertheless, cardiac complications are still the leading cause of death in BTM and account for more than half of all deaths in transfused patients [4, 5]. Iron-overload cardiomyopathy is the central mechanism: When hepatic and splenic storage capacity is exhausted, non-transferrin-bound iron gradually accumulates in the myocardium, leading to systolic and diastolic dysfunction [6, 7]. Chronic anemia superimposes an independent second insult through a persistent hyperdynamic state — diminished peripheral vascular resistance, increased venous return, and elevated cardiac output — that over several years drives ventricular dilation and ultimately contractile dysfunction [8].

The most convenient proxy of iron stores is serum ferritin, a commonly used biomarker for this purpose. Only 25% of patients with sustained values >2500 ng/mL were free of cardiac disease at 5 years, while peak troponin levels ≤1000 ng/mL significantly decreased the likelihood of developing heart failure [7, 8]. Notably, subclinical iron-induced cardiomyopathy progresses slowly — overt symptoms may not become clear for many years — and illustrates the necessity of serial cardiac evaluation [9, 10]. For example, in resource-limited settings, the practical monitoring toolkit includes twelve-lead ECG, chest radiography and TTE-FS%/EF [11–12]. Overall, cardiac MRI T2* is considered the gold standard for myocardial iron quantification [13], and values < 20 ms predict the development of progressive left ventricular dysfunction, but this modality is still often unavailable in other parts of the Middle East and many low- or middle-income countries.

The availability of systematic data on cardiac function in children with BTM in Iraq is limited, and the interaction among chelation adequacy, ferritin levels, pre-transfusion hemoglobin, and other relevant clinical factors is poorly characterized for risk-stratifying patients for optimal management. Because MRI T2* is not routinely available in our setting, the bedside ECG–chest radiography–echocardiography triad is the only practical monitoring strategy, and its real-world power to reveal subclinical cardiac involvement warrants further scrutiny. In this context, we aimed to characterize the burden of cardiac abnormalities in a contemporary cohort of Iraqi pediatric patients with BTM and their association with chelation adequacy, serum ferritin, and pre-transfusion hemoglobin.

Patients and Methods

Study Design

We enrolled 49 patients with confirmed beta thalassemia major attending the Thalassemia Center at AL-Zahraa Teaching Hospital for Maternity and Children, Al-Najaf Al-Ashraf, Iraq, in this cross-sectional study conducted between 1 June and 1 November 2025. Participants were recruited by convenience sampling from patients presenting for routine transfusions.

Eligible patients had confirmed BTM (diagnosed by hemoglobin electrophoresis with HbF >90%), were aged 10 months to 17 years, and were registered at the center with an established regular transfusion schedule. We excluded patients with congenital heart disease, other hemoglobinopathies (e.g., sickle-β thalassemia), acute febrile illness at the time of assessment, or incomplete medical records.

1. Medical History

History taking was conducted in a structured manner with the parents, and the history records were reviewed. Age, sex, address (for population background comparisons), age at diagnosis (letter), age at first blood transfusion (letter), number of transfusions per year since the start of treatment, age and frequency on deferoxamine onset use current frequency in dollars/ week/day) Pre-transfusion hemoglobin status, most recent agreed by mail to 7 or splenectomy status.

2. Physical Examination

All patients received a complete physical examination. Anthropometric measures (weight & height), pallor, jaundice and thalassemic facies were evaluated. Hepatomegaly and splenomegaly were assessed by abdominal examination as centimeters below the costal margin, if splenectomy was performed or not. Cardiovascular examination involved heart rate, blood pressure, jugular venous pressure, cardiac murmurs, gallop rhythm, pedal edema and clinical signs of congestive heart failure.

3. Investigations

Thorough laboratory investigations were performed in all cases, including complete blood count (CBC) with peripheral smear review, hemoglobin electrophoresis, serum ferritin measurement, liver function tests, posteroanterior chest radiography, 12-lead electrocardiogram (ECG), and transthoracic echocardiography. For pre-transfusion hemoglobin, we extracted the value from the most recent clinic visit prior to enrollment.

3.1. Serum Ferritin

Serum ferritin was measured by ACC U-BIND ELISA. Fasting morning venous samples were collected in plain red-top tubes (no additive or anticoagulant) allowing for clotting at room temperature followed by centrifugation for serum separation. Results are expressed in ng/mL.

3.2. Chest X-Ray

Posteroanterior chest radiographs were obtained with a Shimadzu unit (Model R-20J, No. 0166M14645, Japan) at standard settings (approximately 110–125 kVp; effective dose ~0.1 mGy), with the patient erect at a source-to-film distance of 6 feet. Cardiomegaly was defined as a cardiothoracic ratio exceeding 50%.

3.3. Electrocardiography (ECG)

Twelve-lead ECGs were recorded using a KENZ Cardico 302 machine (Japan). Each tracing was interpreted for rate, rhythm, axis, QRS voltage and duration, P-wave morphology, PR interval, ST-segment changes, T-wave abnormalities, and ventricular hypertrophy. Left ventricular hypertrophy (LVH) was defined by increased QRS voltage in the left leads at normal QRS duration — R wave in V6, I, aVL, and V5, and S wave in V1 each exceeding the 98th percentile for age — in association with left axis deviation and inverted T waves in V6, I, and/or aVF. Right ventricular hypertrophy (RVH) required both an R wave in V1 and an S

wave in V6 above the 98th percentile, together with an upright T wave in V1 [14].

3.4. Echocardiography

Transthoracic echocardiography was performed at AL-Zahraa Teaching Hospital using a GE Logiq system, incorporating M-mode, two-dimensional, pulsed-wave, and continuous-wave Doppler modalities. Systolic ventricular function was characterized by fractional shortening (FS%, normal range 28–40%) and ejection fraction (EF%, normal ≥55%).

Statistical Analysis

All analyses were performed using SPSS version 20. Categorical variables are summarized as counts and percentages; continuous variables as mean ± standard deviation. Associations between categorical variables were evaluated by the chi-square test, or Fisher's exact test when any expected cell count fell below 5; group means were compared with Student's independent t-test. Odds ratios with 95% confidence intervals are reported where appropriate. Statistical significance was set at $P < 0.05$ throughout.

Ethical Considerations

The study was approved by the Scientific and Ethical Committee of the College of Medicine, University of Kufa. Given the routine, non-interventional nature of the assessments and the variable literacy levels of participating families, the committee approved verbal rather than written parental consent, which was obtained from all parents or legal guardians prior to enrollment. Children aged ≥7 years who were capable of doing so also provided verbal assent. All participant data were handled confidentially in accordance with institutional policy.

Results

The cohort comprised 49 patients (29 males [59.2%], 20 females [40.8%]) with a mean age of 8.6 ± 4.2 years (range 10 months to 17 years); the 7–10-year age group was the largest, accounting for 38.9% of participants. Of the 42 patients (85.7%) receiving deferoxamine, 16 (38.1%) were on an optimal regimen (≥4 days/week) and 26 (61.9%) on a suboptimal one (<4 days/week); a further 7 patients (14.3%) were not on chelation at the time of assessment. Serum ferritin was ≥1000 ng/mL in 17 patients (34.7%). Six patients (12.2%) had undergone splenectomy. Congestive heart failure was present in 6 patients (12%). Mean FS across the cohort was 34.2 ± 5.1% and mean EF was 65.8 ± 7.3%; four patients (8.2%) had values below the lower limit of normal. Tables 1–3 stratify cardiac findings by chelation adequacy (42 chelated patients only); Tables 4–6 include the full cohort, stratified by ferritin level and pre-transfusion hemoglobin.

Table 1: ECG findings by chelation adequacy among 42 chelated BTM patients

Deferoxamine frequency	Normal ECG, n (%)	Abnormal ECG, n (%)	Total	P value
< 4 days/week	18 (69.2)	8 (30.8)	26	0.04*
≥ 4 days/week	6 (37.5)	10 (62.5)	16	
Total	24	18	42	

ECG, or electrocardiography. *P value calculated using the chi-square test; statistical significance set at $P < 0.05$.

Table 2: Cardiomegaly on chest radiograph by chelation adequacy among 42 chelated BTM patients

Deferoxamine frequency	Cardiomegaly present, n (%)	Cardiomegaly is absent. n (%)	Total	P value
< 4 days/week	2 (7.7)	24 (92.3)	26	0.18*
≥ 4 days/week	4 (25.0)	12 (75.0)	16	
Total	6	36	42	

CXR, chest radiograph. *P value calculated using Fisher's exact test (expected cell count < 5); statistical significance set at $P < 0.05$.

Table 3: Echocardiographic findings by chelation adequacy among 42 chelated BTM patients

Deferoxamine frequency	Normal, n (%)	Pericardial thickening, n (%)	Other abnormalities, n (%)	Total	P value
< 4 days/week	12 (46.2)	11 (42.3)	3 (11.5)	26	0.62*
≥ 4 days/week	5 (31.3)	8 (50.0)	3 (18.7)	16	
Total	17	19	6	42	

Other abnormalities included left ventricular hypertrophy, biventricular dilatation, and mild mitral valve prolapse. *P value calculated using the chi-square test; statistical significance set at $P < 0.05$.

Table 4: Cardiomegaly on chest radiograph by serum ferritin level among all 49 BTM patients

Serum ferritin (ng/mL)	Cardiomegaly present, n (%)	Cardiomegaly is absent. n (%)	Total	P value
< 1000	1 (3.1)	31 (96.9)	32	0.01*
≥ 1000	5 (29.4)	12 (70.6)	17	
Total	6	43	49	

CXR, chest radiograph. *P value calculated using Fisher's exact test (expected cell count < 5); statistical significance set at $P < 0.05$.

Table 5: ECG findings by serum ferritin level among all 49 BTM patients

Serum ferritin (ng/mL)	Abnormal ECG, n (%)	Normal ECG, n (%)	Total	P value
< 1000	6 (18.8)	26 (81.3)	32	< 0.001*
≥ 1000	12 (70.6)	5 (29.4)	17	
Total	18	31	49	

ECG, or electrocardiography. *P value calculated using the chi-square test; statistical significance set at P < 0.05.

Table 6: Cardiomegaly and ECG findings by pre-transfusion hemoglobin level among all 49 BTM patients

Pre-transfusion Hb (g/dL)	Cardiomegaly, n (%)	Abnormal ECG, n (%)	Total	P value
< 9.5	6 (14.0)	15 (34.9)	43	> 0.05*
≥ 9.5	0 (0.0)	3 (50.0)	6	
Total	6	18	49	

Hb, hemoglobin; CXR, chest radiograph; ECG, electrocardiography. *P value calculated using Fisher's exact test; statistical significance set at P < 0.05.

Table 7: Distribution of ECG abnormalities and associated findings among the 18 BTM patients with abnormal ECG

ECG abnormality	n	Ferritin ≥ 1000 ng/mL	Ferritin < 1000 ng/mL	Normal FS	Increased FS	Cardiomegaly present	Cardiomegaly is absent.
LVH	12	8	4	10	2	2	10
Biventricular hypertrophy	1	1	—	—	1	1	—
LVH + LAH	1	1	—	—	1	1	—
Sinus tachycardia	4	2	2	2	2	2	2
Total	18	12	6	12	6	6	12

LVH, left ventricular hypertrophy; LAH, left atrial hypertrophy; FS, fractional shortening; CXR, chest radiograph. Cardiomegaly is absent; the total includes 12 patients. The cardiomegaly column totals 6 of 18 patients with abnormal ECG who also had cardiomegaly on CXR.

Among the 18 patients with abnormal ECG (Table 7), LVH was by far the most common finding, present in 12 patients (66.7%), followed by sinus tachycardia in 4 (22.2%). Interestingly, the majority of LVH patients — 10 of 12 (83.3%) — had preserved fractional shortening, suggesting that volume-related hypertrophy from chronic anemia rather than systolic dysfunction was the dominant mechanism at this stage. The remaining 2 LVH patients showed elevated FS consistent with a hyperdynamic state.

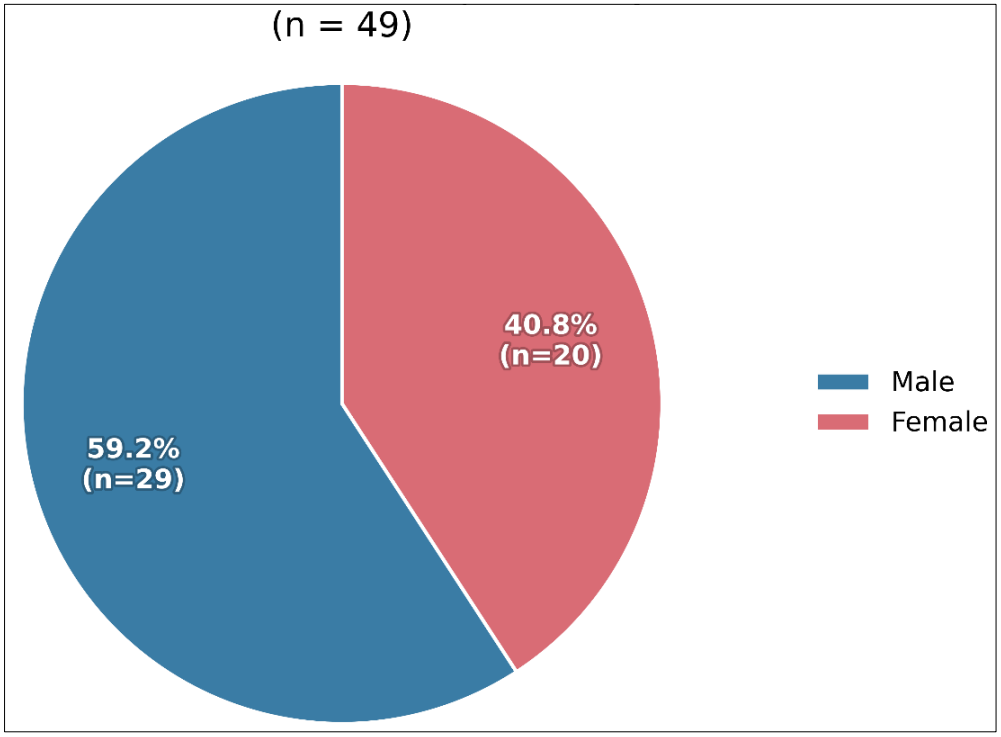

Fig 1: Distribution of BTM patients by sex—males 59.2%, females 40.8%.

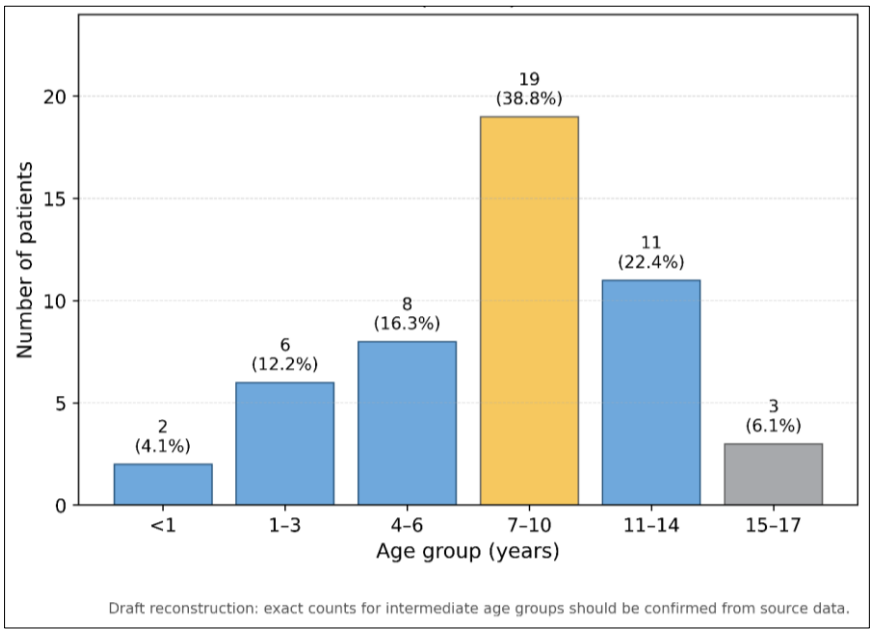

Fig 2: Distribution of thalassemic patients by age group. Six age groups were arranged by number and percentage. The largest age group (7–10 years) accounted for 38.9% of the total, while the smallest group (15–17 years) accounted for 6.1%.

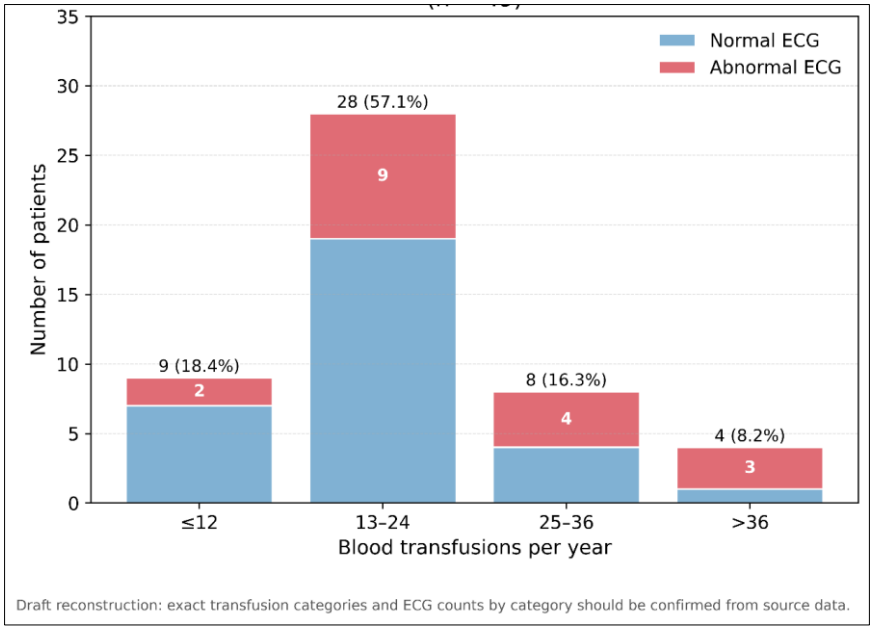

Fig 3: Distribution of BTM patients according to frequency of blood transfusion per year. The majority received 13–24 transfusions per year (28 patients), with several patients having abnormal ECGs.

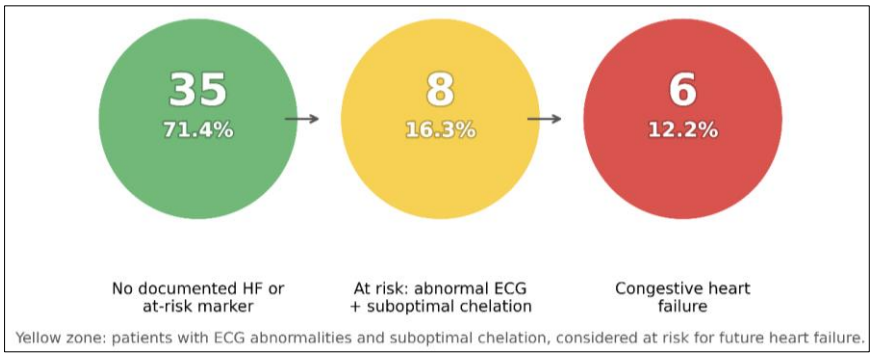

Fig 4: Final distribution of 49 BTM patients in three zones regarding cardiac complications. The yellow region represents patients with ECG abnormalities and suboptimal chelation, who were considered at risk for developing heart failure in the future.

Discussion

Clinically overt congestive heart failure was seen in 6 of our 49 patients (12%); half men and half women, and all except one in those with ferritin ≥1000 ng/mL on high frequency transfusion schedules. This proportional mortality rate lies between rates of 13.5% observed in a Greek multicenter cohort [16] and 15.6% from a Pakistani series [15] suggesting, even considering substantial variation in cardiac care systems and access to treatment across populations, broadly similar burden of cardiac disease amongst these settings.

When chelation was stratified by adequacy, a notable, albeit somewhat surprising, pattern emerged in cardiac findings. In F317(A0532), of the 42 chelated patients, ECG abnormalities (62.5% vs. 30.8%), echocardiographic abnormalities (68.7% vs. 53.8%), and cardiomegaly on CXR (25.0% vs. 7I77%) were significantly more frequent in those receiving optimal deferoxamine (=4 days/week) than those receiving suboptimal treatment (0.05). Importantly, 87.8% of the cohort was under-transfused relative to standard targets, and thus chronic anemia — via the persistent hyperdynamic state described above — may act independently of iron overload to sustain cardiac remodeling. This may also explain ECG abnormalities—mainly LVH (Table 7)—recorded in patients across a broad ferritin range (790–5000 ng/mL), with the anemic drive overlapping, in many cases, with the iron-overload drive.

Methodological note on denominators: Tables 1 and 2 are limited to patients on active chelation (n = 42) and compare only treated patients to analyze cardiac outcomes; thus, subjects receiving no chelation (n = 7) were excluded from these analyses. In contrast, Tables 4–6 presented analyses of all 49 subjects and broadly address the relationship between cardiac status (cardiac iron load/severity) with ferritin level and transfusion adequacy. The paradox in Tables 1–3 is most probably confounding by indication — patients with cardiac abnormalities seen during monitoring were subsequently up-titrated to optimal schedules, not higher chelation frequency causing the abnormalities. Similar patterns have been observed in other longitudinal cohorts of thalassemia [18, 19]. This emphasizes an inherent limitation with observational cross-sectional data: it is not possible to assume a causal interpretation of the relationship between cardiac alterations and whether or not chelation was intensified before or after these changes appeared. Future prospective studies with a detailed history of exposure to chelating agents and serial cardiac evaluations are needed to disentangle these temporal relationships.

Comparison Study

Table 8: Comparison of cardiac findings in the present study with previously published cohorts

Cardiac finding	Present study	Comparison study	Reported rate
Clinically overt heart failure	12.2% (6/49)	Khan & Mahsud, Pakistan, 2006 [15]	15.6%
		Aessopos *et al*., Greece, 2001 [16]	13.5%
Cardiomegaly on CXR	12.2% (6/49)	Muhsin, Baghdad, 2002 [17]	14%
		Khan & Mahsud, Pakistan, 2006 [15]	25.5%
ECG abnormalities	36.7% (18/49)	Muhsin, Baghdad, 2002 [17]	40%
		Aessopos *et al*., Greece, 2001 [16]	6.9%
		Khan & Mahsud, Pakistan, 2006 [15]	42%
Echocardiographic abnormalities	59.5% (25/42 chelated)	Muhsin, Baghdad, 2002 [17]	20%

CXR, chest radiograph; ECG, electrocardiography. Denominators differ across rows: heart failure, cardiomegaly, and ECG abnormalities are calculated over all 49 enrolled patients; echocardiographic abnormalities are calculated over the 42 chelated patients and represent any abnormal finding on TTE (pericardial thickening, left ventricular hypertrophy, biventricular dilatation, or mitral valve prolapse).

Limitations

Several limitations should be mentioned in interpreting our findings. The sample is relatively small (n = 49), which limits statistical power and generalizability outside the study population. The within-person design, by construction, however, cannot establish the direction of causation between iron overload/chelation practice and cardiac outcomes — a particularly important caveat given the above-described confounding-by-indication pattern. While the reference standard for myocardial iron quantification is cardiac MRI T2, this imaging approach was unavailable at our center; echocardiography and serum ferritin therefore represent indirect (albeit imperfect) markers of myocardial iron burden. Two limitations regarding chelation adequacy — self-reported deferoxamine frequency (which is to some extent prone to recall bias) and the lack of systematic cumulative chelation dose captures — together result in an incomplete picture of how well we are able to account for the paradoxical aspects of the findings related to chelation. This calls for future studies to be conducted in larger prospective cohorts using cardiac MRI T2, longitudinal follow-up, and validated adherence measures to better define future iron-related mediators of this growing long-term health burden facing Iraqi children with BTM.

Conclusions

A notable characteristic of this cohort was clinically relevant cardiac complications, including overt congestive heart failure in 12% of Patients. The observed prevalence of cardiac disease among under-transfused patients indicates that transfusion practices at the center are not uniformly reassured through a structured program, thereby suggesting an important modifiable factor in need of urgent attention.

Most patients with cardiac abnormalities were apparently receiving chelation therapy, but most had serum ferritin ≥1000 ng/mL — a pattern which more closely resembled undertreated or interrupted rather than genuine treatment failure. The key drivers are likely to be poor adherence and gaps in drug availability.

Lastly, in this disease, it is genuinely difficult to disentangle the contribution of chronic anemia from that of iron overload to myocardial dysfunction. This observation of cardiac abnormalities in patients with ferritin <1000 ng/mL emphasizes the role for an anemia-driven hyperdynamic state as a disconnection between iron overload and poor prognosis: although both pathways can likely coexist in many patients,

this suggests an independent mechanism.

Recommendations

- This supports the individualized clinical assessment for each patient with prespecified pre-transfusion Hb thresholds together with clear documentation of definitive target Hb level.
- Early, ongoing iron chelation with vitamin C and folic acid as supplements. Serum ferritin levels need to be assessed frequently, at least once every 3–6 months, so that the dose can either be increased if serum ferritin is too low or decreased when high.
- All patients, particularly those with inadequate/transfusion or chelation, need cardiac surveillance by periodic ECG, echocardiography and chest radiography at intervals adapted to clinical risk.
- Hospitalization of patients with congestive heart failure or cardiomyopathy and proper treatment admission.
- Health education for parents of thalassemic patients to enhance knowledge about the disease, prenatal diagnosis and genetic counseling.

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