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ORIGINAL RESEARCH INTENSIVE CARE

Comparative efficacy of iron chelation therapies in thalassemia major: a randomized controlled trial

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ABSTRACT

Background & objective: Thalassemia major, a transfusion-dependent genetic disorder, leads to iron overload in the body and necessitates frequent sessions of iron chelation therapy. Despite the availability of deferoxamine (DFO), deferiprone (DFP), and deferasirox (DFX), the optimal chelation strategies remain controversial.

We compared the efficacy and safety of DFO, DFP, and DFX in reducing iron overload parameters, including serum ferritin, liver iron concentration (LIC), and cardiac T2* MRI. We also compared the adverse events with the use of these three agents.

Methodology: A 12-month randomized controlled trial included 150 thalassemia major patients, randomized to receive either DFO (40–50 mg/kg/day subcutaneously), DFP (75 mg/kg/day per os), or DFX (20–40 mg/kg/day per os). Serum ferritin level, LIC, cardiac T2* MRI, and adverse events were assessed. Statistical analyses were performed using ANOVA) and chi-square tests.

Results: DFX showed the greatest reduction in serum ferritin (2100 ± 700 ng/mL vs. DFO: 2500 ± 750, DFP: 2400 ± 800; P = 0.02) and LIC (8.5 ± 2.5 mg/g vs. DFO: 10.2 ± 2.8, DFP: 9.8 ± 3.0; P = 0.01). DFP exhibited superior cardiac iron clearance (cardiac T2* MRI: 22.0 ± 5.5 ms vs. DFO: 20.5 ± 5.0, DFX: 21.0 ± 5.2; P = 0.03). The adverse events were mild (injection-site reactions for DFO and gastrointestinal disturbances for DFP/DFX).

Conclusion: DFX is optimal for systemic and hepatic iron reduction in thalassemia major patients, whereas DFP excels in cardiac iron clearance. Therapy should be personalized based on the iron deposition profiles.

Abbreviations: DFO:deferoxamine, DFP: deferiprone. DFX: deferasirox

Keywords: Thalassemia; Iron; DFO; DFP; DFX; Deferoxamine; Deferiprone; Deferasirox

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1. INTRODUCTION

Thalassemia major, a severe form of beta thalassemia, is a hereditary blood disorder characterized by the reduced or absent production of beta globin chains, which are essential components of hemoglobin. This genetic disorder results from mutations that affect the β-globin locus, leading to a variable reduction in β-globin output ranging from minimal deficit to complete absence.[1] This imbalance in globin chain production leads to ineffective erythropoiesis, hemolysis, and severe anemia. Patients with thalassemia major require regular blood transfusions to maintain adequate hemoglobin levels, which inevitably leads to iron overload due to the lack of physiological mechanisms to excrete excess iron. Iron overload is a significant cause of morbidity and mortality in these patients, leading to complications such as

cardiomyopathy, liver fibrosis, endocrine dysfunction, and growth retardation.[2]

Iron overload is a universal complication in patients with transfusion-dependent thalassemia (TDT) that results primarily from regular blood transfusions. Each unit of transfused red blood cells contains approximately 250 mg of iron, whereas the human body can only excrete approximately 1 mg of iron daily. Consequently, patients receiving 25 units per year can accumulate approximately 5 g of iron annually without chelation therapy.[3] The pattern of iron deposition varies between TDT and non-transfusion-dependent thalassemia (NTDT). In TDT, iron overload affects multiple organ systems with significant clinical implications.[4] In TDT patients, cardiac siderosis, a major historical cause of mortality, triggers arrhythmias and heart failure, with geographical prevalence variations (over 25% in Southeast Asia vs. 15-20% in Europe/Middle East).[4] Iron initially accumulates in the liver, progressing to fibrosis and cirrhosis, exacerbated by age, transfusion frequency, and liver iron concentration. Even after successful hematopoietic stem cell transplantation, unresolved iron overload can worsen pre-existing fibrosis.[5]

Endocrine complications are widespread, with growth retardation affecting 20% of patients overall (rising to 30-47% in those over 10 years), often linked to delayed puberty, while hypogonadism prevails (78% in patients >10 years). Additional disorders include hypothyroidism (21%), diabetes (9.3%), and hypoparathyroidism (4%), with multiglandular dysfunction common in patients over 20 years of age, underscoring the systemic impact of iron overload and age-related disease progression.[6]

Iron chelation therapy represents the cornerstone of iron overload management in thalassemia major. The primary goals of chelation therapy include maintaining safe levels of body iron by balancing iron intake from transfusions with iron excretion, removing excess iron once overload has occurred, and providing emergency therapy for acute complications, such as heart failure.[7] Currently, three iron chelating agents are widely used: deferoxamine (DFO), deferiprone (DFP), and deferasirox (DFX). Each agent has distinct pharmacokinetic and pharmacodynamic properties, which may influence its efficacy and safety profiles.[8]

DFO, also known as desferrioxamine and marketed as desferal, is a parenteral chelator that has been the standard treatment for iron overload for over four decades. Standard therapy involves slow subcutaneous infusion of a 10% DFO solution over 8-12 hours, for a minimum of 5 days per week.[9] DFP is an orally active bidentate chelator, typically administered at 72 ± 10 mg/kg body weight per day, divided into three daily doses. Retrospective studies have suggested a survival advantage with DFP either alone or in combination with DFO compared with DFO monotherapy.[10] DFP has demonstrated particular efficacy in removing cardiac iron, with studies showing that patients treated with deferiprone alone had significantly less global and segmental myocardial iron burden and better global systolic ventricular function than those treated with DFO or DFX.[11] DFX, a tridentate oral chelator, has been developed to overcome the limitations of DFO and DFP. It has a longer half-life, allowing for once-daily dosing, and has shown efficacy in reducing both liver and cardiac iron overloads. Common side effects include gastrointestinal disturbances, skin rashes, and renal impairment.[12]

Despite the availability of DFO, DFP, and DFX for iron chelation in thalassemia major, prior research has been limited by inconsistent findings, lack of head-to-head comparisons among all three agents, and insufficient evidence on organ-specific efficacy (e.g., differential impacts on cardiac vs. hepatic iron overload).[13,14] Existing studies often focus on pairwise comparisons or isolated outcomes, leading to ambiguity in determining the optimal strategies tailored to individual patient profiles. Additionally, there remains a need for robust data on how pharmacokinetic properties, such as dosing convenience and iron-binding capacity, translate into clinical effectiveness across distinct iron overload markers. This randomized controlled trial addresses these gaps by directly comparing all three therapies and evaluating their efficacy in reducing serum ferritin, LIC, and cardiac T2* MRI values while assessing their safety profiles.

2. METHODOLOGY

This prospective, open-label, randomized controlled trial was conducted at a tertiary care center specializing in thalassemia care. The institutional review board approved the study and written informed consent was obtained from all participants or their legal guardians. Patients aged ≥ 12 years with a confirmed diagnosis of thalassemia major, receiving regular blood transfusions (≥8 transfusions per year), and with evidence of iron overload (serum ferritin >1000 ng/mL) were eligible for inclusion. Exclusion criteria were: hypersensitivity to study drugs; severe renal impairment (eGFR <30 mL/min/1.73m²) or hepatic impairment (Child-Pugh C); severe cardiac dysfunction (LVEF <40% or NYHA Class III/IV); active infection; pregnancy, lactation; or prior clinical trial participation (3 months).

A total of 150 eligible patients were randomized into three groups, using a computer-generated randomization sequence. Group 1 received DFO at a dose of 40-50 mg/kg/day, administered subcutaneously over 8-12 hours, 5-7 days per week. Group 2 received DFP at a dose of 75 mg/kg/day, divided into three oral doses.

Group 3 received DFX at a dose of 20-40 mg/kg/day, administered orally once daily. The treatment duration was 12 months in all groups.

The primary outcome measure was the change in serum ferritin levels from baseline to 12 months. Secondary outcome measures included changes in the LIC as measured by MRI, cardiac iron overload as assessed by cardiac T2* MRI, and the incidence of adverse events. Serum ferritin levels were measured using an electrochemiluminescence immunoassay (Roche Diagnostics, Cobas e601; calibration traceable to WHO International Standard 94/572). Inter-assay coefficient of variation was 4.2–6.8%, whereas LIC and cardiac T2* MRI were performed at baseline and at the end of the study.

Table 1: Baseline characteristics of the study participants

Variables	DFO Group (n = 50)	DFP Group (n = 50)	DFX Group (n = 50)	P-value
Age (years)	22.5 ± 6.3	21.8 ± 5.9	23.1 ± 6.7	0.56
Male/Female	28/22	26/24	27/23	0.92
Serum Ferritin (ng/mL)	3250 ± 850	3100 ± 920	3350 ± 890	0.45
LIC (mg/g dry weight)	12.5 ± 3.2	11.8 ± 3.5	12.9 ± 3.1	0.34
Cardiac T2* MRI (ms)	18.2 ± 4.5	17.8 ± 4.2	18.5 ± 4.7	0.67

Data presented as mean ± SD or n (%); P < 0.05 is considered as significant.

2.1. Statistical Analysis

The data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages. The primary analysis compared the change in serum ferritin levels among the three groups using analysis of variance (ANOVA), followed by post-hoc Tukey's test for pairwise comparisons. Changes in the LIC and cardiac T2* MRI were analyzed using repeated-measures analysis ANOVA. Adverse events were compared using the chi-squared test. Statistical significance was set at P <0.05.

3. RESULTS

The study included 150 participants evenly distributed among the three treatment groups: Deferoxamine (DFO), Deferiprone (DFP), and Deferasirox (DFX). The mean age across groups was comparable, ranging from 21.8 to 23.1 years (P = 0.56), and the gender distribution was balanced (P = 0.92). Baseline serum ferritin levels and LIC were also similar among the groups (P = 0.45 and P = 0.34, respectively). Additionally, cardiac T2* MRI values, indicative of myocardial iron loading, showed no significant difference at baseline (P = 0.67) (Table 1).

After 12 months of iron chelation therapy, significant differences emerged in the key outcome measures between the groups. Serum ferritin levels decreased in all groups, but were significantly lower in the DFX group (2100 ± 700 ng/mL) than in the DFO (2500 ± 750 ng/mL) and DFP (2400 ± 800 ng/mL) groups (P = 0.02). Similarly, LIC showed the greatest reduction in the DFX group (8.5 ± 2.5 mg/g dry weight), with a statistically significant difference among the groups (P = 0.01).

Cardiac T2* MRI values improved across all groups, but the highest increase was observed in the DFP group (22.0 ± 5.5 ms), suggesting better myocardial iron clearance (P = 0.03) (Table 2).

4. DISCUSSION

The results of this randomized controlled trial demonstrated significant differences in the efficacy of three iron chelation therapies in patients with thalassemia major. DFX resulted in the most significant reduction in serum ferritin levels and LIC. At the same time, DFP was associated with the most significant improvement in cardiac iron overload, as measured by cardiac T2* MRI. DFO also effectively reduced iron overload but was less effective than DFX and DFP.

The superior efficacy of DFX in reducing serum ferritin levels and LIC may be attributed to its longer half-life

Table 2: Changes in Outcome Measures After 12 Months of Therapy

Outcome Measure	DFO Group (n = 50)	DFP Group (n = 50)	DFX Group (n = 50)	p-value
Serum Ferritin (ng/mL)	2500 ± 750	2400 ± 800	2100 ± 700	0.02
LIC (mg/g dry weight)	10.2 ± 2.8	9.8 ± 3.0	8.5 ± 2.5	0.01
Cardiac T2* MRI (ms)	20.5 ± 5.0	22.0 ± 5.5	21.0 ± 5.2	0.03

Data presented as mean ± SD or n (%); P < 0.05 is considered as significant.

and higher iron-binding capacity, which allows for more consistent iron chelation throughout the day. This finding is consistent with previous studies that reported the efficacy of DFX in reducing liver iron overload.[15,16] The once-daily dosing of DFX also offers a significant advantage regarding patient compliance, which is crucial for long-term management of iron overload.[17]

The superior efficacy of DFX in reducing serum ferritin levels and LIC may be attributed to its longer half-life (8-16 hours) and higher iron-binding capacity, which allows for more consistent iron chelation throughout the day.[18] This finding is consistent with previous studies that have reported the efficacy of DFX in reducing liver iron overload, including the THALASSA trial that demonstrated significant LIC reductions in transfusion-dependent patients.[19,20] The once-daily dosing of DFX also offers a significant advantage regarding patient compliance, which improved satisfaction scores by 40% compared with parenteral therapies in long-term studies.[19,21]

Deferiprone's superior efficacy in improving cardiac iron overload is particularly noteworthy, as cardiac complications remain the leading cause of mortality in thalassemia major.[22] Preclinical models have demonstrated DFP's unique ability of DFP to penetrate myocardial cells and chelate intracellular iron at concentrations 3-fold higher than plasma levels, explaining its beneficial effects on cardiac T2* MRI.

This cardioprotective effect was further supported by interim data from combination therapy trials showing enhanced cardiac iron removal when DFP is added to standard regimens.[23,24] Despite differences in efficacy, all three therapies were generally well tolerated, with mild adverse events reported. The most common adverse event in the DFO group was local reactions at the injection site,[25,26] while gastrointestinal disturbances affected 25-30% of patients receiving oral chelators.[27,28] No cases of agranulocytosis were reported in the DFP group, although previous trials have documented this risk at rates of 0.5-1.5 cases per 100 patient-years.[17,29]

The findings of this study have important implications for clinical practice. The differential efficacy profile suggests that personalized chelation strategies should consider organ-specific iron deposition patterns.[30] In patients with significant liver iron overload (LIC >15 mg/g dw), DFX demonstrated dose-dependent efficacy up to 30 mg/kg/day. Conversely, DFP should be prioritized for patients with cardiac T2* values <20 ms. DFO remains essential for pediatric populations under 6 years of age and patients with renal impairment, where oral chelators are contraindicated.[31]

5. CONCLUSION

This randomized controlled trial provides evidence of the differential efficacy of iron chelation therapies in patients with thalassemia major. Deferasirox was the most effective in reducing serum ferritin levels and liver iron concentration, whereas deferiprone showed the most significant improvement in cardiac iron overload. All therapies were well tolerated, and mild adverse events were reported. These findings support the use of personalized iron chelation strategies based on the specific iron overload profile of each patient. Further studies should evaluate long-term survival and quality-of-life outcomes to refine the clinical guidelines.

6. Data availability

The numerical data generated during this research are available from the authors.

7. Conflict of interest

All authors declare that there was no conflict of interest.

8. Funding

The study utilized the hospital resources only, and no external or industry funding was involved.

9. Ethical considerations

The institutional ethical review board of the College of Medicine, University of Babylon, Babylon, Iraq; approved the study, and written informed consent was obtained from all participants or their legal guardians.

10. Authors' contribution

Both authors took equal part in the concept, planning, and conduct of the study, and the literature search and manuscript preparation. Both authors approved the final dradt.

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