ISSN: 2457-0400

Impact Factor: 7.675

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

www.wjahr.com

Volume: 10, Issue: 3

Page N. 111-118

Year: 2026

Original Article

Coden USA: WJAMA3

# ROLE OF DEFERIPRONE IN CHELATION THERAPY FOR TRANSFUSIONAL IRON OVERLOAD IN THALASSEMIA AND OTHER ANEMIAS

**[*1]Dr. Ansam Sami Mustafa Zakariya, [2]Dr. Ghadah Abdulwahab Ahmed, [3]Dr. Esraa M. Al-Sardar**

[1]M.B.Ch.B./F.A.B.H.S.
[2]M.B.Ch.B./F.A.B.H.S.
[3]M.B.Ch.B./F.A.B.H.S.

**Article Received:** 02 February 2026 **Article Revised:** 23 February 2026 **Article Published:** 01 March 2026

***Corresponding Author: Dr. Ansam Sami Mustafa Zakariya**

M.B.Ch.B./F.A.B.H.S.

**DOI:** https://doi.org/10.5281/zenodo.18815367

**How to cite this Article:** [*1]Dr. Ansam Sami Mustafa Zakariya, [2]Dr. Ghadah Abdulwahab Ahmed, [3]Dr. Esraa M. Al-Sardar, (2026). Role Of Deferiprone In Chelation Therapy For Transfusional Iron Overload In Thalassemia And Other Anemias. World Journal of Advance Healthcare Research, 10(3), 111–118.



## ABSTRACT

**Background:** Thalassemia and other chronic anemias that necessitate regular blood transfusions are seriously complicated by iron overload. Progressive iron accumulation causes toxic deposits in vital organs, including the liver, heart, and endocrine glands, leading to substantial morbidity and mortality if not treated properly. Deferiprone has been used to eliminate iron in patients with a high iron load or cardiac siderosis. Despite its established usefulness, deferiprone linked to side effects necessitating regular monitoring. **Objectives:** Is to assess the role of deferiprone in chelation therapy among patients with blood transfusion related iron overload resulting from thalassemia and other chronic anemias. The study specifically evaluates its efficacy in lowering blood ferritin levels as well as its safety profile throughout therapy, giving more proof for its clinical value in real-world practice. **Methods:** This is a hospital-based prospective observational study. It was conducted between the 1st of September of year 2024 to the 31st of December of year 2025 at Al Hadbaa speciality hospital for haematology and bone marrow transplantation in Mosul city, Republic of Iraq. The study included patients with thalassemia or other chronic anemias who received frequent blood transfusions and were given deferiprone as part of their iron chelation treatment. Additionally, it included patients should have serum ferritin level of 2500 ng/mL or higher indicating iron overload; as well as, the included patients' age should be between 8 to 65 years, have been on deferiprone on the dose of (75 mg/kg/day) for at least six months and have available baseline and follow-up laboratory data. On the other hand, the study excluded patients with acute infections or inflammatory diseases that alter ferritin levels, patient with specific bone marrow condition such as myelodysplastic syndrome, myelofibrosis, leukemia. Furthermore, the study excluded patients with significant hepatic or renal impairment, patients with known hypersensitivity to deferiprone and those with incomplete clinical or laboratory records. **Results:** The study includes 88 patients; of them 40 patients were male and 48 patients were females. Male: Female ratio was 1:1.2. The mean age ± standard deviation of the study patients was 20.62 ± 7.11 years. The study found the mean of serum ferritin level was significantly decrease from (7306.86 ± 3459.82) before deferiprone to (6066.64 ± 3062.68) after deferiprone at the first checking and to (4978.18 ± 2407.63) at the second checking. Blood urea was statistically significant difference (lowered) after treatment (P value = 0.048), while no statistically significant difference found after treatment regarding serum creatinine, ALT and AST (P value >0.05) for all of them. As well as, after deferiprone the number of patients with each of pulmonary hypertension, left ventricular hypertrophy (LVH), mitral regurgitation (MR) and systolic dysfunction were significantly decrease (P value <0.001). Moreover, the mean of ejection fraction was also significant elevated (P value <0.001) after deferiprone treatment. Chromaturia was reported by 48 (54.5%) patients, followed by appetite changes was reported among 34 (38.6%) patients and dyspepsia in 30 (34.2%) patients. Lastly, 41 (46.5%) patients of the study participants reported frequent discontinuity and 14 (15.9%) patients reported definitive stop of deferiprone.

**Conclusion:** Deferiprone is an effective oral iron chelator for patients with transfusion-related iron overload from thalassemia and other chronic anemias. It effectively lowers serum ferritin levels, improves heart function, and has an acceptable safety profile when administered with proper monitoring. To avoid major side effects, neutrophil counts, renal and liver function tests should be monitored on a regular basis.

**KEYWORDS:** Chelating, Deferiprone, Excess, Iraq, Iron, Mosul.

## 1. INTRODUCTION

Thalassemia and other chronic anemias that necessitate regular blood transfusions are seriously complicated by iron overload.[1] Each transfused packed red blood cell contains between 200 and 250 mg of elemental iron, which exceeds the body's physiological capability for iron excretion.[2] Progressive iron accumulation causes toxic deposits in vital organs, including the liver, heart, and endocrine glands, leading to substantial morbidity and mortality if not treated properly.[3]

Iron overload is a leading cause of heart failure, hepatic fibrosis, cirrhosis, diabetes mellitus, hypogonadism, and growth retardation in transfusion-dependent patients.[4] Effective iron chelation therapy is crucial for managing patients with β-thalassemia major, thalassemia intermedia, sickle cell disease, aplastic anemia, and other transfusion-dependent anemias over time.[5]

Deferoxamine was the first widely used chelating drug for iron overload, but its parenteral route of administration, long infusion times, and poor patient compliance limited its long-term efficacy. The introduction of oral iron chelators, such as deferiprone and deferasirox, has greatly improved therapy adherence and outcomes.[6] Deferiprone is an oral iron chelator that forms stable complexes with ferric iron and facilitates excretion mostly through the urine.[7]

Several studies have shown that deferoxamine is particularly effective at lowering myocardial iron burden and improving cardiac outcomes in patients with transfusion-induced iron excess.[8-10] Additionally, deferiprone can remove intracellular iron from the heart more efficiently than deferoxamine.[11] Furthermore, studies show that long-term deferiprone therapy is more effective than subcutaneous deferoxamine in protecting the heart against the toxicity of iron overload.[12-13]

Deferiprone has been used as a monotherapy or in conjunction with deferoxamine to improve iron elimination in patients with a high iron load or cardiac siderosis.[14] Despite its established usefulness, deferiprone use has been linked to side effects such as gastrointestinal discomfort, arthralgia, myopathy and fatigue necessitating monthly monitoring, in addition to neutropenia, and agranulocytosis, necessitating weekly hematological monitoring. The balance between therapeutic benefit and possible adverse effects is still a significant concern in clinical practice.[2,15]

The aim of this study is to assess the role of deferiprone in chelation therapy among patients with blood transfusion related iron overload resulting from thalassemia and other chronic anemias. The study specifically evaluates its efficacy in lowering blood ferritin levels as well as its safety profile throughout therapy, giving more proof for its clinical value in real-world practice.

## 2. PATIENTS AND METHODS

This is a hospital-based prospective observational study. It was conducted between the 1st of September of year 2024 to the 31st of December of year 2025 at Al Hadbaa speciality hospital for haematology and bone marrow transplantation in Mosul city, Republic of Iraq. Ethical approval was obtained from the Directorate of Health at Mosul and the administration of Hadbaa Hospital (number 2025081 at the 7th of August 2024). Written informed consent was obtained from the parents. Patient confidentiality was strictly maintained.

The study included patients with thalassemia or other chronic anemias who received frequent blood transfusions and were given deferiprone as part of their iron chelation treatment. Additionally, it included patients should have serum ferritin level of 2500 ng/mL or higher indicating iron overload; as well as, the included patients' age should be between 8 to 65 years, have been on deferiprone for at least six months and have available baseline and follow-up laboratory data. On the other hand, the study excluded patients with acute infections or inflammatory diseases that alter ferritin levels, patient with specific bone marrow condition such as myelodysplastic syndrome, myelofibrosis, leukemia. Furthermore, the study excluded patients with significant hepatic or renal impairment, patients with known hypersensitivity to deferiprone and those with incomplete clinical or laboratory records.

Deferiprone was given orally in doses of (75 mg/kg/day). Doses were adjusted based on serum ferritin levels and patient tolerance. Data were acquired from medical records and patient files, including the demographic data (age, gender), underlying hematological diagnosis, the frequency of transfusions. In addition to baseline and follow-up serum ferritin levels, liver function tests (AST and ALT), blood urea, serum creatinine, complete blood count (with special emphasis on neutrophil count), echocardiographic findings. Patients' adverse events were reported (gastrointestinal symptoms, arthralgia, headache, neutropenia, agranulocytosis, myopathy, fatigue, hypotension, chromaturia and dermatological complications). Moreover, history of deferiprone discontinuation and causes of this discontinuation were reported.

Regarding biochemical analysis, each patient gave 8 cc of blood (2 cc for each of serum ferritin, blood urea, serum creatinine and complete blood count). The blood was kept for a half hour to coagulated then by centrifuge the precipitate was isolated and analyzed.

The primary outcome measures were changes in serum ferritin levels before and after deferiprone treatment. While, the secondary outcome measures were change in cardiac, liver and renal parameters, the incidence of unfavorable pharmacological responses and treatment discontinuation.

All patients did regular hematological monitoring, including weekly or biweekly complete blood counts to detect neutropenia or agranulocytosis. Deferiprone was stopped if the absolute neutrophil count fell below 1.5 × $10^9$/L or significant adverse events occurred.

The information gathered was processed. Statistical analysis was conducted using SPSS version 30.0 (SPSS Inc., Chicago, USA). Continuous variables were reported as mean ± SD or median (interquartile range) based on distribution. The serum ferritin levels were compared before and after therapy using a paired t-test or the Wilcoxon signed-rank test. Categorical variables were represented as frequencies and percentages. A p-value of <0.05 was considered statistically significant.

## 3. RESULTS

The study includes 88 patients; of them 40 patients were male and 48 patients were females. Male: Female ratio was 1:1.2. The mean age ± standard deviation of the study patients was 20.62 ± 7.11 years. As shown in figure 1.

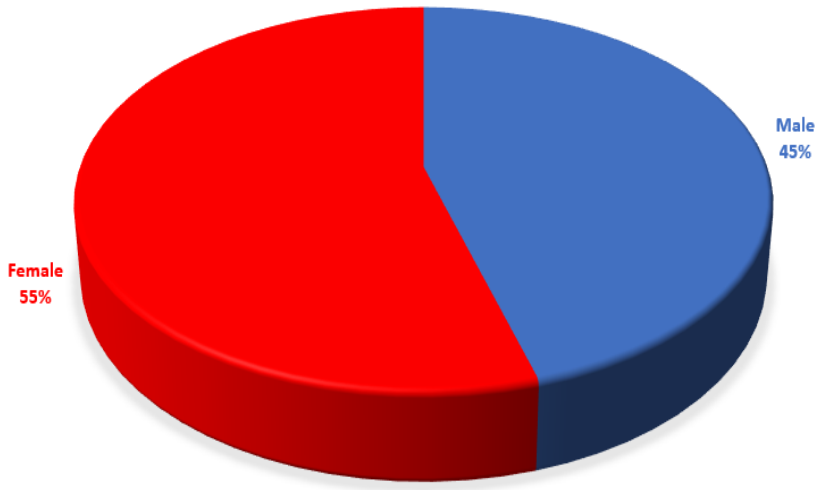


**Figure 1: Distribution of the study patients according to their gender.**

The majority of patients already were on deferoxamine (desferal) and deferasirox (exjod) in (39.8%) followed by desferal alone in (38.6%), exjod alone in (20.5%), while only (1.1%) was not received any drug. As shown in figure 2.

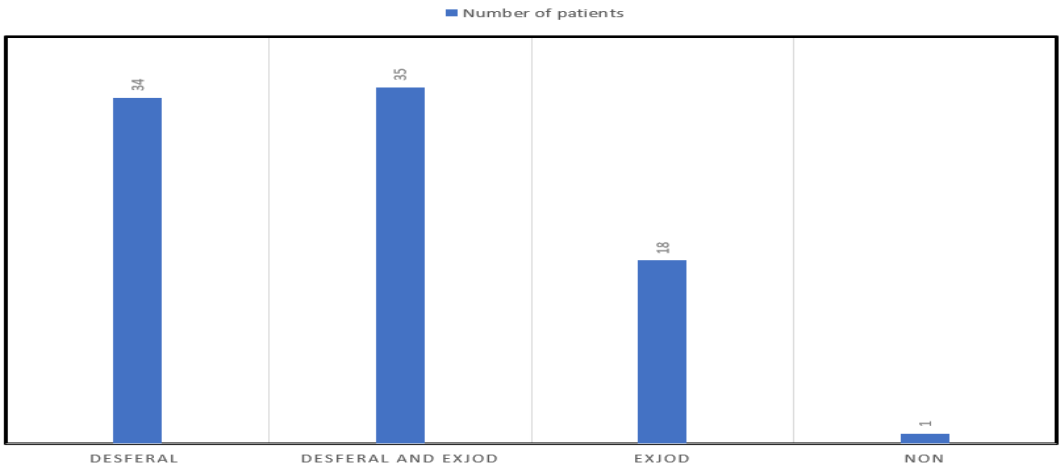


**Figure 2: Distribution of the study patients according to their previous received drugs.**

Figure 3 shows comparison between means of serum ferritin levels before and after deferiprone at the first and the second checking. It's evident that the mean of serum ferritin level was significantly decrease from (7306.86 ± 3459.82) before deferiprone to (6066.64 ± 3062.68) after deferiprone at the first checking and to (4978.18 ± 2407.63) at the second checking.

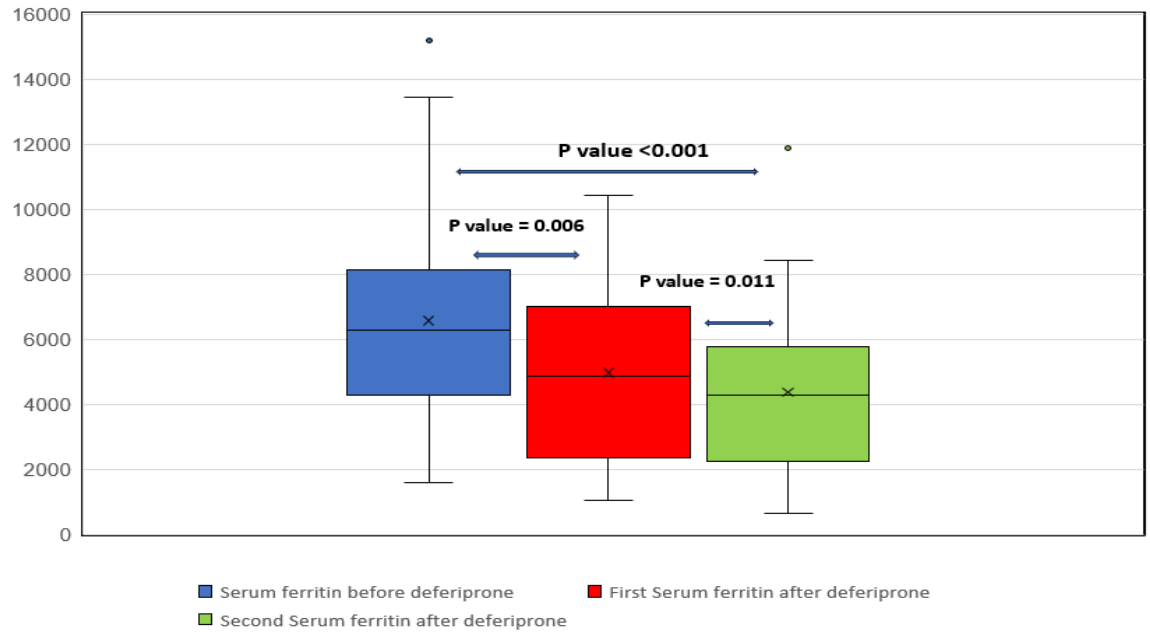


**Figure 3: Comparison between means of serum ferritin levels before and after deferiprone at the first and the second checking.**

Table 1 shows comparison between the patients' blood urea, serum creatinine, ALT and AST before and after deferiprone treatment. Blood urea was statistically significant difference (lowered) after treatment (P value = 0.048). While no statistically significant difference found after treatment regarding serum creatinine, ALT and AST (P value >0.05) for all of them.

**Table 1: Comparison between the patients' blood urea, serum creatinine, ALT and AST before and after deferiprone treatment (Number = 88).**

| Variable | Before deferiprone | After deferiprone | P value |
|---|---|---|---|
| Blood urea, mean ± standard deviation | 5.14 ± 1.37 | 4.11 ± 1.24 | **0.048** |
| Serum creatinine, mean ± standard deviation | 41.15 ± 14.49 | 37.31 ± 12.93 | 0.071 |
| Alanine aminotransferase, mean ± standard deviation | 51.89 ± 28.44 | 54.36 ± 40.45 | 0.643 |
| Aspartate aminotransferase, mean ± standard deviation | 52.62 ± 28.62 | 56.21± 20.50 | 0.620 |

Table 2 shows comparison between the patients' Echocardiography before and after deferiprone treatment. Among 15 patients with echocardiographic findings before deferiprone use. Statistically significant difference found (P value <0.001) after given deferiprone regarding the presence of pulmonary hypertension, left ventricular hypertrophy (LVH), mitral regurgitation (MR) and systolic dysfunction. Moreover, the mean of ejection fraction was also significant elevated (P value <0.001) after deferiprone treatment.

**Table 2: Comparison between the patients' Echocardiography before and after deferiprone treatment.**

| Variable | Before deferiprone | After deferiprone | P value |
|---|---|---|---|
| Pulmonary hypertension, number (%): | 7 (46.66%) | 3 (20%) | **<0.001** |
| Left ventricular hypertrophy, number (%): | 8 (53.34%) | 2 (13.33%) | **<0.001** |
| Mitral regurgitation, number (%): | 6 (40%) | 1 (6.66%) | **<0.001** |
| Systolic dysfunction, number (%): | 7 (46.66%) | 3 (20%) | **<0.001** |
| Ejection fraction, mean ± standard deviation | 58.61 ± 5.39 | 62.37 ± 5.43 | **<0.001** |

Figure 4 shows reported adverse effects of deferiprone. Chromaturia was reported by 48 (54.5%) patients, followed by appetite changes was reported among 34 (38.6%) patients and dyspepsia in 30 (34.2%) patients.

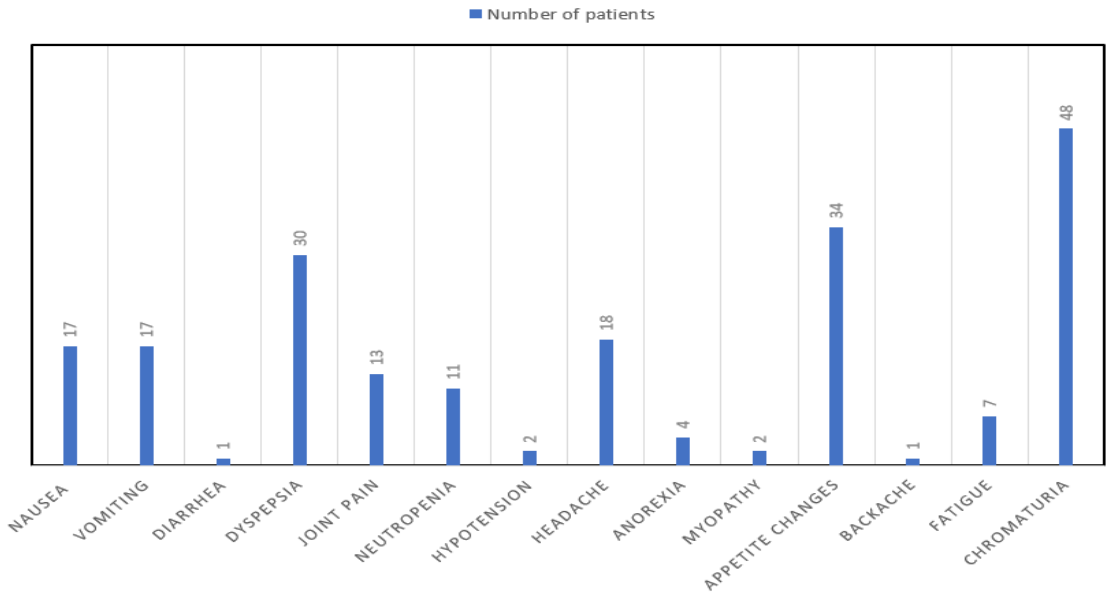


**Figure 4: Reported adverse effects of deferiprone.**

Table 3 shows numbers and percentages of patients who had period of discontinuity and those with definitive stop. It's evident that 41 (46.5%) patients of the study participants reported frequent discontinuity and 14 (15.9%) patients reported definitive stop of deferiprone.

**Table 3: Reported period of discontinuity and definitive stop of deferiprone (Number = 88).**

| Variable | Number | Percentage |
|---|---|---|
| Period of discontinuity: | | |
| -Yes | 41 | 46.5% |
| -No | 47 | 53.5% |
| Definitive stop: | | |
| -Yes | 14 | 15.9% |
| -No | 74 | 84.1% |

Figure 5 shows cause of temporal deferiprone discontinuity. Absolute neutrophil decrease was the commonest cause among 11 patients, followed by gastro-intestinal disorders in 10 patients and elevated liver enzyme in 9.

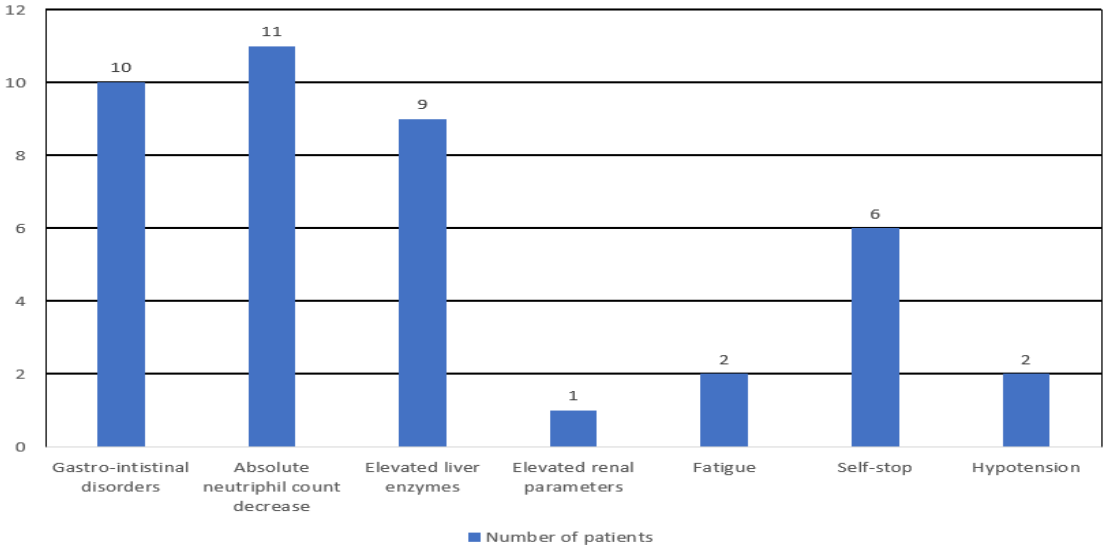


**Figure 5: Causes of temporal deferiprone discontinuity.**

Figure 6 shows cause of permanent deferiprone stop. Absolute neutrophil count decrease was the commonest cause of permanent deferiprone stop in 4 patients followed by gastro-intestinal disorders in 3 patients.

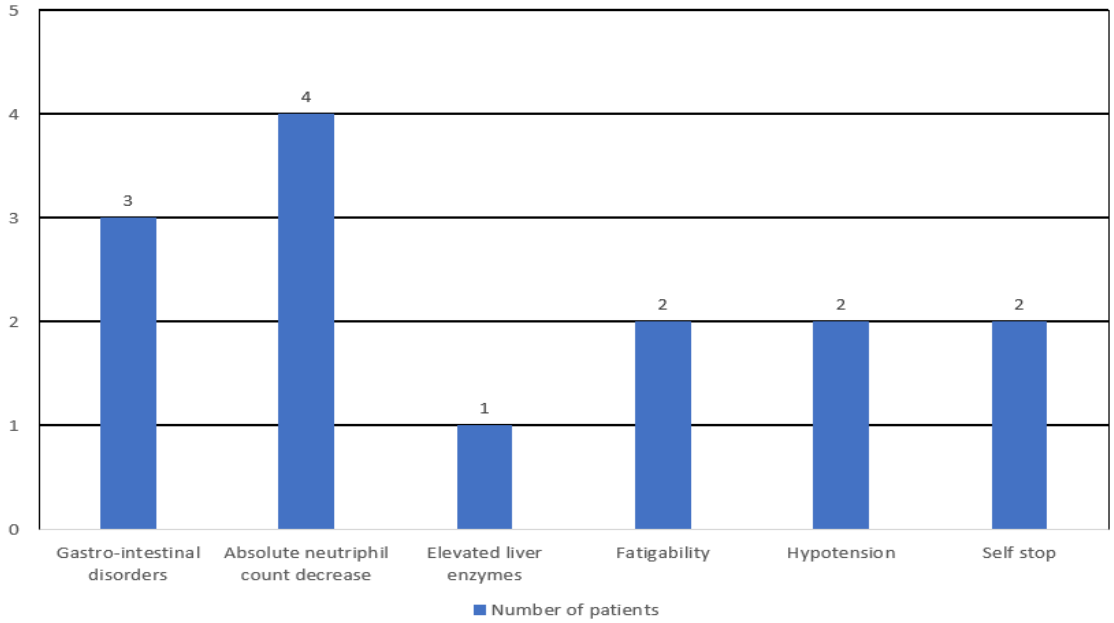


**Figure 6: Causes of permanent deferiprone discontinuity.**

## 4. DISCUSSION

The current study's most important finding is a notable decreased in blood ferritin levels after deferiprone therapy. The mean serum ferritin levels declined gradually from baseline to the first follow-up, then again to the second follow-up. These findings are consistent with previous studies that has shown deferiprone is effective in enhance iron excretion, notably via urinary routes.[16-17] The studies suggest that deferiprone is highly effective in managing iron overload, and received patients might reached target serum ferritin levels over time. Moreover, the magnitude of ferritin decrease reported in the present study could partly be attributed to the study population's high baseline iron burden, with the majority having previously administered deferoxamine and/or deferasirox with inadequate treatment of iron overload. This meant that deferiprone can be useful even in patients with high iron levels and prior chelation exposure.

In the current study, blood urea levels fell significantly after deferiprone therapy, although serum creatinine levels remained stable. This suggests that deferiprone had little or no impact on renal function when properly monitored. In line with Rajapurkar et al[18] who found despite small changes in blood urea levels, renal function remained steady throughout treatment. Fradette et al[19] also found that deferiprone was well tolerated with no significant renal safety problems.

In the context of hepatic function, there were no significant differences between ALT and AST values before and after treatment. This shows that deferiprone did not cause hepatotoxicity in the study population. Given that iron excess contributes to hepatic harm, the absence of considerable liver enzyme rise lends confidence to deferiprone's hepatic safety when administered under close clinical supervision. Taher et al[20] found in his long-term observational study that liver enzymes don't affect by deferiprone. Won et al[21] found that AST and ALT showed some fluctuation after deferiprone therapy, but they not reaching the level of significancy. These findings are clinically significant because concerns regarding hepatic and renal toxicity may limit the use of oral chelators in the clinical practice.

One of the most interesting findings from this study is a considerable improvement in echocardiographic parameters following deferiprone therapy. The number of individuals with pulmonary hypertension, left ventricular hypertrophy, mitral regurgitation, and systolic dysfunction dropped significantly after treatment. Furthermore, the mean of ejection fraction increased significantly from 58.61% to 62.37%. These findings provide significant support for deferiprone's cardioprotective properties as iron-induced cardiomyopathy and heart failure, which are the primary cause of death among transfusion-dependent patients. Because of its small molecular size and intracellular penetration, deferiprone has been shown to have a higher potential for iron chelation in the heart than other iron chelating drugs. This enables it to remove iron directly from cardiomyocytes and enhancing cardiac function. Filosa et al[22] and Smith et al[23] reported that deferiprone can reverse cardiac siderosis and enhance survival. This emphasizes the value of deferiprone as a preferred chelator in individuals with cardiac iron overload or early cardiac dysfunction.

Chromaturia was the most commonly reported adverse effect in this study, occurring in more than half of the patients which runs with Piga et al[12] study findings (60%). This is a well-known and harmless side effect of urine excretion of iron-deferiprone complexes, represents just the expected removal of iron-chelate complexes in the urine rather than actual medication toxicity and it is typically not necessary to discontinue treatment. Furthermore, other frequent reported adverse effects included appetite changes and dyspepsia, which corresponded to the clinical data from large deferiprone trials and safety studies consistently show that gastrointestinal symptoms such as nausea, abdominal pain/discomfort, dyspepsia, and appetite changes (both increases and decreases) are among the most common non-serious adverse reactions experienced by deferiprone patients, typically occurring early in therapy and resolving without the need to discontinue treatment.[24]

Additionally, although major side effects such as agranulocytosis are well reported with deferiprone, this study discovered that a decrease in neutrophil count was the most common reason for treatment cessation. As well as, the study found approximately half of the patients in the present study had periods of treatment discontinuity, with about 16% discontinuing deferiprone permanently. Neutropenia, gastrointestinal discomfort, and elevated liver enzyme were the most common causes of permanent cause of deferiprone stop, which is parallel to Galanello et al[25] study findings, highlighting the importance of patient education, continuous follow-up, and personalized treatment options in order to increase adherence.

This study has some limitations. First, it is an observational study with no control group, which limits the capacity to demonstrate causality. Second, serum ferritin was utilized as the primary indicator of iron excess, despite exclusion criteria, which could be altered by inflammation or infection. Third, the follow-up period may be insufficient to assess long-term outcomes like as survival or endocrine problems. Finally, cardiac iron load was not directly evaluated by cardiac MRI, which is considered the gold standard for assessing myocardial iron. Despite these limitations, the study provides essential real-world data on deferiprone use in a limited resource setting, demonstrating its effectiveness and safety.

## 5. CONCLUSIONS AND RECOMMENDATIONS

The current study shows that deferiprone is an effective oral iron chelator for patients with transfusion-related iron overload from thalassemia and other chronic anemias. It effectively lowers serum ferritin levels, improves heart function, and has an acceptable safety profile when administered with proper monitoring. Deferiprone is a viable therapeutic alternative, especially for patients with cardiac involvement or poor response to conventional chelators. Deferiprone is suggested as a first-line or adjuvant chelation therapy for patients with transfusion-dependent anemias with a high iron load, particularly those with cardiac problems. To avoid major side effects, neutrophil counts, renal and liver function tests should be monitored on a regular basis. More large-scale, randomized controlled studies with extended follow-up and cardiac MRI monitoring are required to corroborate these findings and develop appropriate chelation protocols.

**Conflict of intertest**

The authors of this study report no conflicts of interest.

## REFERENCES

1. Mahmoud HQ, Mhana RS, Mohammed AA. Therapeutic options and management approach on thalassemia an overview. International Journal of Medical Science and Dental Health, 2024; 10(01): 17-28.
2. Kontoghiorghes GJ. Iron load toxicity in medicine: from molecular and cellular aspects to clinical implications. International journal of molecular sciences, 2023 Aug 18; 24(16): 12928.
3. Eke S. Unveiling the Hidden Dangers of Iron Overload–A Comprehensive Literature Review. International Journal of High School Research, 2025 May 1; 7(5).
4. Hsu CC, Senussi NH, Fertrin KY, Kowdley KV. Iron overload disorders. Hepatology communications, 2022 Aug; 6(8): 1842-54.
5. Fasano RM, Meier ER, Chonat S. Sickle cell disease, thalassemia, and hereditary hemolytic anemias. Rossi's Principles of Transfusion Medicine, 2022 Aug 23: 326-45.
6. Entezari S, Haghi SM, Norouzkhani N, Sahebnazar B, Vosoughian F, Akbarzadeh D, Islampanah M, Naghsh N, Abbasalizadeh M, Deravi N. Iron Chelators in Treatment of Iron Overload. J Toxicol, 2022 May 5; 2022: 4911205.
7. Dar SA, Altaf S, Dar AM. Chelation Therapy: Types of Chelating Agents, Limitations and Utility.
8. Mirghani HO, Alanazi NK, ALqarni MB, Alshehri AS, Alshareif AA, Alanazi TB, Alanazi GB, Asseiri RA, Al Makir MK, Alhwiati AS, Alzahrani TO. Updates on Management of transfusion induced Iron Overload: A Systematic Review. Saudi Medical Horizons Journal, 2023 Oct 29; 3(3): 149-57.
9. Salem A, Desai P, Elgebaly A. Efficacy and safety of combined deferiprone and deferasirox in iron-overloaded patients: a systematic review. Cureus, 2023 Nov 4; 15(11).
10. Yadav PK, Singh AK. A review of iron overload in beta-thalassemia major, and a discussion on alternative potent iron chelation targets. Plasmatology, 2022 May; 16: 26348535221103560.
11. Pepe A, Meloni A, Capra M, Cianciulli P, Prossomariti L, Malaventura C, Putti MC, Lippi A, Romeo MA, Bisconte MG, Filosa A. Deferasirox, deferiprone and desferrioxamine treatment in thalassemia major patients: cardiac iron and function comparison determined by quantitative magnetic

resonance imaging. Haematologica, 2010 Sep 30; 96(1): 41.
12. Piga A, Gaglioti C, Fogliacco E, Tricta F. Comparative effects of deferiprone and deferoxamine on survival and cardiac disease in patients with thalassemia major: a retrospective analysis. Haematologica, 2003 Jan 1; 88(5): 489-96.
13. Wood JC, Otto-Duessel M, Gonzalez I, Aguilar MI, Shimada H, Nick H, Nelson M, Moats R. Deferasirox and deferiprone remove cardiac iron in the iron-overloaded gerbil. Translational research, 2006 Nov 1; 148(5): 272-80.
14. Shah R, Shah A, Badawy SM. An evaluation of deferiprone as twice-a-day tablets or in combination therapy for the treatment of transfusional iron overload in thalassemia syndromes. Expert review of hematology, 2023 Feb 1; 16(2): 81-94.
15. Premawardhena A, Wanasinghe S, Perera C, Wijethilaka MN, Rajakaruna RH, Samarasinghe RA, Williams S, Mettananda S. Deferoxamine, deferasirox, and deferiprone triple iron chelator combination therapy for transfusion-dependent β-thalassaemia with very high iron overload: a randomised clinical trial. The Lancet Regional Health-Southeast Asia, 2024 Nov 1; 30.
16. Morales NP, Rodrat S, Piromkraipak P, Yamanont P, Paiboonsukwong K, Fucharoen S. Iron chelation therapy with deferiprone improves oxidative status and red blood cell quality and reduces redox-active iron in β-thalassemia/hemoglobin E patients. Biomedicine & Pharmacotherapy, 2022 Jan 1; 145: 112381.
17. Taher A, Sheikh-Taha M, Koussa S, Inati A, Neeman R, Mourad F. Comparison between deferoxamine and deferiprone (L1) in iron-loaded thalassemia patients. European Journal of Haematology, 2001 Jul; 67(1): 30-4.
18. Rajapurkar MM, Hegde U, Bhattacharya A, Alam MG, Shah SV. Effect of deferiprone, an oral iron chelator, in diabetic and non-diabetic glomerular disease. Toxicol Mech Methods, 2013 Jan; 23(1): 5-10.
19. Fradette C, Pichette V, Sicard É, Stilman A, Jayashankar S, Tsang YC, Spino M, Tricta F. Effects of renal impairment on the pharmacokinetics of orally administered deferiprone. Br J Clin Pharmacol, 2016 Oct; 82(4): 994-1001.
20. Taher A, Aoun E, Sharara AI, Mourad F, Gharzuddine W, Koussa S, Inati A, Dhillon AP, Hoffbrand AV. Five-year trial of deferiprone chelation therapy in thalassaemia major patients. Acta Haematol, 2004; 112(4): 179-83.
21. Won SC, Han DK, Seo JJ, Chung NG, Park SK, Park KB, Kook H, Lyu CJ. Efficacy and safety of deferiprone (Ferriprox), an oral iron-chelating agent, in pediatric patients. Korean J Hematol, 2010 Mar; 45(1): 58-61.
22. Filosa A, Vitrano A, Rigano P, Calvaruso G, Barone R, Capra M, Cuccia L, Gagliardotto F, Pitrolo L, Prossomariti L, Casale M, Caruso V, Gerardi C, Campisi S, Cianciulli P, Rizzo M, D'Ascola G, Ciancio A, Maggio A. Long-term treatment with deferiprone enhances left ventricular ejection function when compared to deferoxamine in patients with thalassemia major. Blood Cells Mol Dis, 2013 Aug; 51(2): 85-8.
23. Smith GC, Alpendurada F, Carpenter JP, Alam MH, Berdoukas V, Karagiorga M, Ladis V, Piga A, Aessopos A, Gotsis ED, Tanner MA, Westwood MA, Galanello R, Roughton M, Pennell DJ. Effect of deferiprone or deferoxamine on right ventricular function in thalassemia major patients with myocardial iron overload. J Cardiovasc Magn Reson, 2011 Jul 6; 13(1): 34.
24. Chiesi Farmaceutici S.p.A. Proposal for the Inclusion of Deferiprone in the WHO Model List of Essential Medicines for the Treatment of Transfusional Iron Overload in Adult and Paediatric Patients with Thalassaemia Syndromes, Sickle Cell Disease or Other Anaemias. December 2022.
25. Galanello R. Deferiprone in the treatment of transfusion-dependent thalassemia: a review and perspective. Ther Clin Risk Manag, 2007 Oct; 3(5): 795-805.