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Original Article

# Effect of Deferoxamine and Deferasirox on Blood Parameters in Children with Transfusion-Dependent Thalassaemia with Iron-Overload

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***ABSTRACT***

*Iron-chelation therapy is widely regarded as the primary option for reducing iron accumulation. Excessive accumulation of iron may significantly contribute to the morbidity and mortality with transfusion-dependent thalassaemia major. Two widely recognised iron chelators are deferoxamine and deferasirox, despite the presence of certain adverse effects. However, there is a lack of data regarding the impact of these medications on blood parameters. An observational study was carried out at the department of pharmacology and therapeutics, Sylhet MAG Osmani Medical College, Sylhet. The age of the enrolled children were 2-15 years with transfusion-dependent thalassaemia and serum ferritin levels exceeding 1500 ng/mL. The study included a total of 25 patients who received injectable deferoxamine (DFO) therapy and 26 patients who received oral deferasirox (DFX) treatment for a period of 6 months. Serum ferritin, serum creatinine, serum ALT and CBC were assessed at the beginning of the study and again after 6 months of treatment. Serum ferritin level was decreased significantly in both deferoxamine and deferasirox group ($p<0.001$). Haematological (haemoglobin, neutrophil and platelet count) and biochemical (serum creatinine, serum ALT) parameters did not change significantly ($p<0.05$). Deferoxamine and deferasirox reduced serum ferritin levels significantly but did not have any significant effect on blood parameters in children with iron overload who had transfusion-dependent thalassaemia.*

***Keywords:*** *Deferoxamine, Deferasirox, Blood transfusion, Blood parameters, Iron chelator, Thalassaemia.*

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## INTRODUCTION

Thalassaemia is a hereditary haemoglobinopathies distinguished by diminished production of one or more globin chains[1]. Blood transfusion is commonly required in individuals diagnosed with β-thalassaemia major[2]. Iron overload is an inevitable condition experienced by individuals with thalassaemia major due to the frequency of blood transfusions[3]. Excess iron cannot be eliminated by the human body. Patients with β-thalassaemia who receive repeated trasfusions will build up harmful and perhaps fatal quantities of iron in their bodies[2].

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To eliminate heavy metals and minerals from the body, chelation is used. Chelation therapy increases urinary and faecal iron excretion to prevent iron accumulation[4]. Because iron is needed for normal physiological functions, chelation therapy must balance its benefits with its side effects. A careful dose adjustment and treatment plan are needed to avoid excess chelation when iron levels fall[5]. Without extensive iron chelation therapy, thalassaemia major patients die in their early life from the complications of iron deposition[6]. Organ dysfunction causes various chronic difficulties in these patients over time[7]. Blood transfusions 10-20 times is likely to raise serum ferritin levels above 1000 ng/L[8]. For this reason, iron chelators should be administered promptly as the serum ferritin level rises above 1000 ng/L[9].

One of the most well-known iron chalators over the past few decades is deferoxamine. There are some disadvantages associated with deferoxamine administration. These include prolonged parenteral infusions over 8-12 hours for 5-7 days in a week, local infection, intolerance of a particular chelator, psychosocial issues, psychological wellbeing, lack of institutional support, unavailability, side effects of variable severity, patient's refusal or non-compliance, and sudden increase of serum creatinine level[10,11,12].

There is still a requirement for an efficient and well-tolerated iron chelator that has a less demanding treatment regimen. This could potentially aid in maintaining long-term adherence in patients who receive regular blood transfusions, regardless of their age. Deferasirox belongs to a novel class of oral tridentate chelators. Serum ferritin levels were assessed monthly to monitor the effectiveness of deferasirox[2,13]. Deferasirox has been shown to be well tolerated in most of the patients, with the most commonly observed adverse effects including gastrointestinal disturbance, and skin rash. On blood parameters, deferasiroxis evident in a mild non-progressive increase in creatinine levels and raised liver enzymes[14,15].

Limited data has been available regarding the impact of deferoxamine and deferasirox on blood parameters. Consequently, the purpose of this study was to observe the changes in blood parameters of these iron-chelating agents in patients with transfusion-dependent thalassaemia.

## MATERIALS AND METHODS

This observational study was conducted in the department of pharmacology and therapeutics, Sylhet MAG Osmani Medical College, Sylhet, in collaboration with the Department of Paediatrics, Sylhet MAG Osmani Medical College Hospital, Sylhet. Prior to the commencement of the study, approval of the ethical committee of Sylhet MAG Osmani Medical College, Sylhet, was taken. Convenient sampling techniques were adopted for data collection. The study included 56 transfusion-dependent beta thalassaemia patients, aged 2 to 15 years, who were admitted to the paediatric department of the Sylhet MAG Osmani Medical College Hospital in Sylhet between January 2020 and December 2020 and had a serum ferritin level above 1500 ng/ml. Exclusion criteria were serum creatinine >0.7 mg/dl, serum ALT >120 u/l, clinically relevant auditory and/or ocular toxicity related to iron chelation therapy, congenital heart diseases, neutrophil count <30% and platelet count <100,000/cmm[15].

Following a thorough explanation of the study procedure and the aim of the study, informed written consent was obtained from patients' legal guardians. Every case started with a thorough history that included the patient's age, gender, the number, duration and type of blood transfusions as well as the type, duration, and compliance with the treatment plan for chelation therapy. Comprehensive clinical assessments were carried out. Study variables were haemoglobin level, neutrophil count, platelet count, serum creatinine, serum ferritin, and alanine aminotransferase (ALT).

The study population was divided into two groups, Group-A (n=29) and Group-B (n=27). A paediatric consultant prescribed medications to either Group A or Group B for six months. The patients in Group-A received intravenous infusions of deferoxamine at a dose of 20 mg/kg/day over eight to ten hours for five days in a row. On the other hand, Group-B was given deferasirox orally as a single dose of 30 mg/kg/day after it had been dissolved in water and taken on an empty stomach. For six months, each patient received a monthly check-up. All haematological and biochemical parameters were measured at the beginning of the study and again after 6 months of treatment. Four patients from group A and one from group B were omitted from analysis after failing to complete the study follow-up visit following the start of treatment. Thus, in this study, 26 patients from group B (deferasirox treated group) and 25 patients from group A (deferoxamine treated group) were analyzed. SPSS (Statistical Package for the Social Sciences) version 26 was used for data analysis. Statistical analysis was done by unpaired t test and repeated measure ANOVA. A P value <0.05 was considered statistically significant.

## RESULTS

A total of 51 children were studied. Among them 25 in group A and 26 children in group B. The mean age was 8.52±3.98 years in group A and 8.06±3.12 years in group B. There was no significant difference of age in two groups (table-I). Table-II showed the gender distribution of both groups showed no significant difference (p>0.05).

The effect of deferoxamine and deferasirox on serum ferritin estimated at baseline and 6 months of treatment showed that the serum ferritin was decreased significantly in both groups (p<0.001). In the deferoxamine treated group, the mean haemoglobin (gm/dl) difference from the baseline to the end point of treatment was significant

***Table-I:*** *Age distribution of the study children, n=51.*

| Age group | Group A n (%) | Group B n (%) | p-value |
|---|---|---|---|
| ≤5 years | 8 (32) | 9 (34.6) | 0.924 |
| 6-10 years | 7 (28) | 8 (30.8) | |
| ≥10 years | 10 (40) | 9 (34.6) | |

*Statistical analysis was done by a Chi-square ($\chi^2$) test. Group-A: deferoxamine treated group; Group-B: deferasirox treated group.

***Table-II:*** *Gender distribution of the study children, n=51.*

| Gender | Group A Number (%) | Group B Number (%) | p-value |
|---|---|---|---|
| Male | 12 (48) | 14 (53.8) | |
| Female | 13 (52) | 12 (46.2) | 0.676 |

*Statistical analysis was done by a Chi-square ($\chi^2$) test. Group-A: deferoxamine treated group; Group-B: deferasirox treated group.

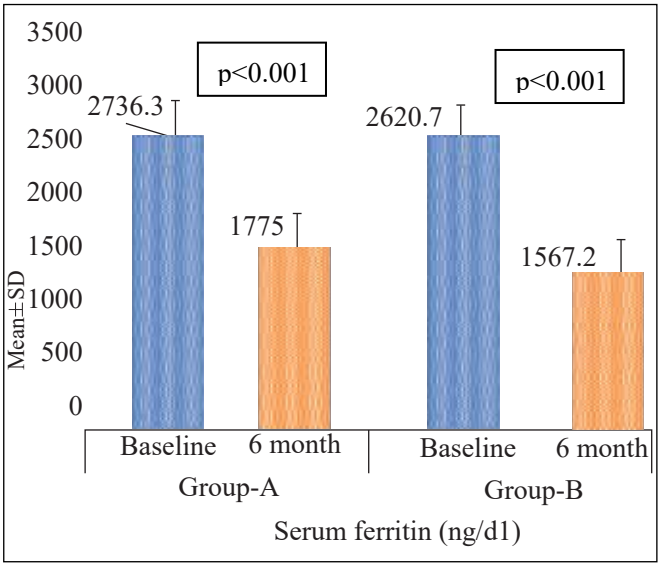


***Figure-1:*** *Comparison of the effect of deferoxamine and deferasirox on serum ferritin estimated at baseline and 6th month of treatment.*

***Table-III:*** *Comparison of the effect of deferoxamine and deferasirox on haemoglobin level estimated at baseline, 1st, 2nd, 3rd, 4th, 5th and 6th month of treatment.*

| Haemoglobin (gm/dl) | Study group | | †p-value |
|---|---|---|---|
| | Group-A (n=25) | Group-B (n=26) | |
| At Baseline | 6.56±1.22 | 7.13±1.37 | 0.122 |
| At 1 month | 6.65 ± 0.72 | 6.95±1.35 | 0.329 |
| At 2 month | 6.78 ±0.35 | 6.91 ±1.39 | 0.647 |
| At 3 month | 7.02±0.41 | 7.38± 0.99 | 0.092 |
| At 4 month | 7.05±0.44 | 7.19±1.00 | 0.513 |
| At 5 month | 6.85±0.53 | 7.08±0.71 | 0.195 |
| At 6 month | 7.06±0.47 | 7.33±0.62 | 0.092 |
| **p-value | 0.016 | 0.168 | |

*Statistical analysis was done by an †unpaired t test and a **repeated measure ANOVA. Data were presented as Mean±SD; Group-A: deferoxamine treated group; Group-B: deferasirox treated group.

***Table-IV:*** *Comparison of the effect of deferoxamine and deferasirox on neutrophil count estimated at baseline, 1st, 2nd, 3rd, 4th, 5th and 6th month of treatment.*

| Neutrophil count (%) | Study group | | †p-value |
|---|---|---|---|
| | Group-A (n=25) | Group-B (n=26) | |
| At Baseline | 45.94±3.11 | 44.85±1.93 | 0.135 |
| At 1 month | 45.70±2.49 | 45.08±1.52 | 0.288 |
| At 2 month | 45.38±2.48 | 44.53±1.52 | 0.149 |
| At 3 month | 45.28±2.66 | 44.70±1.26 | 0.324 |
| At 4 month | 45.60±1.95 | 44.80±1.57 | 0.113 |
| At 5 month | 46.10±2.33 | 45.05±1.76 | 0.077 |
| At 6 month | 45.36±1.29 | 44.62±1.29 | 0.091 |
| **p-value | 0.625 | 0.559 | |

*Statistical analysis was done by an †unpaired t test and a **repeated measure ANOVA. Data were presented as Mean±SD; Group-A: deferoxamine treated group; Group-B: deferasirox treated group.

(p=0.016). But, in the deferasirox treated group, the overall difference from the baseline to the end point of treatment was not significant (p=0.168). When the changes in haemoglobin were compared between two treatment groups, no significant difference was observed before initiation of treatment and throughout the treatment (p>0.05) (table-III).

In the deferoxamine treated group, the difference in mean neutrophil count (%), mean platelet count (K/µL), mean serum ALT (U/L) and serum creatinine (mg/dl) from the baseline to the end point of treatment was not significant (p>0.05). In the deferasirox treated group, the overall difference of the variables from the baseline to the end

***Table-V:*** *Comparison of the effect of deferoxamine and deferasiroxon platelet count estimated at baseline, 1st, 2nd, 3rd, 4th, 5th and 6th month of treatment.*

| Platelet count (K/µL) | Study group | | †p-value |
|---|---|---|---|
| | Group-A (n=25) | Group-B (n=26) | |
| At Baseline | 277.52±16.51 | 288.27±37.72 | 0.197 |
| At 1 month | 280.48 16.96 | 288.08±28.39 | 0.254 |
| At 2 month | 282.40±23.55 | 285.08±24.41 | 0.692 |
| At 3 month | 282.48±18.03 | 284.73±21.00 | 0.684 |
| At 4 month | 283.44±18.24 | 289.35±25.74 | 0.351 |
| At 5 month | 282.40±21.73 | 283.04±20.82 | 0.915 |
| At 6 month | 285.56±19.68 | 280.62±21.38 | 0.395 |
| **p-value | 0.167 | 0.120 | |

*Statistical analysis was done by an †unpaired t test and a **repeated measure ANOVA. Data were presented as Mean±SD; Group-A: deferoxamine treated group; Group-B: deferasirox treated group.

***Table-VI:*** *Comparison of the effect of deferoxamine and deferasirox on serum ALT estimated at baseline, 1st, 2nd, 3rd, 4th 5th and 6th month of treatment.*

| Serum ALT (U/L) | Study group | | †p-value |
|---|---|---|---|
| | Group-A (n=25) | Group-B (n=26) | |
| At Baseline | 28.04±3.93 | 27.08±4.00 | 0.390 |
| At 1 month | 29.36±3.16 | 27.54±4.18 | 0.086 |
| At 2 month | 28.36±2.64 | 28.31±3.53 | 0.953 |
| At 3 month | 29.28±3.73 | 28.04±4.07 | 0.262 |
| At 4 month | 29.00±2.29 | 27.69±3.81 | 0.095 |
| At 5 month | 29.44±3.29 | 27.92±4.29 | 0.164 |
| At 6 month | 29.20±2.55 | 27.35±4.89 | 0.082 |
| **p-value | 0.143 | 0.453 | |

*Statistical analysis was done by an †unpaired t test and a **repeated measure ANOVA. Data were presented as Mean±SD; Group-A: deferoxamine treated group; Group-B: deferasirox treated group.

***Table-VII:*** *Comparison of the effect of deferoxamine and deferasiroxon serum creatinine estimated at baseline, 1st, 2nd, 3rd, 4th 5th and 6th month of treatment.*

| Serum creatinine (mg/dl) | Study group | | †p-value |
|---|---|---|---|
| | Group-A (n=25) | Group-B (n=26) | |
| At Baseline | 0.39±0.06 | 0.37±0.03 | 0.116 |
| At 1 month | 0.39±0.02 | 0.39±0.03 | 0.973 |
| At 2 month | 0.40±0.03 | 0.38±0.02 | 0.072 |
| At 3 month | 0.39±0.04 | 0.38±0.03 | 0.547 |
| At 4 month | 0.40±0.04 | 0.38±0.03 | 0.063 |
| At 5 month | 0.39±0.04 | 0.38±0.03 | 0.163 |
| At 6 month | 0.40±0.04 | 0.38±0.03 | 0.057 |
| **p-value | 0.968 | 0.066 | |

*Statistical analysis was done by an †unpaired t test and a **repeated measure ANOVA. Data were presented as Mean±SD; Group-A: deferoxamine treated group; Group-B: deferasirox treated group.

point of treatment was not significant (p>0.05). When the changes in mean neutrophil count (%), mean platelet count (K/µL), mean serum ALT (U/L) and serum creatinine (mg/dl) were compared between two treatment groups, no significant difference was observed before initiation of treatment and throughout the treatment (p>0.05) (table- IV-VII).

## DISCUSSION

The excessive accumulation of iron is a prominent contributor to both diseases and fatalities among individuals with β-thalassaemia major who rely on blood transfusions. The implementation of iron chelation therapy has resulted in a notable enhancement in the survival rates of individuals diagnosed with thalassaemia. However, the effective regulation of iron overload over an extended period remains poor for a considerable number of patients, primarily due to challenges related to adherence.

Our study showed that in the deferoxamine treated group, the mean serum ferritin (ng/dl) was 2736.30±275.20 before the initiation of treatment which changed to 1775.00±443.54 at 6th month of treatment. Serum ferritin was significantly decreased from baseline to 6 months of treatment (t=9.206; p<0.001). In the deferasiroxtreated group, the mean serum ferritin (ng/dl) was 2620.70±518.27 before the initiation of treatment, which changed to 1567.20±6.73.51 at the 6th month of treatment. Serum ferritin was significantly decreased from baseline to 6 months of treatment (t=9.995; p<0.001). Lal et al. observed that concurrent dosing of deferoxamine and deferasirox significantly decreased serum iron levels without any toxicity[16]. In a study, Kontoghiorghe and Kontoghiorghe observed that both deferoxamine and deferasiroxcan effectively reduce serum ferritin level[17].

In this study, the mean haemoglobin was raised from baseline to the end of treatment of 6 months in the deferoxamine treated group (p=0.016). But the mean haemoglobin did not change from baseline to the end of treatment of 6 months in the deferasirox treated group (p=0.168). Al-Momen et al. found that mean pre-transfusion haemoglobin (g/dl) was 7.5±0.8 in the desferrioxamine group and 7.8±0.9 in the deferasirox group; difference was not significant (p=0.1688)[15].

This study showed that the mean platelet count did not change from baseline to the end of treatment of 6 months in the deferoxamine treated group (p=0.167). Similarly, the mean platelet count did not change from baseline to the end of treatment of 6 months in the deferasirox treated group (p=0.559). Thrombocytopenia was reported in three patient (1.6%) in a study by Vishinski et al. after the start of deferasirox. In one of these patients, deferasirox was discontinued prior to death because of intracranial haemorrhage. Thrombocytopenia resolved without alteration of deferasirox treatment in the other two patients, who both completed the study[18].

In the present study, the mean neutrophil count did not change from baseline to the end of treatment of 6 months in the deferoxamine treated group (p=0.625). Similarly, the mean neutrophil count did not change from baseline to the end of treatment of 6 months in the deferasirox treated group (p=0.120). Neutropenia was reported in one patient (0.5%) by Vichinsky et al. in their study. But the patient was not receiving deferasirox at the time of the reported

neutropenia, as serum ferritin levels were <500 lg/l[18].

This study revealed that the mean serum ALT did not change from baseline to the end of treatment of 6 months in the deferoxamine treated group (p=0.143). Similarly, the mean serum ALT did not change from baseline to the end of treatment of 6 months in the deferasirox treated group (p=0.453). Cassinerio et al. indicated that no considerable hepatic side-effects were seen in deferoxamine and deferasirox receivers. Jasim et al. observed that there is no meaningful difference in operational tests of hepatic function between desferal (deferoxamine) and deferasirox simultaneously and deferasirox receiving alone. Arandi et al. observed that drug treatment therapy can not lead to drug side-effects such as increasing creatinine and hepatic enzyme[19].

In this study, the mean serum creatinine did not change from baseline to the end of treatment of 6 months in the deferoxamine treated group (p=0.968). Similarly, the mean serum creatinine did not change from baseline to the end of treatment of 6 months in the deferasirox treated group (p=0.066). Ashayeri et al. found that serum creatinine elevated in 53.8% and 26.7% in two deferasirox (Exjade and Osveral, respectively) of different companies in Iran[20]. In another study, Eshghi et al. reported an increase in serum creatinine in 24% of cases using deferasirox (Osveral)[21]. Cappellini et al. also reported increase in serum creatinine level as the most common adverse effect of Exjade[22]. In a study, Keikhaei B found that 21% of patients experienced at least one of the side effects, so the most prevalent side effect was an increase in creatinine level[23]. Cassinerio et al. indicated that no considerable renal side-effects were seen in desferal (deferoxamine) and osveral (deferasirox) receivers[24]. Arandi et al. observed that drug treatment therapy can not lead to drug side-effects such as increasing creatinine[20].

## CONCLUSION

Both deferoxamine and deferasirox have the ability to significantly decrease serum ferritin levels after a treatment period of six months. However, neither of these medications had significant effects on blood parameters in children with transfusion-dependent thalassaemia who were experiencing iron overload.

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