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AN OBSERVATIONAL STUDY TO ASSESS THE ADVERSE DRUG REACTIONS OF DEFERASIROX IN TREATING IRON OVERLOAD AMONG PEDIATRIC PATIENTS SUFFERING FROM HAEMOGLOBINOPATHIES AT A TERTIARY CARE HOSPITAL IN EASTERN INDIA

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ABSTRACT **Background:** Haemoglobinopathies, including thalassemia and sickle cell disease, are among the most common monogenic disorders globally. Patients with transfusion-dependent haemoglobinopathies often experience iron overload due to repeated blood transfusions, necessitating iron chelation therapy. Deferasirox, an oral iron chelator, is widely used but associated with adverse drug reactions (ADRs), particularly hepatotoxicity and nephrotoxicity. This study aimed to evaluate the ADR profile of Deferasirox in pediatric patients at a tertiary care hospital in Eastern India. **Objectives:** To assess ADRs, liver and kidney function abnormalities related to Deferasirox in transfusion-dependent paediatric haemoglobinopathy patients; to validate a causality assessment tool; and to contribute toward a standard treatment protocol for iron overload management. **Methods:** A cross-sectional observational study was conducted over 18 months (March 2021–September 2022) at the Thalassemia OPD and indoor pediatric ward of Medical College, Kolkata. 100 children (aged 2–11 years) receiving Deferasirox were included. Data on clinical history, liver and renal function tests, and ADRs were collected and analyzed. **Results:** Elevated SGPT and SGOT levels were observed in 93% and 94% of patients, respectively, indicating significant hepatotoxicity. Alkaline phosphatase and bilirubin were elevated in 81% and 70% of cases. Nephrotoxicity was less common, with only 6% showing reduced eGFR. Hepatic derangements were more prominent in males, and worsening liver function was significantly associated with increasing age ($p<0.05$). **Conclusion:** Deferasirox is associated with a high incidence of hepatotoxicity and a lower incidence of nephrotoxicity in paediatric patients. Regular monitoring of liver and renal function is crucial to detect early toxicity and adjust dosing. Clinician awareness and further research are essential for establishing safer chelation protocols.

KEYWORDS : Haemoglobinopathy, Thalassemia, Deferasirox, Iron overload, Adverse drug reactions, Hepatotoxicity, Nephrotoxicity.

BACKGROUND

Inherited hemoglobin disorders, including thalassemia and sickle-cell disease, are the most common monogenic diseases worldwide. The hallmarks of the disease include imbalance in the α/β- globin chain ratio, ineffective erythropoiesis, chronic hemolytic anemia, compensatory hematopoietic expansion, hypercoagulability, and increased intestinal iron absorption.[1]

The main treatment of thalassemia is blood transfusion and others are supportive therapy, treatment of iron overload symptoms and monitoring.[2] Repeated blood transfusion causes deposition of iron in liver, kidney, spleen etc. Most of the thalassemia patients who have to receive blood transfusions on a regular basis have developed frequent manifestations due to iron overload such as developing heart failure, diabetes, tachyarrhythmia[3].

For detection of iron overload now serum ferritin level is more widely used[4] and various iron chelators have been marketed e.g. Desferioxamine, Defereprone, Deferasirox for the management of that condition.

Deferasirox is an oral iron chelator which is now extensively used to reduce hepatic iron concentration in patients with chronic iron overload due to repeated blood transfusion of patients having haemoglobinopathy with high affinity for iron but less affinity for other trace metals as an advantage.

A comparative study was conducted to evaluate the efficacy of Deferasirox and Deferoxamine. The result demonstrated satisfactory outcomes, indicating that the oral iron chelator Deferasirox exhibited mild adverse effects, including headache, pruritus, and skin discoloration, in contrast to Deferoxamine[5], however the discontinuation rates of Deferasirox and Deferoxamine were found to be approximately equivalent.

The systemic side effects which are documented after administration of Deferasirox are renal failure, hepatic derangement, gastro-intestinal haemorrhages etc.[6] Deferasirox also precipitate hepatic necrosis and mild to moderate microvascular steatosis with derangement of liver enzymes[7]. Nephrotoxicity, as known, is the most serious and frequent adverse effect which usually presents as an acute or chronic reduction of GFR[8].

Aims And Objectives

Primary:

To assess of adverse drug reactions for Deferasirox as an iron chelator agent in transfusion dependent paediatric patients with hemoglobinopathies with special references to liver function and renal function.

Secondary:

1. To prepare and validate appropriate causality assessment tools for suspected adverse drug events caused by Deferasirox.
2. To develop the standard treatment protocol which will help the clinician's decision regarding the management of iron overload in the patients suffering from transfusion dependent haemoglobinopathy.

MATERIALS AND METHODS

A hospital-based, cross-sectional, single-centered, observational study was conducted within the Thalassemia Outpatient Department (OPD) under the aegis of the department of Haematology, the Indoor Thalassemia Paediatric Ward and the Clinical Pharmacology Department at Medical College, Kolkata, spanning an 18-month duration (March 2021 to September 2022). The study cohort comprised paediatric patients (aged 2-11 years) diagnosed with haemoglobinopathies who met the predefined inclusion criteria and were receiving therapeutic blood transfusions in conjunction with Deferasirox as an iron-chelating agent. The sample size, determined to be 100 based on the prevalence of Deferasirox-induced nephrotoxicity

(65%), was estimated using the formula $N = 4pq/l^2$ (where p denotes nephrotoxicity prevalence, q represents 1-p, and l signifies the margin of error), with an attrition rate of 10% accounted for. The inclusion criteria encompassed paediatric subjects (aged 2-11 years) diagnosed with haemoglobinopathies, undergoing transfusion therapy and Deferasirox administration for at least three months, and providing assent for study participation. Exclusion criteria involved patients outside the specified age range, those with established renal or hepatic dysfunction (e.g., chronic kidney disease, hepatitis B or C, nephrotic syndrome, HIV, hepatocellular carcinoma), and individuals on other nephrotoxic or hepatotoxic medications. The sampling framework encompassed all eligible patients whose legal guardians provided informed consent for participation in this tertiary care hospital-based study in Eastern India. Subject recruitment transpired weekly on Mondays at the Thalassemia Clinic and biweekly at the Indoor Paediatric Ward. Data collection entailed meticulous documentation of demographic details, clinical evaluations, and pertinent laboratory investigations in the Case Report Form (CRF), with adverse drug reactions (ADRs) systematically recorded in both the CRF and the CDSCO Suspected ADR Reporting Form (Version 1.4). The study methodology entailed structured clinical interviews, anthropometric assessments (height, weight), comprehensive physical examinations, and extensive laboratory evaluations. Statistical analysis was conducted within the Department of Pharmacology using IBM SPSS Statistics (Version 22) and Microsoft Excel (Office 2022), employing descriptive statistics (mean ± standard deviation, median, and percentages) and inferential analyses with a 95% confidence interval, considering a p-value < 0.05 as indicative of statistical significance.

RESULTS

A cross-sectional observational study was conducted specially in the Thalassemia clinic under the purview of the department of Haematology. The acquired data underwent rigorous scrutiny for completeness and accuracy before undergoing statistical analysis in the Department of Pharmacology. The study comprised a sample size of 100 patients diagnosed with haemoglobinopathies who met the inclusion criteria as delineated in the study protocol.

Regarding the age distribution, among the total cohort (n=100), the majority (80%) were classified within the late childhood demographic. A male preponderance was noted in the study population, with a male-to-female ratio of 1.17. In the Beta Thalassemia cohort, the proportion of female patients was 40%, while male patients constituted 62.22%. Conversely, within the e-Beta Thalassemia cohort, females accounted for 52.72%, whereas males comprised 49.09%.

The body mass index (BMI) distribution was analyzed among the study participants. Of the 100 enrolled subjects, 27% were categorized as underweight (BMI <13), 4% were classified as overweight (BMI >20), and the remaining 69% fell within the normal BMI range (13-19). The transfusion-dependent haemoglobinopathy population undergoing Deferasirox therapy exhibited a mean BMI of 14.60 ± 2.17 kg/m².

Table 1: Table Showing Analysis Of BMI

PARAMETER	VALUE
MEAN	14.60
STANDARD DEVIATION	2.17
MINIMUM	10.41
MAXIMUM	20.41
MEDIAN	14.67

Liver function parameters were assessed in the study cohort. Only 7% of subjects demonstrated normal serum glutamate pyruvate transaminase (SGPT) levels, whereas 93% exhibited SGPT abnormalities. The SGPT levels ranged from a minimum of 25 U/L to a maximum of 405 U/L, with a mean value of 153.2 ± 82.89 U/L. Among male participants, SGPT abnormalities were observed in 57%, whereas 42% of female subjects exhibited abnormal SGPT levels, with normalization occurring in 3% of males and 4% of females.

Table 2: Table Showing Analysis Of SGPT

PARAMETER	VALUE
MEAN	153.2
STANDARD DEVIATION	82.89
MINIMUM	25
MAXIMUM	405
MEDIAN	138.5
95% CONFIDENCE INTERVAL	169.44 – 136.95

Similarly, serum glutamate oxaloacetate transaminase (SGOT) abnormalities were detected in 94% of the study population, while only 6% exhibited normal SGOT levels. Among male subjects (n=54), 52% exhibited abnormal SGOT values, whereas 54.2% maintained normal levels. Among female participants (n=45), 36% exhibited SGOT abnormalities, while 9% demonstrated normal levels. SGOT levels varied between 22 U/L and 335 U/L, with a mean value of 128.5 ± 64.47 U/L.

Table 3: Table showing analysis of SGOT

PARAMETER	VALUE
MEAN	128.5
STANDARD DEVIATION	64.67
MINIMUM	22
MAXIMUM	335
MEDIAN	138.5
95% CONFIDENCE INTERVAL	141.13 – 115.86

Alkaline phosphatase levels were elevated in 81% of the study population, whereas 19% exhibited normal levels following Deferasirox therapy. Among male participants (54%), 45% demonstrated elevated alkaline phosphatase levels, while 9% maintained normal levels. Among female participants (45%), 35% exhibited elevated levels, while 10% demonstrated normal values. The alkaline phosphatase levels ranged between 100 U/L and 566 U/L, with a mean value of 321.55 ± 107.35 U/L.

Serum bilirubin abnormalities were observed in 41% of the male population, whereas 54% exhibited normal levels. Among female participants, 28% had elevated bilirubin levels, while 18% maintained normal values.

Statistical correlation analysis revealed a significant association between age and hepatic dysfunction, as evidenced by the following Pearson's correlation coefficients and p-values: SGPT (r = 0.8839, p = 0.046), SGOT (r = 0.7465, p = 0.013), alkaline phosphatase (r = 0.7959, p = 0.0027), and serum bilirubin (r = 0.702, p = 0.002). These findings suggest that advancing age correlates with a progressive decline in liver function, likely attributable to prolonged exposure to Deferasirox therapy.

Table 4: Table showing Pearson's correlation with respect to age

Parameter	Pearson's Correlation Coefficient 'r'	P Value
SGPT	0.8839	0.046 (S)
SGOT	0.7465	0.013 (S)
Bilirubin	0.702	0.002 (S)
ALP	0.7959	0.0027 (S)

Regarding renal function, estimated glomerular filtration rate (eGFR) abnormalities were detected in 6% of the study population. Among female participants (46%), 42% exhibited normal eGFR values, whereas 4% demonstrated abnormalities. On the other hand, 2% of the males studied showed eGFR anomalies. The mean eGFR across the study population was 152.48 ± 29.33 mL/min/1.73 m².

Table 5: Table showing analysis of eGFR

PARAMETER	VALUE
MEAN	152.48
STANDARD DEVIATION	29.33
MINIMUM	39
MAXIMUM	191
MEDIAN	157
95% CONFIDENCE INTERVAL	158.22 – 146.73

Additionally, unconjugated hyperbilirubinemia was observed in association with hepatocellular jaundice, with a mean bilirubin level of 1.75 ± 1.363 mg/dL.

The most frequently observed adverse drug reaction was hepatotoxicity, with nephrotoxicity occurring to a lesser extent. Notably, no significant gastrointestinal, dermatological, central nervous system, cardiovascular, or neurological adverse effects were documented.

In response to adverse drug reactions, Deferasirox dosage adjustments were implemented, with some cases necessitating discontinuation of therapy. Among documented adverse drug reactions (91%), 52% were observed in male subjects, while 9% were noted in female subjects.

Table 6: Table showing distribution of abnormalities of SGPT, SGOT, ALP, Bilirubin and eGFR amongst the total population according to gender.

PARAMETERS	%AGE OF MALES	%AGE OF FEMALES
SGPT	57	42
SGOT	52	36
ALP	45	35
Bilirubin	41	28
eGFR	2	4

DISCUSSION

Chronic iron overload in thalassemia patients as a result of repeated blood transfusion is an important problem in the day-by-day treatment. This causes early mortality unless adequate chelation therapy has been administered. Iron chelation therapy becomes mandatory when serum ferritin level crosses 1000 ng/mL or after 10-20 units of packed RBCs have been transfused[9].

Deferasirox is an oral iron chelating agent used to treat chronic iron overload. Deferasirox has been linked to transient serum aminotransferase elevations during therapy and to rare instances of clinically apparent liver injury that can be severe and even fatal[10].

The pattern of the liver injury is typically hepatocellular or mixed with prominent elevation serum Transaminase level. The injury may be due to direct, intrinsic toxicity and have a more severe outcome in patients with pre-existing liver disease as a result of iron overload or concurrent hepatitis B or C. Deferasirox is metabolised in liver largely by glucuronidation and biliary excretion[11].

In most of the cases the Deferasirox induced adverse drug reactions especially development of hepatotoxicity usually take to develop for about several years but in most of the cases it can develop within 1-3 months[12].

In this study, the number of male patients was less than that of females. Out of 100 transfusion dependent haemoglobinopathy paediatric patients (n=100) attending the Thalassaemia OPD of Medical College Kolkata between "March 2021- Sep 2022", 46% (n=46) were female and 64% (n=64) were male. (Male: Female=1.17).

Table 7: Table circumscribing the results in a nutshell.

Parameter	BMI	SGPT	SGOT	Alkaline Phosphatase	Serum Bilirubin	eGFR
Mean	14.60	153.2	128.5	321.55	1.75	15.248
Standard Deviation	2.17	82.89	64.47	107.35	1.363	157
Minimum	10.41	25	22	100	6.33	29.33
Maximum	20.41	405	335	329	1.21	191
Median	14.67	138.5	138.5	566	0.12	39
95% Confidence Interval		169.44-136.95	141.13-115.86	342.59-300.5	2.01-1.48	158.22-146.73

A study done by Ladis VL et al on 85 patients with transfusion-dependent iron overload noted more female patients than males (female: male=1.3)[13] .This difference in gender distribution might be due to geographical variation.

All the patients were treated with oral Deferasirox as the iron chelating agent. The mean age of the recruited population was 9.54 ± 2.07 years. The patients receiving Deferasirox who had transfusion dependent haemoglobinopathy had a mean BMI of 14.60 ± 2.17 kg/m^2. In a large multicentric study of 123 deferasirox receiving transfusion dependent haemoglobinopathy done by Piolatto A et al, the patients' documented BMI was 20.9 ± 3.8.[14] In another study (Allegra et al), the median BMI was 15.45 kg/m^2,[11] but in this study the median BMI is 14.67 kg/m^2.

In the present study, numbers of adverse drug reactions were reported. Among them drug induced hepatotoxicity (n=91) and drug induced nephrotoxicity (n=6) have been seen. In this study, recruited patients did not present with any Gastrointestinal, Cardiovascular, Central Nervous system, and any Dermatological adverse effect.

Though in Western countries even in East Asia there was more prevalence of nephrotoxicity whereas in this study LFT derangement is mostly found. Though fewer nephrotoxicity cases were found in this study. There was no development of other major adverse effects due to Deferasirox intake.

A multi centered study completed by Vichinsky et al represent that among 247 patients, 5 patients had serum creatinine elevation, while increased SGPT level more than 5 times the normal was observed among 11 patients[15].

The present study showed that the maximum value of SGPT was 405 and minimum value of SGPT was 25 among 100 recruited population who had received Deferasirox as an iron chelator suffering from transfusion dependent haemoglobinopathies. The mean value of SGPT was 153.2 (± 82.89), and the mean value of SGOT in the recruited population was 128.5 ± 64.47. Another observational single centered study was conducted by Thakor DR et al, in Gujarat, India where they found 14% hepatic derangement patients among their 55 study population[16].

Another study was conducted by Soliman A et al in Middle East where they found significant decrease in SGOT and SGPT levels in patients receiving Deferasirox in 20 mg/kg dosing[15].

In the present study, the maximum value of Alkaline Phosphatase was 566 and minimum value was 100. The mean value of ALP is 321.55 ± 107.35. The abnormality in alkaline phosphatase level was seen in 86%, while normal level was seen among 14%.

A single centred study was done on 85 transfusion dependent haemoglobinopathy patients by Younis MA, in Middle East, Iraq, where 25.8% of population has elevated alkaline phosphatase level[17]. In the present study, Among 100 study populations, 70% of subjects have presented with elevated levels of serum Bilirubin and 30% have presented with normal levels of bilirubin. The mean Bilirubin value is 1.75 (± 1.363). Maximum level=6.33, Minimum level=0.12.

There was reduction in eGFR only in n=6 of the total population. Mean eGFR was 152.48 ± 29.33 in this study.

Though in Western countries even in East Asia most of the cases of nephrotoxicity as an adverse drug reaction of Deferasirox treatment were seen frequently. Although, in the present study there was less development of nephrotoxicity in comparison.

Hence, it has been proven that Deferasirox causes hepatotoxicity, nephrotoxicity, and GI intolerance. However, in this study we have seen that there were mostly cases of Hepatotoxicity and a number of cases with nephrotoxicity were reported.

Limitations Of The Study

Single- centered, cross sectional, small small size were the limitations of this study. As it was an observational study so due to inability to conclude about the cause- effect relationship the underneath pathophysiology for hepatocellular and renal impairment could not be explored.

To recommend the standard treatment protocol which will help the clinician's decision regarding the management of iron overload in the patients suffering from transfusion dependent haemoglobinopathy more multi-centric, analytical or RCT with large scale samples have to be conducted future.

CONCLUSION

Deferasirox is an oral iron chelating agent which is used to treat chronic iron overload due to repeated blood transfusion, this cause transient elevations of serum SGPT, SGOT level and it has ability to precipitate hepatocellular injury (Drug induced hepatitis) which is usually self-limited and benign. However, it has also been implicated in case of clinically apparent liver injury and acute liver failure.

Deferasirox may causes potential nephrotoxicity. It elevates both serum urea and creatinine level. It causes significant reduction in eGFR.

Hence this is recommended to monitor the liver function test and measurement of serum urea and creatinine levels before and every 2 weeks for at least first months of treatment. There after monthly follow up is needed.

Persistent serum SGPT, SGOT and ALP levels elevation along with

rising of serum urea and creatinine level and values above 5 times under normal limit should be taken under serious concern and dose reduction/ dose titration may be initiated or temporary stopping of therapy may be suggested. Administration of Deferasirox in the paediatric age group should be given cautiously.

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