International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

To Evaluate Effectiveness of Ferric Carboxymaltose in Treating Moderate Iron Deficiency Anemia in Pediatric Patients an Observational Study

Dr. Gaurav Mathur[1], Dr. Pankaj Kumar Singhal[2], Dr. Kavya R[3], Dr. Monika Dhakad[4]

Abstract: ***Background**: Iron deficiency anemia (IDA) is a major public health concern in pediatric populations, particularly in developing countries such as India. Oral iron therapy is limited by poor compliance and gastrointestinal side effects. Intravenous ferric carboxymaltose (FCM) offers a potential alternative for rapid correction of anemia. **Aim**: To evaluate the effectiveness and safety of intravenous ferric carboxymaltose in children with moderate iron deficiency anemia. **Methods**: This study was conducted in 93 pediatric patients aged 1–18 years with moderate iron deficiency anemia. Baseline hemoglobin (Hb), mean corpuscular volume (MCV), and serum ferritin were assessed. Quantitative C-reactive protein (Q-CRP) and stool occult blood tests were performed to exclude inflammatory states and blood loss. Mentzer index was used to exclude thalassemia. All patients received a single dose of intravenous ferric carboxymaltose (15 mg/kg). Hemoglobin levels were reassessed after 21 days. Statistical analysis was performed using paired t-test. **Results**: The mean baseline hemoglobin was 8.02 ± 0.82 g/dL, which increased to 9.52 ± 1.08 g/dL after 21 days, with a mean rise of 1.50 ± 1.02 g/dL ($p < 0.000001$). A total of 94.6% of patients demonstrated improvement. No statistically significant correlation was observed between serum ferritin and hemoglobin levels ($r = –0.09$, $p > 0.05$). Adverse effects were minimal, occurring in 2.15% of patients. **Conclusion**: Intravenous ferric carboxymaltose appears to be a safe and effective option for short-term hematological improvement in children with moderate iron deficiency anemia. Further controlled studies with longer follow-up are warranted to confirm these findings.***

Keywords: Iron deficiency anemia; Ferric carboxymaltose; Intravenous iron therapy

1. Introduction

Iron deficiency anemia (IDA) is the most prevalent nutritional deficiency worldwide and remains a major public health concern, particularly in developing countries such as India.(1,11,13) It is estimated that a substantial proportion of children suffer from anemia, with data from the National Family Health Survey (NFHS-5, 2019–21) indicating that approximately 67.1% of children aged 6–59 months are anemic.(23) The burden of anemia extends beyond hematological parameters, significantly impacting neurocognitive development, immune function, physical growth, and overall quality of life. Early identification and effective treatment are therefore crucial to prevent long-term morbidity.

Iron plays a fundamental role in multiple physiological processes, including oxygen transport, cellular respiration, and enzymatic reactions. Deficiency of iron leads to impaired hemoglobin synthesis(12) and reduced oxygen-carrying capacity of blood, resulting in tissue hypoxia. In children, this can lead to irreversible cognitive deficits, reduced school performance, and increased susceptibility to infections.(10,14)

Traditionally, oral iron therapy has been the cornerstone of treatment for iron deficiency anemia due to its low cost and ease of administration. However, oral iron therapy is associated with several limitations that significantly impact its effectiveness in real-world settings. Gastrointestinal side effects such as nausea, vomiting, constipation, and abdominal discomfort are common and often lead to poor compliance.(3,11) Additionally, the metallic taste and poor palatability of oral iron preparations further reduce adherence, particularly in pediatric patients. The requirement for prolonged therapy, often extending over several months, also contributes to treatment discontinuation. Furthermore, conditions such as malabsorption, chronic inflammation, and concurrent infections can impair intestinal iron absorption, limiting the efficacy of oral therapy.

Comparison of oral and intravenous iron therapy

Type of therapy	Duration of therapy	Cost	Quality of life
Gastrointestinal	At least three months	Low, but the need for laboratory and clinical follow up increases cost	It depends upon patient compliance,anemia correction and follow up visits for clinical and laboratory work up
Infusional	Correction of total iron deficits with a single or few transfusions	High, due to the acquisition cost of parenteral iron	Outpatient or inpatient administration, frequently only once

In contrast, intravenous iron preparations offer several advantages by bypassing gastrointestinal absorption and enabling rapid replenishment of iron stores. Among the available formulations, ferric carboxymaltose (FCM) is a newer, non-dextran intravenous iron preparation that allows administration of large doses in a single sitting with a favorable safety profile.(15,17) FCM is a stable iron-carbohydrate complex that releases iron in a controlled manner, minimizing the risk of free iron toxicity and hypersensitivity reactions.(5,15) Unlike older iron formulations, it does not require a test dose and can be administered over a short duration, making it more convenient for both patients and healthcare providers.

Although FCM has been extensively studied and widely used in adult populations, its use in pediatric patients has been limited until recently.(7,8) Regulatory approval for pediatric use has been granted only in recent years, and there remains a relative paucity of data regarding its safety and efficacy in children, particularly in developing countries. Most available pediatric studies are limited in sample size or are restricted to specific subgroups such as chronic kidney disease or inflammatory bowel disease. Therefore, there is a need for well-designed studies evaluating the role of FCM in the broader pediatric population with iron deficiency anemia.

The present study focuses on children with moderate iron deficiency anemia. This subgroup was specifically chosen to ensure a clinically stable population, allowing for safe administration of intravenous iron therapy without the immediate need for blood transfusion, which is often required in severe anemia. Studying moderate anemia also provides a clearer assessment of therapeutic response without confounding effects of acute interventions or physiological instability.

An additional strength of this study lies in its rigorous methodological approach to accurately diagnose iron deficiency anemia and exclude potential confounders. Serum ferritin, although widely used as a marker of iron stores, is an acute-phase reactant and may be falsely elevated in the presence of inflammation. To address this limitation, quantitative C-reactive protein (Q-CRP) was measured in all patients to exclude inflammatory conditions. Stool examination for occult blood was performed to rule out chronic gastrointestinal blood loss as an alternative cause of anemia. Furthermore, Mentzer index was calculated to differentiate iron deficiency anemia from thalassemia trait, which is a common cause of microcytic anemia in the Indian population.

By eliminating these confounding factors, the study ensures a more accurate representation of true iron deficiency anemia, thereby enhancing the validity of the findings. In this context, the present study was undertaken to evaluate the effectiveness and safety of intravenous ferric carboxymaltose in pediatric patients with moderate iron deficiency anemia and to contribute to the growing body of evidence supporting its use in this population

Research Questions

1) What is the efficacy of FCM in increasing hemoglobin levels in children with moderate iron deficiency anemia?
2) What are the safety and side effects associated with FCM treatment in this population?

2. Aims and Objectives

Aim

To study the effectiveness of Ferric carboxymaltose in treatment of iron deficiency anemia in pediatric patients aged 1-18 years.

Objectives:

1) To study the change in Hemoglobin level pre and post FCM infusion
2) To study the adverse effects following FCM infusion.

3. Materials and Method

- **Type of Study**: Observational Study
- **Period of Study**: The study will start after obtaining institutional ethical committee approval for a period of 1 year
- **Place of Study**: Department of Paediatrics, J K LON Mother and Child Hospital Kota.
- **Study Population:** Pediatric age group from 1 year to 18 years with moderate microcytic hypochromic anemia

Sample Size: 93

Sample Size Formula

Sample size to estimate a simple proportion (apparent prevalence). Prevalence of children with moderate iron deficiency anemia in India	0.40 (40.00%)
Desired precision of estimate [Error10%]	0.10
Confidence level	95
Population size	N/A

Sample size required for specified inputs
Largepopulation-93

The sample size required to estimate a proportion (or prevalence) with a specified level of confidence and precision.

Inputs are the assumed or estimated value for the proportion, the desired level of confidence, the desired precision of the estimate and the size of the population for limited population sizes. The desired precision of the estimate (also sometimes called the allowable or acceptable error in the estimate) is half the width of the desired confidence interval. For example, if you would like the confidence interval width to be about 0.1(10%) you would enter a precision error of +/-0.1(10%). The sample sizes required to estimate the true value with the desired precision (errors) and confidence, for both an infinite population and for a population of the specified size. If population size is left blank or zero, only the sample size for an infinite population is calculated.

Note: Adjustment for finite population size may underestimate required sample size unless this is also taken into account when estimating variance and resulting confidence interval.

Sample size is calculated using the formula:

$$n = (Z^2 \times P \times (1 - P))/e^2$$

Where:
Z = value from standard normal distribution corresponding to desired confidence level(Z=1.96for 95% CI)
P is expected true proportion
E is desired precision(half desired CI width).

Inclusion Criteria

Children of age group 1 year to 18 years presenting to IPD of J K LON hospital with Hemoglobin 7-9.9gm/dl with Microcytic Hypochromic Anemia

Exclusion Criteria

1) Patient less then 1year of age and more than 18years of age
2) Patient presenting with severe anemia i.e Hb <7.0 gm/dl OR with mild anemia >10g/dl

3) Patient having Macrocytic Anemia
4) Patient who are known case of/ diagnosed to have Metabolic/ Genetic disorders (Thalassemia/sickle cell anemia/Pure red cell aplasia/Congenital dyserythropoetic anemia/Aplastic anemia)
5) Patient with raised Q-CRP (> 6) and Occult blood in stool
6) Patients whose parents or legal guardians did not consent to the use of the child's data for scientific research upon admission to the hospital.

4. Methodology

A detailed history and consent will be taken of all patients presenting with moderate iron deficiency anemia , Enrolled children will treated according to standard PICU protocols Initial blood samples will be collected within 24 hours of ICU admission. Detailed history regarding symptoms, past/medical history will be collected from parents/relatives. All children will undergo anthropometric measurements, detailed general/systemic examination. Relevant laboratory investigations will done at admission, such as total white blood cells count, differential count, platelet count and serum ferritin levels.

2 Samples will be collected 1 in EDTA vial for Complete Blood Count and 1 in Red serum vial, Samples will be sent to Central laboratory of J K LON Hospital, Complete blood count will be done using automated hematological analyser. And Serum ferritin, that will be estimated using Beckman Coulter kit by chemi luminescence method, The Beckman Coulter AU procedure is based on the measurement of the decrease in light intensity transmitted (increase in absorbance) through particles suspended in solution as a result of complexes formed during the antigen-antibody reaction. The anti-ferritin reagent is a suspension of polystyrene latex particles, of uniform size, coated with polyclonal rabbit anti-ferritin antibody. When serum, containing ferritin, is mixed with the anti-ferritin reagent, an agglutination mixture occurs. This is measured spectrophotometrically on Beckman Coulter AU Chemistry Analyzers.

1 sample will be sent for Qcrp and 1 stool sample will be sent for examination for occult blood, patients with high CRP or having occult blood present in stool sample will be excluded from study.

After which, Injection Ferric Carboxymaltose (FCM) transfusion will be done in ICU setup at a dose of 15mg/kg/dose , transfused slowly over one hour after doing AST , patients vitals will be closely monitored during transfusion inorder to watch for any adverse drug reaction Date and time of transfusion done will be recorded in the data and patient will followed up till 21 days of transfusion , after 21 days a repeat complete blood count of the patient will be done inorder to compare the changes in levels of Hematocrits

5. Review of Literature

Bartosz Korczowski et al (2023) FCM is well tolerated by pediatric patients. Improvements in Hb were greater with the higher dose, supporting use of the FCM 15mg/kg dose in pediatric patients. They compared effectiveness of two dose of FCM i.e 7.5MG/kg/dose and 15mg/kg/dose.The results of this Phase 2 trial show that treatment with IV FCM was generally well tolerated and produced clinically meaningful, dose-proportional increases in mean hemoglobin concentrations in pediatric patients with IDA. FCM has very low immunogenic potential, and its properties permit rapid administration (e.g., 15 min) of large, single doses, which allows for rapid repletion of total body iron and supports the utility of FCM in the treatment of IDA in pediatric patients, it was concluded from the study that the overall most commom side effect post transfusion was Urticaria.

Hulya Ozsahin et al (2020) conducted a study on patients already taking oral Iron therapy and showing no signs of resolution, study concluded that the most common cause of Anemia was nutritional and post transfusion 49% of patients showed a complete hematological response over 6-12weeks of post transfusion, the study concluded that FCM is safe and efficacious in children aged 18 months and older. Single-dose FCM treatment was followed by clinical improvement with advantages of safety, compliance and lower cost compared with the previous generation parenteral iron preparations.

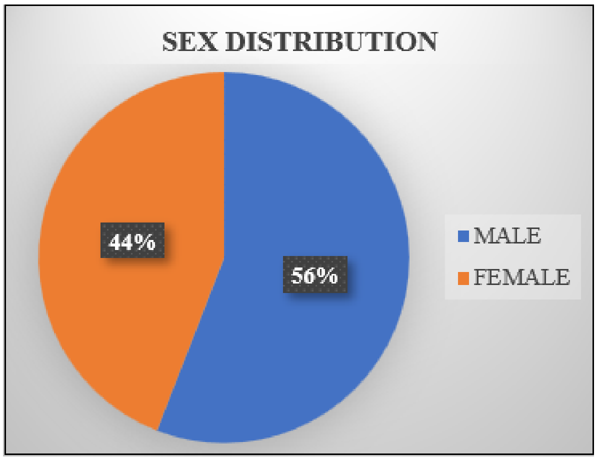
Nicholas Carman et al (2018) FCM is an effective and practical way to reduce the burden of iron deficiency anemia in inflammatory bowel disorder patients and allows for repeated infusions without overstraining resources. The study showed A total of 44% of patients underwent treatment for Iron deficiency Anemia, while 56% were for Inflammatory bowel disease without anemia. Following FCM infusion, 64% of patients with IDA had resolution of anemia, with 81% showing resolution for ID without anemia. Elevation of CRP throughout the study period had no influence on resolution of Iron Deficiency anemia with FCM.

Jacquelyn M. Powers et al (2015) Intravenous FCM administered as one or two short IV infusions and without need for a test dose is safe and effective in most children and adolescents with IDA refractory to oral iron therapy

6. Observations and Results

Table 1: Demographic Details- Sex Distribution of Cases Taken for Study

Variable	Number (n=93)	Percentage (%)
Male	52	55.9
Female	41	44.1



Paper ID: SR26704160308 DOI: https://dx.doi.org/10.21275/SR26704160308

Table 2: Age Wise Distribution of Patient Taken for Study

Variable	Number (n=93)	Percentage (%)
1–5 years	28	30.1
6–10 years	34	36.6
11–18 years	31	33.3

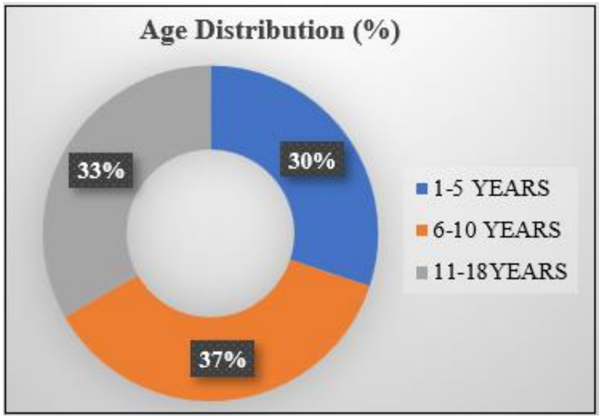
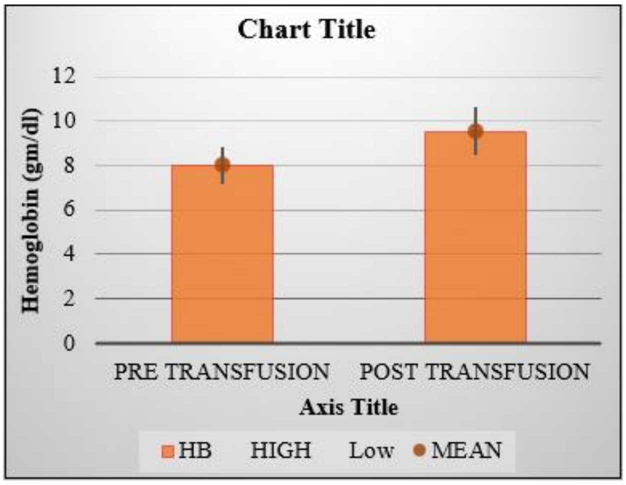


Table 3: Base Line Hematological Parameters before FCM Trial

Parameter	Mean ± SD
Hemoglobin (g/dL)	8.02 ± 0.82
MCV (fL)	65.15 ± 35.74
Serum Ferritin (ng/mL)	76.49 ± 108.37

Table 4: Variation of Hemoglobin Post FCM Transfusion

Parameter	Mean ± SD	Mean Difference	p-value
Pre-treatment Hb	8.02 ± 0.82	1.50 ± 1.02	<0.000001
Post-treatment Hb	9.52 ± 1.08		



The mean hemoglobin level increased significantly from **8.02 ± 0.82 g/dL at baseline** to **9.52 ± 1.08 g/dL at 21 days post-infusion**, with a mean rise of **1.50 ± 1.02 g/dL**, which was found to be **statistically highly significant (p < 0.000001)**.

Table 5: Age-stratified Hematological Response to Ferric Carboxymaltose

Age Group	n	Mean Hb Rise (g/dL)	SD
1–5 years	71	1.46	0.91
6–10 years	10	1.39	1.10
11–18 years	12	1.84	1.51

Age-wise analysis revealed that the mean rise in hemoglobin was highest in the 11–18 years age group (1.84 ± 1.51 g/dL), followed by the 1–5 years group (1.46 ± 0.91 g/dL), while the lowest rise was observed in children aged 6–10 years (1.39 ± 1.10 g/dL). This suggests a trend toward better hematological response with increasing age.

Table 6: Hemoglobin Increment

Hb Rise (g/dL)	Number	Percentage (%)
<1	21	22.6
1–2	49	52.7
>2	23	24.7

A clinically meaningful improvement in hemoglobin levels was observed in most patients, with **77.4%** achieving a rise of **≥1 g/dL**, including **24.7%** with an increase of **>2 g/dL**, while **22.6%** showed a rise of less than **1 g/dL**.

Table 7: Treatment Outcome

Outcome	Number	Percentage (%)
Improved	88	94.6
No improvement	5	5.4

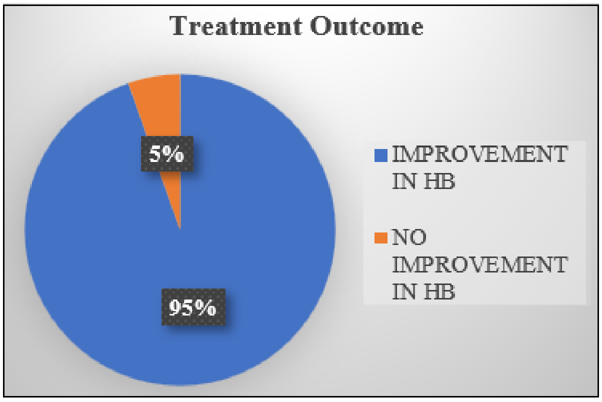
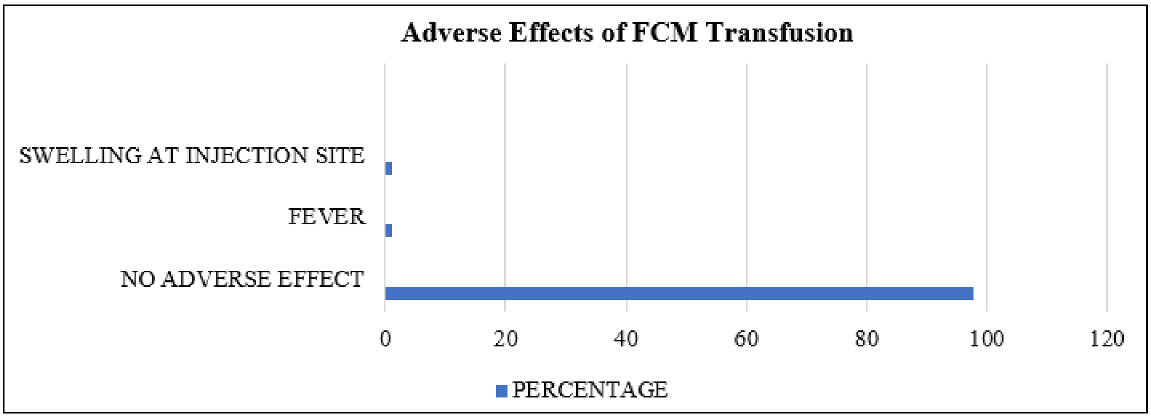


Table 8: Adverse Effect of FCM Transfusion

Effect	Number	Percentage (%)
None	91	97.8
Fever	1	1.1
Injection site swelling	1	1.1



Ferric carboxymaltose demonstrated an excellent safety profile, with **only 2.15% of patients experiencing mild adverse effects**, while the majority (**97.8%**) had no complications, and no serious adverse reactions were reported.

Table 9: Relationship of Serum Ferritin and Hemoglobin Levels

Parameter	Value
Mean Serum Ferritin (ng/mL)	76.49 ± 108.37
Mean Hemoglobin (g/dL)	8.02 ± 0.82
Correlation Coefficient (r)	-0.09
Statistical Significance (p-value)*	>0.05 (Not significant)

No statistically significant correlation was found between serum ferritin and baseline hemoglobin levels ($r = -0.09$, $p > 0.05$), indicating variability in ferritin levels among patients with iron deficiency anemia.

Table 10: Comparison Of Pre Transfusion And Post Transfusion Level Of MCV

Parameter	Mean ± SD (fL)	Mean Difference (fL)	p-value
Pre-treatment MCV	65.15 ± 35.74	+2.64	0.48
Post-treatment MCV	67.79 ±		

Although a slight increase in mean MCV was observed following ferric carboxymaltose therapy, the change from **65.15 fL to 67.79 fL** was **not statistically significant (p = 0.48)**.

7. Discussion

The present study demonstrates that intravenous ferric carboxymaltose administered at a dose of 15 mg/kg is effective in achieving rapid hematological improvement in pediatric patients with moderate iron deficiency anemia. A statistically significant rise in mean hemoglobin levels was observed within 21 days of administration, indicating the efficacy of a single high-dose infusion in correcting anemia. This finding is consistent with previous studies, which have demonstrated that higher doses of ferric carboxymaltose are associated with greater and more sustained improvements in hemoglobin levels while reducing the need for repeated hospital visits.

The study population showed a slight male predominance, with males constituting approximately 55.9% of cases. This distribution may reflect sociocultural and healthcare-seeking patterns, as well as possible nutritional and environmental differences influencing anemia prevalence. However, the relatively balanced representation of both sexes suggests that iron deficiency anemia remains a significant concern across the pediatric population irrespective of gender.

Age-wise distribution revealed that the majority of patients belonged to the 6–10 years age group, followed by adolescents and younger children. This pattern may be attributed to increased nutritional demands during periods of rapid growth, coupled with inadequate dietary iron intake, making this age group particularly vulnerable to iron deficiency anemia.

In addition to age distribution, age-wise analysis of treatment response revealed that the mean rise in hemoglobin was highest in the 11–18 years age group (1.84 ± 1.51 g/dL), followed by the 1–5 years group (1.46 ± 0.91 g/dL), while the lowest rise was observed in children aged 6–10 years (1.39 ± 1.10 g/dL). This suggests a trend toward better hematological response with increasing age. The relatively higher response observed in adolescents may be attributed to increased physiological demands during growth spurts, enhanced bone marrow responsiveness, and potentially better treatment compliance compared to younger children.

A marked and statistically highly significant increase in hemoglobin levels was observed following treatment, with mean values rising from 8.02 g/dL at baseline to 9.52 g/dL at 21 days. The majority of patients demonstrated a clinically meaningful response, with more than three-fourths achieving a rise of at least 1 g/dL and nearly one-fourth showing an increase exceeding 2 g/dL. This rapid improvement highlights the advantage of intravenous ferric carboxymaltose over oral iron therapy, which typically requires several weeks to months to achieve comparable results.

Baseline red cell indices revealed microcytic anemia, as indicated by reduced mean corpuscular volume values. Following therapy, a slight increase in mean corpuscular volume was observed; however, this change was not statistically significant. This finding is expected, as hemoglobin levels tend to improve earlier than red cell indices during iron therapy. The observed trend toward normalization of erythropoiesis further supports the therapeutic effectiveness of ferric carboxymaltose.

No statistically significant correlation was observed between serum ferritin and hemoglobin levels. This finding highlights the limitation of serum ferritin as a standalone marker of iron status, as it is an acute-phase reactant and may be influenced by subclinical inflammation and other factors. The lack of correlation, despite the exclusion of overt inflammatory conditions, suggests inherent variability in ferritin levels and supports the need for a comprehensive diagnostic approach when evaluating iron deficiency anemia.

The safety profile of ferric carboxymaltose in the present study was favorable. The majority of patients did not experience any adverse effects, and only a small proportion reported mild reactions such as fever and injection site swelling. No serious adverse events were observed. These findings are consistent with existing literature, which demonstrates improved safety profiles of newer intravenous iron formulations compared to older dextran-based preparations.

The findings of this study are in agreement with previously published literature. Korczowski et al. demonstrated dose-dependent improvements in hemoglobin levels with ferric carboxymaltose, supporting the use of the 15 mg/kg dosing regimen. Similarly, Ozsahin et al. reported significant hematological response in pediatric patients who were unresponsive to oral iron therapy, while Powers et al. confirmed the safety and efficacy of ferric carboxymaltose in children and adolescents. The magnitude of hemoglobin improvement observed in the present study is comparable to these studies, further reinforcing the role of ferric carboxymaltose in pediatric anemia management.

An important consideration highlighted by this study is the limitation of oral iron therapy in pediatric patients. Poor palatability, gastrointestinal side effects, and the requirement for prolonged treatment significantly reduce compliance, leading to suboptimal outcomes. In contrast, ferric carboxymaltose enables rapid correction of iron deficiency with a single infusion, thereby improving adherence and reducing the burden on both patients and caregivers.

The selection of patients with moderate anemia allowed for evaluation of treatment efficacy in a clinically stable group without the confounding effects of blood transfusion, which is often required in severe anemia. This provides a clearer understanding of the therapeutic potential of ferric carboxymaltose in routine clinical practice.

Ferric carboxymaltose is a relatively recent addition to treatment options for pediatric iron deficiency anemia, with approval for use in children granted only in recent years. Consequently, there is limited real-world data on its use in pediatric populations, particularly in developing countries. The present study contributes to this growing body of evidence by providing real-world insights into its effectiveness and safety.

Despite its strengths, the study has certain limitations. It was conducted at a single center and did not include a comparison group receiving oral iron therapy. Additionally, the follow-up period was limited to 21 days, which may not reflect long-term outcomes such as complete normalization of iron stores and red cell indices. Further multicentric studies with longer follow-up and comparative design are warranted to validate these findings.

8. Conclusion

Intravenous ferric carboxymaltose appears to be a safe and effective option for achieving short-term hematological improvement in children with moderate iron deficiency anemia, with minimal adverse effects. A greater hemoglobin response was observed in older children. However, given the single-arm design and short follow-up period, further controlled studies with longer duration are warranted to confirm these findings.

References

[1] Givens DI, Anitha S, Giromini C. Anaemia in India and its prevalence and multifactorial aetiology: a narrative review. Nutrients. 2024;16(11):1673.
[2] Chandra J, Dewan P, Kumar P, Mahajan A, Singh P, Dhingra B, et al. Diagnosis, treatment and prevention of nutritional anemia in children: recommendations of the joint committee of PHO Chapter and PAN Society of IAP. Indian Pediatr. 2022;59(10):782–801.
[3] Subramaniam G, Girish M. Iron deficiency anemia in children. Indian J Pediatr. 2015;82(6):558–64.
[4] Kailas L, Umair CH, Sankar VH. An untold tale of iron deficiency anemia. Indian Pediatr. 2020;57(6):575–6.
[5] Korczowski B, Farrell C, Falone M, Blackman N, Rodgers T. Safety, pharmacokinetics, and pharmacodynamics of intravenous ferric carboxymaltose in children with iron deficiency anemia. Pediatr Res. 2023;94(4):1547–54.
[6] Carman N, Muir R, Lewindon P. Ferric carboxymaltose in the treatment of iron deficiency in pediatric inflammatory bowel disease. Transl Pediatr. 2019;8(1):28–35.
[7] Powers JM, Shamoun MP, McCavit TL, Adix L, Buchanan GR. Efficacy and safety of intravenous ferric carboxymaltose in children with iron deficiency anemia unresponsive to oral iron therapy. Blood. 2015;126(23):4552.
[8] Ozsahin H, Schaeppi M, Bernimoulin M, Allard M, Guidard C, van den Ouweland F. Intravenous ferric carboxymaltose for iron deficiency anemia or iron deficiency without anemia after poor response to oral iron treatment. Pediatr Blood Cancer. 2020;67(10): e28614.
[9] World Health Organization. Iron deficiency anaemia: assessment, prevention and control. Geneva: WHO; 2001.
[10] Baker RD, Greer FR. Diagnosis and prevention of iron deficiency and iron-deficiency anemia in infants and young children. Pediatrics. 2010;126(5):1040–50.
[11] Lopez A, Cacoub P, Macdougall IC, Peyrin-Biroulet L. Iron deficiency anemia. Lancet. 2016;387(10021):907–16.
[12] Camaschella C. Iron-deficiency anemia. N Engl J Med. 2015;372(19):1832–43.
[13] Kassebaum NJ. The global burden of anemia. Blood. 2016;128(17):113–21.
[14] Stoltzfus RJ. Iron deficiency: global prevalence and consequences. J Nutr. 2001;131(2):697S–701S.

[15] Auerbach M, Macdougall IC. Safety of intravenous iron formulations. Am J Hematol. 2014;89(1):31–38.
[16] Evstatiev R, Gasche C. Iron sensing and therapy in IBD. Gastroenterology. 2012;142(5):978–85.
[17] Onken JE, Bregman DB, Harrington RA, Morris D, Acs P, Akright B, et al. Ferric carboxymaltose in iron deficiency anemia. Am J Hematol. 2014;89(1):7–12.
[18] Van Wyck DB, Martens MG, Seid MH, Baker JB, Mangione A. Intravenous ferric carboxymaltose vs oral iron. Transfusion. 2007;47(12):201–10.
[19] Breymann C. Iron deficiency anemia in pregnancy and beyond. Hematology Am Soc Hematol Educ Program. 2015; 2015: 373–81.
[20] Powers JM, Buchanan GR. Diagnosis and management of iron deficiency anemia. Hematology Am Soc Hematol Educ Program. 2014; 2014: 210–16.
[21] Yates AM, O'Brien SH. Iron deficiency anemia in children. Pediatr Clin North Am. 2018;65(3):431–45.
[22] Pasricha SR, Tye-Din J, Muckenthaler MU, Swinkels DW. Diagnosis and management of iron deficiency. Lancet Haematol. 2021;8(7): e524–32.
[23] International Institute for Population Sciences (IIPS), ICF. National Family Health Survey (NFHS-5), India, 2019–21. Mumbai: IIPS; 2021.
[24] Ministry of Health and Family Welfare. Anemia Mukt Bharat: Intensified National Iron Plus Initiative. New Delhi: Government of India; 2018.
[25] Toteja GS, Singh P. Micronutrient deficiency disorders in India. Indian J Med Res. 2004;120(5):634–41.