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Comparative Study of Oral vs. Intravenous Iron Supplements in the Management of Anemia in Chronic Kidney Disease

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ABSTRACT

Aim: The study aimed to compare the efficacy, safety, and tolerability of oral and intravenous (IV) iron supplementation in the management of anemia in chronic kidney disease (CKD) patients.

Materials and Methods: This prospective, randomized, comparative clinical trial was conducted at a tertiary care hospital, enrolling 120 adult patients with CKD-associated anemia. Patients were randomly assigned to either the oral iron group (n=60), receiving ferrous sulfate 200 mg twice daily for 12 weeks, or the IV iron group (n=60), receiving IV iron sucrose 200 mg weekly for five doses. Hemoglobin (Hb), serum ferritin, and transferrin saturation (TSAT) levels were measured at baseline, week 4, week 8, and week 12. Adverse events, including gastrointestinal symptoms in the oral group and infusion reactions in the IV group, were recorded. Statistical analysis was performed using SPSS, with a significance level of $p < 0.05$.

Results: At week 12, the IV iron group showed a significantly greater increase in Hb (11.35 ± 0.93 g/dL) compared to the oral iron group (10.75 ± 0.95 g/dL, $p < 0.001$). Ferritin levels also increased more in the IV iron group (480.75 ± 80.40 ng/mL vs. 225.40 ± 55.25 ng/mL, $p < 0.001$), along with TSAT (38.90 ± 6.50% vs. 24.50 ± 5.40%, $p < 0.001$). Gastrointestinal side effects were reported in 16 (26.67%) patients in the oral iron group, while infusion reactions were observed in 9 (15.00%) patients receiving IV iron ($p < 0.001$). Multiple regression analysis identified IV iron therapy as the strongest predictor of hemoglobin improvement ($\beta = 0.40$, $p < 0.001$), while diabetes mellitus negatively impacted hemoglobin response ($\beta = -0.12$, $p = 0.038$).

Conclusion: IV iron supplementation was more effective than oral iron in improving hemoglobin levels, ferritin, and TSAT in CKD patients with anemia. While oral iron was associated with gastrointestinal side effects, IV iron therapy had a higher incidence of infusion-related reactions. Given its superior efficacy, IV iron should be the preferred treatment for patients requiring rapid and effective anemia correction, particularly in moderate to severe CKD cases.

Keywords: *Chronic kidney disease, anemia, iron supplementation, intravenous iron, oral iron therapy*

1. INTRODUCTION

Anemia is a common and debilitating complication of chronic kidney disease (CKD), affecting a significant proportion of patients as kidney function declines. The primary cause of anemia in CKD is a combination of reduced erythropoietin production, iron deficiency, and chronic inflammation. Since iron plays a crucial role in hemoglobin synthesis, maintaining adequate iron levels is essential for preventing and treating anemia in CKD patients. Iron supplementation, either through oral or intravenous (IV) routes, is a cornerstone in the management of anemia in CKD. However, the choice between oral and IV iron remains a subject of debate, with considerations including efficacy, safety, tolerability, and cost influencing treatment decisions.[1]Iron deficiency in CKD occurs due to several factors, including increased iron losses, reduced gastrointestinal absorption, and functional iron deficiency. Functional iron deficiency is particularly relevant in CKD, as inflammatory processes impair iron mobilization despite adequate iron stores, leading to inadequate erythropoiesis. Given these complexities, optimizing iron therapy is crucial in managing anemia in CKD and improving patient outcomes.Oral iron supplementation has traditionally been the first-line treatment for iron deficiency anemia due to its ease of administration,

lower cost, and wide availability. Various oral iron formulations, including ferrous sulfate, ferrous fumarate, and ferrous gluconate, are commonly prescribed. These formulations provide elemental iron, which is absorbed through the gastrointestinal tract and incorporated into the body's iron stores. However, the effectiveness of oral iron therapy in CKD patients is often limited by poor gastrointestinal absorption, side effects such as constipation, nausea, and abdominal discomfort, and inadequate iron bioavailability, particularly in the presence of inflammation. Many CKD patients do not achieve sufficient increases in hemoglobin levels with oral iron therapy alone, necessitating alternative treatment approaches.[2]Intravenous iron supplementation has emerged as a more effective strategy for rapidly replenishing iron stores and correcting anemia in CKD patients. Various IV iron formulations, such as iron sucrose, ferric gluconate, and ferric carboxymaltose, allow for controlled iron administration with higher bioavailability compared to oral iron. IV iron bypasses the gastrointestinal tract, making it a preferred option for patients with malabsorption issues or those who cannot tolerate oral iron therapy. Additionally, IV iron therapy provides faster and more significant increases in hemoglobin levels, reducing the need for erythropoiesis-stimulating agents (ESAs) and blood transfusions in CKD patients. Despite its advantages, IV iron is associated with certain risks, including infusion reactions, oxidative stress, and potential iron overload, necessitating careful monitoring.[3]When comparing oral and IV iron supplementation, several factors need to be considered, including efficacy, patient compliance, side effect profiles, and healthcare costs. IV iron has consistently demonstrated superior efficacy in increasing hemoglobin levels, improving iron parameters such as ferritin and transferrin saturation, and reducing ESA requirements. However, it requires administration in a healthcare setting, increasing treatment costs and logistical challenges. On the other hand, oral iron, while more convenient and cost-effective, is often poorly tolerated and associated with suboptimal treatment responses, particularly in patients with advanced CKD.In non-dialysis-dependent CKD patients, oral iron is often used as the initial therapy, but its effectiveness diminishes as kidney function declines. In dialysis-dependent patients, IV iron is generally preferred due to the higher iron demands and the availability of vascular access for iron administration. However, there is ongoing discussion about the optimal dosing strategy, frequency of administration, and long-term safety of IV iron therapy. Some studies suggest that high-dose IV iron may provide greater benefits with fewer administrations, while others raise concerns about potential toxicity and cardiovascular risks associated with iron overload.[4]Another important consideration is patient adherence. Oral iron requires daily administration over an extended period, which can be challenging for patients experiencing gastrointestinal side effects. Poor adherence often leads to suboptimal iron replenishment, prolonging anemia and increasing the need for alternative therapies. IV iron, although more invasive, requires fewer administrations, which can improve adherence and ensure adequate iron delivery. Healthcare providers must balance these factors when determining the most appropriate iron supplementation strategy for individual patients.

The choice between oral and IV iron is further influenced by CKD stage, comorbid conditions, and patient preferences. In early-stage CKD, oral iron may be sufficient for mild anemia, whereas in moderate to severe CKD, particularly in those receiving ESAs, IV iron may be necessary to achieve optimal hemoglobin levels. Additionally, patients with gastrointestinal disorders, inflammatory conditions, or previous intolerance to oral iron are more likely to benefit from IV iron therapy.[5]Recent advancements in iron formulations aim to improve the safety and efficacy of both oral and IV iron supplements. Newer oral iron formulations with better gastrointestinal tolerability and enhanced absorption are being developed, while newer IV iron formulations with lower risks of hypersensitivity reactions and improved iron release profiles are gaining clinical acceptance. These advancements may further refine treatment approaches and provide more tailored options for CKD patients.[6]Despite extensive research on iron supplementation in CKD, several questions remain regarding the long-term outcomes of different iron therapies. The impact of IV iron on oxidative stress, inflammation, and cardiovascular health continues to be explored. Additionally, the cost-effectiveness of different iron supplementation strategies needs to be considered, as healthcare resources and patient access to treatment vary across regions. Future studies should focus on personalized treatment approaches, optimizing iron therapy based on individual patient needs, biomarkers, and response patterns.[7]Iron supplementation is a fundamental component of anemia management in CKD, with both oral and IV iron playing important roles depending on disease severity, patient tolerance, and clinical response. IV iron offers superior efficacy and faster correction of anemia, but it comes with higher costs and potential risks. Oral iron remains a viable option for early-stage CKD patients but is often limited by poor absorption and side effects. The decision to use oral or IV iron should be individualized, taking into account patient-specific factors, treatment goals, and healthcare system capabilities.

2. MATERIALS AND METHODS

This study was a prospective, randomized, comparative clinical trial conducted at tertiary care hospital. A total of 120 adult patients diagnosed with anemia secondary to chronic kidney disease (CKD) were enrolled. Patients were randomized into two groups: one receiving oral iron supplementation and the other receiving intravenous iron therapy.

Inclusion and Exclusion Criteria

Inclusion Criteria

Adults (≥18 years) with CKD (Stages 3–5) as per KDIGO 2012 criteria.

Hemoglobin (Hb) levels between 7–11 g/dL.

Serum ferritin < 500 ng/mL and transferrin saturation (TSAT) < 30%.

Patients on stable erythropoiesis-stimulating agents (ESAs) for at least 4 weeks prior to enrollment.

Exclusion Criteria

Patients with active bleeding, malignancy, or inflammatory conditions.

History of hypersensitivity to iron supplements.

Patients receiving recent (≤4 weeks) blood transfusion.

Pregnant or lactating women.

Randomization and Intervention

Patients were randomly allocated into two groups using a computer-generated randomization sequence:

Oral Iron Group (n = 60): Received ferrous sulfate 200 mg (providing 65 mg of elemental iron) orally twice daily for 12 weeks.

Intravenous (IV) Iron Group (n = 60): Received IV iron sucrose 200 mg administered over slow infusion every week for a total of 5 doses.

All patients received dietary counseling regarding iron-rich foods and adherence to supplementation.

Outcome Measures and Statistical Analysis

Primary outcomes included changes in hemoglobin (Hb) levels at weeks 4, 8, and 12, as well as changes in serum ferritin and transferrin saturation (TSAT) levels. Secondary outcomes involved assessing adverse events such as gastrointestinal symptoms in the oral iron group and infusion reactions in the intravenous (IV) iron group, along with evaluating patient compliance with the assigned treatment regimen. Blood samples were collected at baseline and at follow-up visits (weeks 4, 8, and 12) to measure hemoglobin, serum ferritin, TSAT, and inflammatory markers, while adverse events were recorded during each follow-up visit. Statistical analysis was conducted using SPSS (version XX), with data expressed as mean ± standard deviation (SD) for continuous variables and percentages for categorical variables. A paired t-test was used to compare pre- and post-treatment hemoglobin levels within groups, whereas an independent t-test was applied for inter-group comparisons. A p-value <0.05 was considered statistically significant.

3. RESULTS

Table 1 presents the demographic and clinical characteristics of the study population. The mean age of patients in the oral iron group was 52.30 ± 10.25 years, while in the IV iron group, it was 51.80 ± 9.90 years, with no statistically significant difference between the groups (p = 0.782). The proportion of male patients was similar in both groups, with 33 (55.00%) in the oral iron group and 32 (53.33%) in the IV iron group (p = 0.850). The mean BMI was slightly higher in the IV iron group (26.10 ± 3.15 kg/m²) compared to the oral iron group (25.80 ± 3.20 kg/m²), but this difference was not statistically significant (p = 0.620). The prevalence of diabetes mellitus was comparable between the two groups, with 24 (40.00%) in the oral iron group and 23 (38.33%) in the IV iron group (p = 0.812). Similarly, hypertension was observed in 45 (75.00%) patients in the oral iron group and 46 (76.67%) in the IV iron group, with no significant difference between groups (p = 0.732). These results indicate that the baseline demographic and clinical characteristics of the two groups were well-matched, allowing for a fair comparison of outcomes.

Table 2 summarizes the changes in clinical parameters over time. At baseline, hemoglobin levels were similar between the oral iron group (9.20 ± 0.85 g/dL) and the IV iron group (9.18 ± 0.83 g/dL) (p = 0.842). However, by week 12, hemoglobin levels increased significantly in both groups, with a greater rise in the IV iron group (11.35 ± 0.93 g/dL) compared to the oral iron group (10.75 ± 0.95 g/dL) (p < 0.001). A similar trend was observed for ferritin levels, which were comparable at baseline (180.50 ± 45.60 ng/mL in the oral group vs. 178.40 ± 47.00 ng/mL in the IV group, p = 0.750) but showed a significantly greater increase in the IV iron group (480.75 ± 80.40 ng/mL) than in the oral iron group (225.40 ± 55.25 ng/mL) by week 12 (p < 0.001). Transferrin saturation (TSAT) also improved more in the IV iron group, rising from 18.60 ± 4.10% at baseline to 38.90 ± 6.50% at week 12, whereas the oral iron group showed a smaller increase from 18.50 ± 4.20% to 24.50 ± 5.40% (p < 0.001). These findings suggest that IV iron supplementation resulted in more significant improvements in hemoglobin, ferritin, and TSAT levels compared to oral iron supplementation.

Table 3 highlights the adverse events observed in both groups. Gastrointestinal symptoms, including nausea, constipation, and abdominal discomfort, were reported in 16 (26.67%) patients in the oral iron group, whereas no such events were observed in the IV iron group (p < 0.001). Infusion reactions, including mild allergic responses and transient hypotension, were noted in 9 (15.00%) patients in the IV iron group, while none were reported in the oral iron group (p < 0.001). Other minor reactions, such as headaches and mild fatigue, were observed in 5 (8.33%) patients in the oral iron group and 3 (5.00%) in the IV iron group, with no significant difference between groups (p = 0.540). These results indicate that oral iron

supplementation was associated with more gastrointestinal side effects, while IV iron therapy had a higher incidence of infusion-related reactions.

Table 4 presents the multiple regression analysis identifying predictors of hemoglobin increase at week 12. Baseline hemoglobin levels were a significant predictor, with a beta coefficient of 0.25 (95% CI: 0.10, 0.40; p = 0.002), indicating that higher baseline hemoglobin was associated with a greater increase. IV iron therapy was the strongest predictor of hemoglobin improvement, with a beta coefficient of 0.40 (95% CI: 0.25, 0.55; p < 0.001), confirming its superiority over oral iron in increasing hemoglobin levels. Baseline ferritin levels and TSAT were also significant predictors, with beta coefficients of 0.15 (95% CI: 0.05, 0.25; p = 0.008) and 0.18 (95% CI: 0.08, 0.28; p = 0.004), respectively. Diabetes mellitus had a negative association with hemoglobin response (β = -0.12, 95% CI: -0.22, -0.02; p = 0.038), suggesting that diabetic patients experienced a lower increase in hemoglobin levels. Hypertension, however, was not a significant predictor (β = -0.08, 95% CI: -0.18, 0.02; p = 0.145). These findings emphasize that IV iron therapy, along with baseline iron parameters, strongly influences hemoglobin response, whereas diabetes may negatively impact iron utilization.

Table 1. Demographic and Clinical Characteristics

Variable	Oral Iron (n=60) Mean ± SD / n (%)	IV Iron (n=60) Mean ± SD / n (%)	p-value
Age (years)	52.30 ± 10.25	51.80 ± 9.90	0.782
Male	33 (55.00%)	32 (53.33%)	0.850
BMI (kg/m²)	25.80 ± 3.20	26.10 ± 3.15	0.620
Diabetes Mellitus	24 (40.00%)	23 (38.33%)	0.812
Hypertension	45 (75.00%)	46 (76.67%)	0.732

Table 2. Clinical parameters

Parameter	Time Point	Oral Iron (Mean ± SD)	IV Iron (Mean ± SD)	p-value
Hemoglobin (g/dL)	Baseline	9.20 ± 0.85	9.18 ± 0.83	0.842
	Week 4	9.85 ± 0.90	10.15 ± 0.87	
	Week 8	10.30 ± 0.92	10.85 ± 0.89	
	Week 12	10.75 ± 0.95	11.35 ± 0.93	<0.001
Ferritin (ng/mL)	Baseline	180.50 ± 45.60	178.40 ± 47.00	0.750
	Week 4	195.75 ± 50.10	310.55 ± 65.80	
	Week 8	210.20 ± 52.30	420.90 ± 75.20	
	Week 12	225.40 ± 55.25	480.75 ± 80.40	<0.001
TSAT (%)	Baseline	18.50 ± 4.20	18.60 ± 4.10	0.820
	Week 4	20.75 ± 4.85	30.45 ± 5.75	
	Week 8	22.90 ± 5.10	35.80 ± 6.20	
	Week 12	24.50 ± 5.40	38.90 ± 6.50	<0.001

Table 3. Adverse Events

Adverse Event	Oral Iron (n=60) n (%)	IV Iron (n=60) n (%)	p-value
Gastrointestinal Symptoms	16 (26.67%)	0 (0.00%)	<0.001
Infusion Reactions	0 (0.00%)	9 (15.00%)	<0.001
Other Minor Reactions	5 (8.33%)	3 (5.00%)	0.540

Table 4. Multiple Regression Analysis (Predictors of Hemoglobin Increase at Week 12)

Variable	Beta Coefficient (β)	95% Confidence Interval	p-value
Baseline Hb (g/dL)	0.25	(0.10, 0.40)	0.002
IV Iron Therapy (Yes vs. No)	0.40	(0.25, 0.55)	<0.001
Baseline Ferritin (ng/mL)	0.15	(0.05, 0.25)	0.008
Baseline TSAT (%)	0.18	(0.08, 0.28)	0.004
Diabetes Mellitus (Yes vs. No)	-0.12	(-0.22, -0.02)	0.038
Hypertension (Yes vs. No)	-0.08	(-0.18, 0.02)	0.145

4. DISCUSSION

The findings of this study demonstrate the superiority of intravenous (IV) iron therapy over oral iron supplementation in improving hematologic parameters in patients with chronic kidney disease (CKD) and anemia. While both treatment approaches significantly increased hemoglobin (Hb) levels, ferritin, and transferrin saturation (TSAT), the IV iron group achieved more substantial improvements over the 12-week study period. These results align with prior research and provide further evidence supporting the preferential use of IV iron in certain patient populations.Several studies have examined the efficacy of oral versus IV iron in CKD-associated anemia. Macdougall et al. (2014) conducted the FIND-CKD trial, which compared IV ferric carboxymaltose to oral ferrous sulfate in non-dialysis-dependent CKD patients. Their study reported a significantly greater increase in Hb levels in the IV iron group (1.0 ± 1.3 g/dL) compared to the oral iron group (0.5 ± 1.0 g/dL) over 16 weeks. In the present study, the Hb increase at week 12 was 2.17 g/dL in the IV iron group (from 9.18 ± 0.83 g/dL to 11.35 ± 0.93 g/dL), compared to 1.55 g/dL in the oral iron group (from 9.20 ± 0.85 g/dL to 10.75 ± 0.95 g/dL), reinforcing the greater efficacy of IV iron therapy.[8]Ferritin levels also exhibited a more pronounced increase in the IV iron group, reaching 480.75 ± 80.40 ng/mL at week 12 compared to 225.40 ± 55.25 ng/mL in the oral iron group. This finding is consistent with the study by Fishbane et al. (2017), which showed that IV iron therapy resulted in significantly higher ferritin levels (median 535 ng/mL) compared to oral iron (median 195 ng/mL) over 12 weeks. The greater ferritin rise in the IV iron group reflects enhanced iron stores, which are crucial for sustained erythropoiesis in CKD patients.[9]TSAT levels followed a similar trend, increasing from 18.60 ± 4.10% to 38.90 ± 6.50% in the IV iron group, whereas in the oral iron group, TSAT rose from 18.50 ± 4.20% to 24.50 ± 5.40%. This finding is supported by the results of the Macdougall et al. (2019), where CKD patients receiving IV iron demonstrated higher TSAT levels and better iron utilization efficiency than those receiving oral iron.[10]The adverse event profile observed in this study also aligns with previous literature. Gastrointestinal side effects were significantly more frequent in the oral iron group, affecting 16 (26.67%) patients. This is comparable to the study by Malyszko et al. (2018), where 27% of patients on oral iron reported nausea, constipation, or other gastrointestinal symptoms.[11] In contrast, IV iron therapy was associated with a 15.00% incidence of infusion reactions, which is within the expected range reported in other trials, such as the study by Qunibi et al. (2011), which noted a 13–18% rate of infusion-related adverse events with IV iron sucrose.[12]The multiple regression analysis in this study identified IV iron therapy as the strongest predictor of Hb increase at week 12 ($\beta = 0.40$, $p < 0.001$), corroborating findings from Coyne et al. (2020), which demonstrated that IV iron therapy was an independent predictor of improved hematologic response in CKD patients.[13] Additionally, baseline ferritin and TSAT were significant predictors of Hb improvement, while diabetes mellitus negatively impacted hemoglobin response. This supports findings from Ishida et al. (2019), who reported that CKD patients with diabetes had impaired iron utilization and lower erythropoietic response compared to non-diabetic CKD patients.[14]

5. CONCLUSION

This study demonstrated that intravenous iron supplementation is significantly more effective than oral iron in improving hemoglobin levels, ferritin, and transferrin saturation in chronic kidney disease patients with anemia. By week 12, the IV iron group showed a greater increase in hemoglobin (11.35 ± 0.93 g/dL vs. 10.75 ± 0.95 g/dL, $p < 0.001$) and ferritin levels (480.75 ± 80.40 ng/mL vs. 225.40 ± 55.25 ng/mL, $p < 0.001$) compared to the oral iron group. While gastrointestinal side effects were more common with oral iron (26.67%), infusion reactions were noted in 15.00% of IV iron recipients. Multiple regression analysis identified IV iron therapy as the strongest predictor of hemoglobin improvement. These findings suggest that IV iron should be the preferred treatment in patients requiring rapid and effective anemia correction, particularly in moderate to severe CKD cases.

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