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Original article

Evaluation of the short and long-term effects of ferric carboxymaltose on phosphorus and parathyroid hormone levels in patients with CKD

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Abstract

Context: Ferric carboxymaltose (FCM) is a widely used therapy in chronic kidney disease (CKD). With its increasing use, hypophosphatemia -an unexpected side effect- has drawn growing attention. While the risk of hypophosphatemia decreases as the chronic kidney disease stage progresses, the impact of ferric carboxymaltose on parathormone levels and how this risk evolves with repeated doses remain less known.

Objective: In our study, we planned to evaluate the relationship of ferric carboxymaltose with phosphorus and parathormone in the long term in patients with stage 2-5 non-dialysis chronic kidney disease.

Method: All chronic kidney disease patients who received ferric carboxymaltose treatment between January 2022 and March 2023 were screened. Basal phosphorus values of the patients and phosphorus values in the first and second controls were recorded as F0, F1, and F2, respectively. Parathormone values were recorded in the first control after the second dose of ferric carboxymaltose.

Keywords: Ferric carboxymaltose, IV iron, Phosphorus, Parathormone, Chronic kidney disease.

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Results: A total of 49 patients met the criteria, of whom 31 patients were female (63 %). Thirty-two patients (65 %) received a single dose of treatment. The mean age was 67±12 years, with a mean dose of 969±483 mg. The mean GFR was 36±14 mL/min/1,73 m^2
When comparing single- and double-dose groups, no significant differences were observed in age, gender distribution, chronic kidney disease stage, baseline phosphorus levels (F0), or the time to the first follow-up.
Between F0 and F1, there was a significant decrease of 0.2 and 0.3 mg/dL in phosphorus levels in both groups, respectively. No patient developed clinically apparent hypophosphatemia. No significant difference was found when the F0-F2 and F1-F2 values were compared in patients who received two treatment doses. Parathormone levels increased slightly in the same patient group, although not statistically significant.
Conclusion: While ferric carboxymaltose causes a mild phosphorus reduction at high doses in chronic kidney disease patients not receiving dialysis treatment, this effect is not permanent. Prospective studies with sufficient patients are needed to elucidate the impact of fluctuations in phosphorus levels caused by this treatment on parathormone and chronic kidney disease -related bone disease.

Evaluación de los efectos a corto y largo plazo de la carboximaltosa férrica sobre los niveles de fósforo y hormona paratiroidea en pacientes con enfermedad renal crónica

Resumen

Contexto: la carboximaltosa férrica (FCM) es un tratamiento frecuentemente empleado en la enfermedad renal crónica (ERC). Con el incremento en la frecuencia de su uso, la hipofosfatemia, un efecto secundario inesperado, ha comenzado a captar atención. Si bien este riesgo disminuye conforme avanza la etapa de la enfermedad renal crónica, el impacto de la carboximaltosa férrica sobre la paratohormona y la variación de este riesgo con dosis repetidas son menos conocidos.
Objetivo: en nuestro estudio, planeamos evaluar la relación a largo plazo de la carboximaltosa férrica con el fósforo y la paratohormona en pacientes con enfermedad renal crónica de etapa 2-5 (sin diálisis).
Método: todos los pacientes con enfermedad renal crónica que recibieron tratamiento con carboximaltosa férrica entre enero de 2022 y marzo de 2023 fueron evaluados. Se registraron los valores basales de fósforo (F0) y los valores de fósforo en la primera y segunda revisión (F1 y F2, respectivamente). Los valores de paratohormona se registraron en la primera revisión después de la segunda dosis de carboximaltosa férrica.
Resultados: un total de 49 pacientes cumplieron con los criterios, de los cuales 31 (63 %) eran mujeres. Treinta y dos pacientes (65 %) recibieron una única dosis del tratamiento y la edad promedio fue de 67±12 años, mientras que la dosis media fue de 969±483 mg. El GFR medio fue de 36±14 mL/min/1,73 m^2. Al comparar pacientes que recibieron una o dos dosis, no se observaron diferencias significativas en la edad, el género, las etapas de la enfermedad renal crónica, los valores de F0 ni el tiempo transcurrido hasta la primera visita. Entre F0 y F1 se observó una disminución significativa de 0.2 y 0.3 mg/dL en los niveles de fósforo en ambos grupos, respectivamente. Ningún paciente desarrolló hipofosfatemia clínicamente aparente. No se encontraron diferencias significativas al comparar los valores de F0-F2 y F1-F2 en los pacientes que recibieron dos dosis de tratamiento. Los niveles de paratohormona incrementaron ligeramente en el mismo grupo de pacientes, aunque la variación no fue estadísticamente significativa.

Conclusión: aunque la carboximaltosa férrica causa una reducción leve de fósforo en dosis altas en pacientes con enfermedad renal crónica que no reciben tratamiento de diálisis, este efecto no es permanente. Se requieren estudios prospectivos con suficientes pacientes para esclarecer el impacto de las fluctuaciones en los niveles de fósforo provocadas por este tratamiento sobre la paratohormona y la enfermedad ósea relacionada con la enfermedad renal crónica.

Palabras clave: carboximaltosa férrica, hierro intravenoso, fósforo, parathormona, enfermedad renal crónica.

Introduction

Chronic kidney disease (CKD) is a prevalent and complex condition characterized by the progressive loss of kidney function over time [1]. It affects millions of people worldwide and is associated with numerous complications, including anemia [2]. Iron deficiency anemia (IDA) is a common complication in CKD patients, resulting from both an absolute iron deficiency and impaired iron utilization. Intravenous iron supplementation has become an integral part of therapy to address IDA in CKD [3]. Ferric carboxymaltose (FCM) has emerged as a widely used formulation due to its convenient administration and favorable safety profile for CKD patients and all those with IDA [4–6]. FCM allows administering a hefty dose of elemental iron (up to 1000 mg) in a single infusion, significantly reducing the frequency of iron therapy and improving patient compliance [7].

While FCM has greatly simplified iron replacement in the outpatient setting, its growing use has brought attention to certain rare side effects. One particular concern is hypophosphatemia. This side effect has been known for more than 40 years [8]. However, FCM is associated with a higher incidence of hypophosphatemia than any other IV iron product [7, 9].

The underlying mechanism of FCM-induced hypophosphatemia is mediated by fibroblast growth factor-23 (FGF-23) pathway. FCM causes an abrupt increase in FGF-23 levels, thereby causing a phosphaturic state and reducing serum phosphorus levels [10, 11]. The clinical significance of this side effect and its long-term consequences are areas of active investigation.

Several risk factors for FCM-induced hypophosphatemia have been recognized, which include younger age, more profound anemia (i.e., Hb<7 gr/dL), higher doses of FCM, and preserved renal function [12]. While it is well-known that a GFR<60 ml/min protects against FCM-induced hypophosphatemia, and anuric patients are unaffected by this side effect, more real-life data is needed for non-dialysis dependent CKD. Available data at hand also suggests safety of FCM in CKD cohort [13]. Additionally, a prospective study found that a single dose of FCM does not impact parathormone (PTH) levels [14]. However, the effect of repeated doses of FCM on PTH levels has yet to be well-known.

Our study aimed to evaluate FCM's effect on phosphorus levels on single or double infusions and its impact on PTH in subjects receiving double FCM doses.

Materials and methods

This retrospective study aimed to evaluate the short and long-term effects of FCM on phosphorus and parathormone levels in patients with CKD.

The study population consisted of CKD patients who received FCM treatment between January 2022 and March 2023 at a single center. Medical records were screened to identify eligible patients for inclusion in the study. Patients with insufficient data and those with glomerular filtration rates greater than 90 ml/min were excluded to ensure a homogenous CKD population. Patients with other microelement/vitamin deficiencies (B12, folate, and copper) and those needing erythropoietin were also excluded.

For data collection, a standardized process was followed, recording demographic characteristics such as age and gender. Baseline hemoglobin (Hb), creatinine, estimated glomerular filtration rate (eGFR) (calculated using the CKD-EPI 2009 formula), and respective CKD stages were also documented.

Baseline phosphorus values (F0) of the patients were documented, along with phosphorus values at the first (F1) and second (F2) follow-up visits. Baseline parathormone levels (PTH0) were recorded. PTH1 was measured at the first follow-up visit after the second dose of FCM treatment. F2 and PTH1 were collected only for the patients who received two doses of FCM. Days between the infusion and follow-up visits were also calculated. See Figure 1 for a visual representation.

In addition to phosphorus and parathormone data, information regarding FCM treatment was collected. The number of FCM doses administered to each patient and the total dosage received were recorded. Supplementary treatments such as vitamin D supplementation, analog usage, phosphorus binders, or calcimimetics were also documented.

The statistical analysis was performed using Statistical Package for the Social Sciences version 20.0 (IBM). Descriptive statistics were used to summarize the demographic characteristics of the study population, including mean values and standard deviations for continuous variables, as well as frequencies and percentages for categorical variables.

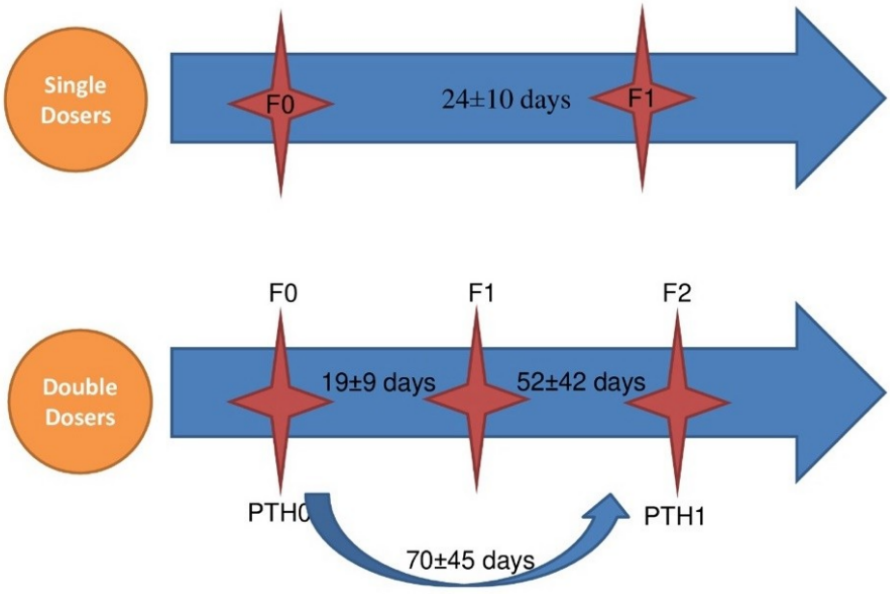

Figure 1. Timelines of the sudy

Source: Author's elaboration.

To assess the changes in phosphorus levels between different time points (F0, F1, and F2), paired t-tests or non-parametric tests (such as the Wilcoxon signed-rank test) were employed, depending on the data distribution. Subgroup analyses were conducted to compare phosphorus and parathormone levels between patients who received a single dose of FCM and those who received two doses.

The significance level for all statistical tests was set at $p<0.05$. The results were reported as means ± standard deviations or medians with interquartile ranges, as appropriate.

Due to the study's exploratory nature and the absence of prior data on the specific research question, a formal sample size calculation was not performed. However, efforts were made to include as many eligible patients as possible within the study period to enhance the representativeness and reliability of the findings.

Results

A total of 49 patients met the criteria, of whom 31 patients were female (63 %). Thirty-two patients (65 %) received a single dose of treatment and the mean age was 67±12, while the mean dose was 969±483 mg. The mean GFR was 36±14.

When single and double-dose patients were compared, age, gender, body mass index, creatinine, eGFR, CKD stages, F0 values, and the time to first visit were similar. Double-dosed patients had lower Hb values and received higher doses of FCM. Furthermore, their first FCM doses were higher than single-dosed patients (Table 1).

Table 1. Baseline characteristics and comparisons of two patient groups

Criteria	All patients n=49	Single dose n=32	Double dose n=17	p value
Male/female	18/31	10/22	8/9	0,275
Age	67±12	65±13	71±9	0,067
CKD stage				
-2	3	2	1	
-3	24	15	9	0,904
-4	17	11	6	
-5	5	4	1	
BMI	26,3±4,8	25,8±4,5	27,2±5,3	0,328
Total dose of FCM (mg)	969±483	687±245	1500±353	<0,001
First dose of FCM (mg)	755±252	687±245	882±218	0,007
Days between F0-F1	22±10	24±10	19±9	0,875
Creatinine (mg/dL)	2,25±1,6	2,47±1,91	1,8±0,5	0,210
GFR (ml/min, CKD-EPI)	34±16	32±16	34±13	0,598
Baseline Hb (gr/dL)	10,4±1,0	10,7±1,0	9,7±0,8	<0,001

Source: Author's elaboration.

Between F0 and F1, there was a significant decrease of 0.2 and 0.3 mg/dL in phosphorus levels in both groups, respectively (Table 2). No patient developed clinically apparent hypophosphatemia.

Table 2. F0 and F1 values of the cohort

Criteria	F0	F1	p value
All patients n=49	4,1±0,6	3,9±0,8	0,004
Single dose n=32	4,1±0,7	3,9±0,9	0,043
Double dose n=17	4,2±0,5	3,9±0,7	0,030

Source: Author's elaboration.

A subgroup analysis of patients who received two doses of FCM revealed no significant difference between F0-F2 and F1-F2 values. Parathormone levels (PTH0 vs. PTH1) increased slightly in the same patient group, although not statistically significant (Table 3).

Table 3. Subgroup comparisons of patients who received two doses (n=17)

F0	**F2**	**p value**
4,2±0,5	4,1±0,6	0,649
F1	**F2**	**p value**
3,9±0,7	4,1±0,6	0,127
PTH0	**PTH1**	**p value**
105±52	118±64	0,123

Source: Author's elaboration.

Discussion

The present study aimed to evaluate the short and long-term effects of FCM on phosphorus levels and parathormone in patients with CKD. The findings provide valuable insights into the implications of FCM administration and its potential risks, particularly the development of hypophosphatemia in non-dialysis-dependent CKD.

The results of our study demonstrated a mild but significant decrease in phosphorus levels between baseline (F0) and the first follow-up visit (F1), in both the single and double-dose groups. This finding aligns with previous studies that have reported FCM-induced hypophosphatemia in CKD patients [10, 15, 16]. However, some of the trials, especially those in patients with heart failure, did not find a significant decrease in phosphorus levels; this may be caused by the relatively low doses used in these trials [17].

Şumnu and Erdem conducted a similar study to ours; however, they did not observe a significant decline in phosphorus [18]. Their cohort had a mean GFR of 22 ml/min against our 34 ml/min. We believe this difference explains the discrepancies between the two studies and highlights the protective effect of CKD from IV FCM-induced hypophosphatemia. Similar results have been obtained by Merino and collegues, reassuring the preventative effect of low GFR from IV FCM-induced hypophopshetmia [13]. The same group also effectively demonstrated the safety of FCM in peritoneal dialysis patients [19,20]. Studies in hemodialysis patients similarly suggest a favorable safety profile [21].

Notably, despite the observed decrease in phosphorus levels, no patients in our study developed clinically significant hypophosphatemia. This absence suggests that FCM administration, even at high doses, is generally well-tolerated in CKD patients.

Interestingly, we did not find a significant difference in phosphorus levels between the first (F1) and second follow-up visit (F2) in patients who received two doses of FCM treatment. This may indicate that repeated doses of FCM do not exacerbate the decline in phosphorus levels or further contribute to hypophosphatemia. These results suggest that FCM-induced hypophosphatemia is transient and reversible, underscoring the importance of monitoring phosphorus levels during FCM therapy for patients with CKD that are not hypophosphatemic.

Regarding parathormone levels, a slight increase was observed in the double-dose group, although it did not reach statistical significance. This finding suggests that FCM administration may have some influence on parathormone regulation. Prats *et al.*, in their subgroup analysis of repeat dose IV iron, did not come across a change in PTH levels [14]. On the contrary, studies on patient populations with normal renal function reveal an increase in PTH levels, especially with repeated doses [22, 23]. This might explain why prolonged IV iron has been linked to osteomalacia over time [24]. It is also important to note that as the use of FCM becomes more widespread, new side effects -such as kidney stone formation in susceptible individuals with a GFR above 60 ml/min- are being reported more frequently. This highlights the need for increased awareness among non-nephrologists regarding these potential side effects [25].

The findings of this study have some implications for managing CKD patients receiving FCM treatment. While FCM administration may lead to a transient reduction in phosphorus levels, the absence of clinically significant hypophosphatemia suggests that iron supplementation with FCM outweighs the potential risks. However, it is crucial to identify patients who may be more susceptible to hypophosphatemia, particularly those with pre-existing hypophosphatemia, severe anemia, or higher eGFR. Monitoring phosphorus levels during FCM therapy and implementing appropriate interventions, such as phosphorus supplementation or dose adjustment, may help mitigate the risk of hypophosphatemia in susceptible individuals.

It is important to note that this study had some limitations. First, the retrospective design and relatively small sample size may have influenced the statistical power and generalizability of the results. Additionally, the study focused on non-dialysis CKD patients, and the findings cannot directly apply to dialysis patients.

Despite these limitations, this study provides valuable insights into the short and long-term effects of FCM on phosphorus levels and parathormone in CKD patients. The findings contribute to understanding the risks and implications of FCM administration, facilitating evidence-based decision-making in managing CKD-related iron deficiency anemia.

Prospective studies with larger patient cohorts aiming for more hard outcomes are needed to provide more comprehensive insights into the impact of FCM-induced phosphorus fluctuations on parathormone regulation and CKD-related bone disease.

Conclusion

FCM administration in CKD patients leads to a transient reduction in phosphorus levels without clinically significant hypophosphatemia. The observed probable slight increase in parathormone levels warrants further investigation to understand its clinical significance. Monitoring phosphorus levels during FCM therapy and individualizing treatment strategies based on patients' baseline phosphorus status and CKD severity are crucial to optimize iron supplementation and minimize the potential risks associated with FCM-induced hypophosphatemia.

Ethical considerations

The study protocol was approved by the local ethical boards (05.09.2023, E-77082166-604.01.02-813551). Patient consent was not required per our centers protocol, as this study is retrospective in nature.

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Conflicts of interest

The authors have no conflict of interest.

Authors contribution

All authors participated with substantial contributions in the conception or design of the work; in the collection, analysis, and interpretation of data; in the writing of the article or in its critical review and, in the final approval of the version to be published.

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