Early Efficacy and Hypophosphatemia After Ferric Carboxymaltose Infusion Therapy in Patients With Iron Deficiency Anemia: A Single-Center Experience

Hana Matijaca[1], Klara Brčić[1], Klara Grgorinić[2], Ivan Krečak[3,4] and Petar Gaćina[1,5]

[1] Department of Hematology, Sestre milosrdnice University Hospital Center, Zagreb, Croatia;
[2] University of Zagreb, School of Medicine, Zagreb, Croatia;
[3] Department of Internal Medicine, General Hospital Šibenik, Šibenik, Croatia;
[4] School of Medicine, University of Rijeka, Rijeka, Croatia;
[5] School of Dental Medicine, University of Zagreb, Zagreb, Croatia

CORRESPONDENCE TO Hana Matijaca, Department of Hematology, Sestre milosrdnice University Hospital Center, Vinogradska cesta 29, HR-10000 Zagreb, Croatia
hanamatijaca@gmail.com

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ABSTRACT

The aim of this study was to investigate the efficacy of ferric carboxymaltose (FCM) in the correction of iron deficiency anemia and to determine the frequency and level of hypophosphatemia as a side effect. The data on 28 patients treated in Sestre milosrdnice University Hospital Center with one or two applications of FCM in a single dose of 1000 mg, depending on the severity of anemia, was retrospectively analyzed. The inclusion criteria were hemoglobin < 110 g/L, iron saturation < 20%, ferritin < 30 µg/L and phosphate > 0.79 mmol/L. Parameters analyzed before and two weeks after FCM application were complete blood count (CBC), ferritin, and calcium and phosphate values, while vitamin D and creatinine levels were determined only once before application. After parenteral iron supplementation, the expected correction of hemoglobin values and iron deficiency occurred. The difference in phosphate values before and after FCM application was statistically significant. The onset of asymptomatic mild to moderate hypophosphatemia was reported in 67.86% patients. In this study, baseline hemoglobin and phosphate values were predictive factors of the degree of hypophosphatemia after FCM administration.

KEYWORDS

Iron deficiency anemia; *Sideropenia*; *Ferric carboxymaltose*; *Hypophosphatemia*

Introduction

Iron deficiency anemia is the most common type of anemia[1,2]. Intravenous iron formulations ferric carboxymaltose (FCM) and iron derisomaltose (IID) allow the administration of high doses of elemental iron resulting in the correction of iron deficit in one or two infusions[1,3]. Despite a favorable overall safety profile, hypophosphatemia is a well-known side effect of parenteral iron administration. Recent data shows that its incidence and severity are highest after FCM administration[4-6]. Across numerous clinical trials, FCM induced a significantly higher incidence of hypophosphatemia than IID (47% vs 4%) accompanied by larger mean decreases in serum phosphate levels (0.4 vs 0.06 mmol/L)[3]. While often transient and asymptomatic, reported clinical manifestations of hypophosphatemia include fatigue, muscle weakness and asthenia[3]. Severe conditions, such as osteomalacia, bone pain and fractures are usually signs of prolonged and severe hypophosphatemia[7]. Other severe manifestations include respiratory failure due to diaphragm weakness, cardiac arrhythmias, constipation, ileus and neurological disturbances[7]. Hypophosphatemia after FCM application is a result of an increase in urinary phosphate excretion. It is still not entirely known why only FCM increases the phosphaturic hormone fibroblast growth factor 23 (FGF23), which causes reduced proximal tubular absorption of phosphate from the glomerular filtrate[8]. An inhibition of FGF23 cleavage by FCM has been proposed as the cause of parenteral iron–induced hypophosphatemia[8,9]. Patients treated with FCM who have impaired kidney function should have a lower risk of developing this side effect, because of a reduced glomerular filtration rate and the filtered amount of phosphate in urine[3]. Besides its effect as a phosphaturic hormone, FGF23 also inhibits the activation of vitamin D. The lower production of calcitriol (the hormonally active form of vitamin D) causes mild hypocalcemia and a compensatory increase in circulating parathyroid hormone (PTH) as a protective mechanism against severe hypocalcemia, but with a phosphaturic effect[9,10]. Consequentially, patients with a pre-existing vitamin D deficiency and low calcium and phosphate levels are more prone to severe and prolonged hypophosphatemia following FCM application[11]. Clear recommendations and protocols for the prevention and treatment of hypophosphatemia are still not available[12]. The aim of this study was to assess the early efficacy and side effects – especially the frequency, severity and risk factors – of hypophosphatemia after treatment with FCM.

Subjects and methods

We conducted a retrospective analysis of 28 patients with iron deficiency anemia treated with FCM at the Department of Hematology in Sestre milosrdnice University Hospital Center between March 1 and July 1, 2025. All procedures were performed in accordance with the 1983 revision of the Declaration of Helsinki. The patients were not required to sign an informed consent form because FCM is the standard treatment for iron deficiency anemia. The inclusion criteria were iron deficiency anemia with hemoglobin < 110 g/L, iron saturation < 20%, ferritin values < 30 µg/L and baseline serum phosphate values > 0.79 mmol/L. Prior to the administration of FCM, data on creatinine and vitamin D were obtained. The hematological parameters, iron status, and serum phosphate and calcium levels were recorded before and two weeks after treatment during a follow-up visit.

Samples for CBC were collected in K_3EDTA tubes, while serum was obtained by a centrifugation of whole blood from tubes containing a clot activator and gel separator (Vacuette, Greiner Bio-One GmbH, Kremsmünster, Austria). Iron, unsaturated iron-binding capacity, calcium, phosphate and creatinine concentrations were measured

spectrophotometrically using the Alinity-c automated biochemistry analyzer (Abbott Laboratories, Illinois, USA). Ferritin and vitamin D concentrations were determined by a chemiluminescent microparticle immunoassay (CMIA) on the Alinity-i immunochemistry analyzer (Abbott Laboratories, Illinois, USA), using the manufacturer's original reagents and protocols. CBC was performed using the Sysmex XN-1000 hematology analyzer (Sysmex Corporation, Kobe, Japan).

Statistical analysis

Descriptive statistics was used to present patient characteristics. Continuous variables were presented as mean ± standard deviation (SD) or as median (interquartile range (IQR)) for skewed data. The normality of distribution was assessed using the Shapiro–Wilk test. A paired t-test or Wilcoxon signed–rank test for dependent samples was used to compare variables before and after treatment. For comparisons between independent groups, a Student's t-test or Wilcoxon rank-sum (Mann–Whitney U test) test were applied. A multivariate analysis was performed using linear regression to assess the impact of predictors on phosphate levels after FCM administration. Predictor selection was based on clinical relevance by means of prior literature and univariate analyses (including baseline and delta values of key laboratory parameters). Multiple models were tested, and the final model was chosen on best fit (adj. R2, AIC), while others were used to explore specific hypotheses. Model assumptions were assessed visually using residual plots and Q-Q plots. No influential outliers were detected in the final model. When appropriate, non-normally distributed variables were log-transformed. All statistical analyses were conducted using SAS software (version 9.4, Cary, NC), with a significance level of $P < 0.05$.

Results

The patients' mean age was 60.79 ± 17.39 years. Only 14.29 % (N = 4) of the patients were male. The most common rationale for intravenous iron administration were gastroenterological causes (GE), accounting for 60.7% (N = 17) of cases, followed by menometrorrhagia in 32.14% (N = 9) of the cases. Half of the patients (50%, N = 14) received two intravenous doses (each application at a dose of 1000 mg with a seven-day time interval), while the other half received a single dose (1000 mg). Table 1 presents the baseline demographic, hematological and biochemical characteristics of the study cohort, as well as post-treatment laboratory values where available. All baseline serum phosphate values were within the reference range of 0.79–1.42 mmol/L, i.e., normal. The baseline vitamin D level was 55.5 nmol/L (IQR 53.5). Other parameters, such as hematological values, were consistent with an anemic cohort indicated for intravenous iron therapy. As a response to FCM administration, hemoglobin and iron parameters improved as expected. An analysis of calcium levels before and after treatment showed no statistically significant difference (Mean diff = 0.009 mmol/L, 95% CI: -0.033–0.050, paired t-test $P = 0.673$).

A statistically significant decrease in serum phosphate levels was observed following intravenous administration of FCM. Median phosphate values before and after treatment are presented in Table 1. Figure 1 shows the distribution of serum phosphate levels before and after treatment, illustrated using a box plot. The median difference in phosphate levels (pre- vs post-treatment) was 0.49 mmol/L (IQR: 0.44). This reduction was consistent across patients, as confirmed by the Wilcoxon signed–rank test (S = 202, $P < 0.0001$). A paired t-test yielded similar results (t (27) = 9.25, $P < 0.0001$, 95% CI Mean: 0.3157–0.4957), with a mean decrease of 0.41 ± 0.23 mmol/L. Furthermore, the consistent direction of change was evident, as almost all differences were positive, indicating that the phosphate

Table 1. Baseline and post-treatment demographic, hematological and biochemical characteristics of patients

Demographics	**Characteristics**		
Age, years	60.79 ± 17.39		
Sex – male	14.29 (4)		
Rationale for intravenous iron administration			
gastroenterological	60.7 (17)		
menometrorrhagia	32.14 (9)		
celiac disease	3.57 (1)		
pregnancy	3.57 (1)		
Number of FCM intravenous applications: 1	50.0 (14)		
Number of FCM intravenous applications: 2	50.0 (14)		
Laboratory	**Baseline**	**After FCM**	***P* value**
Erythrocytes, x10^9/L	3.85 ± 0.65	4.40 ± 0.48	< 0.0001
Hemoglobin, g/L	90.36 ± 13.12	119.57 ± 10.13	< 0.0001
MCV, fL	75.76 ± 6.85	84.51 ± 5.03	< 0.0001
MCH, pg	23.85 (4.65)	27.40 (3.70)	< 0.0001
MCHC, g/L	311 (24)	321.75 ± 11.40	0.0002
RDW, %	18.26 ± 3.43	22.86 ± 7.60	0.0009
Leukocytes, x10^9/L	6.91 ± 2.48	5.8 (3)	0.0257
Platelets, x10^9/L	324.75 ± 121.70	273.07 ± 91.03	0.0043
Serum iron, µmol/L	4.0 (3.0)	16.36 ± 6.29	< 0.0001
UIBC, µmol/L	66.10 ± 13.30	37.18 ± 10.66	< 0.0001
TIBC, µmol/L	70.35 ± 12.29	51.36 ± 9.98	< 0.0001
Fe saturation, %	6 (6)	29.86 ± 12.66	< 0.0001
Ferritin, µg/L	10.4 (11.3)	480.55 (502.2)	< 0.0001
Phosphate, mmol/L	1.25 (0.31)	0.72 (0.41)	< 0.0001
Calcium, mmol/L	2.27 ± 0.12	2.22 ± 0.22	0.673
Creatinine, µmol/L	72.18 ± 20.47	N/A	N/A
Vitamin D, nmol/L	55.5 (53.5)	N/A	N/A

Continuous variables are presented as mean ± standard deviation or as a median (IQR) for skewed data. Categorical variables are presented as percentages (N). Test used: Paired t-test for normally distributed variables, Wilcoxon signed–rank test for non-normally distributed variables.
Abbreviations: MCV = Mean corpuscular volume, MCH = Mean corpuscular hemoglobin, MCHC = Mean corpuscular hemoglobin concentration, RDW = Red cell distribution width, UIBC = Unsaturated iron-binding capacity, TIBC = Total iron-binding capacity, FCM = ferric carboxymaltose.

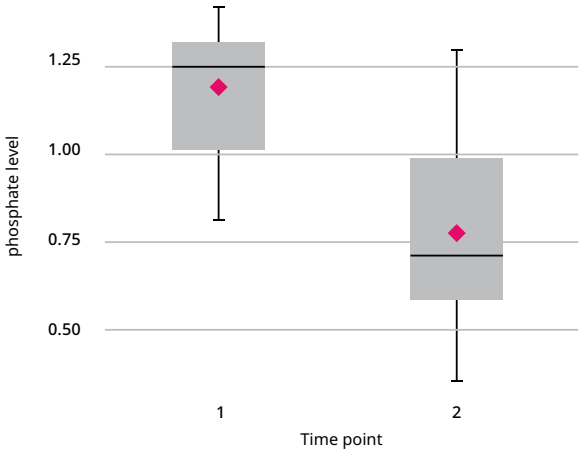

Fig. 1. Box plot of serum phosphate levels at two measurement time points. The diamond indicates the mean, while the horizontal line within the box represents the median.

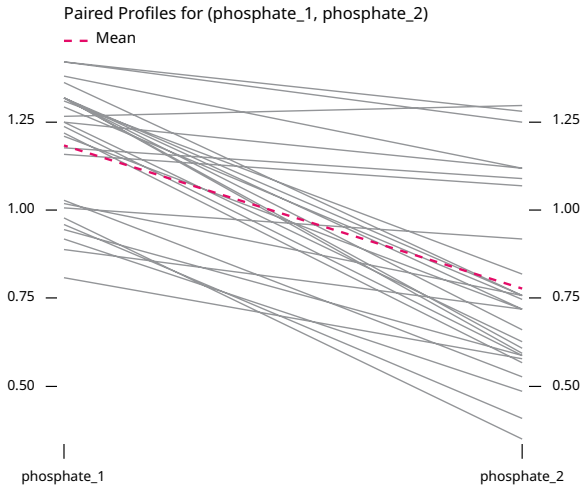

Fig. 2. Individual changes in serum phosphate levels. Each solid line represents an individual patient. The dashed line indicates the change in the mean phosphate level across the entire sample.

levels in the second measurement were consistently lower compared to baseline. This consistent decrease is also visually apparent (Figure 2). Phosphate values after FCM administration were categorized according to predefined intervals (Table 2). Hypophosphatemia (defined as serum phosphate < 0.79 mmol/L) was observed in 67.86% of the patients in this cohort. Most values (39.29%, N = 11 and 28.57%, N = 8) fell within the mild (0.6–0.79 mmol/L) and moderate (0.3–0.59 mmol/L) range. All patients were asymptomatic. No values were observed in the severe category.

Post-treatment phosphate levels were compared between patients with the GE rationale for intravenous administration of FCM (GE, N = 17) and those with the non-GE rationale (N = 11). No

Table 2. Categorical distribution of post-treatment serum phosphate levels

	Category	Phosphate range (mmol/L)	N	%
Detailed categorization	Hyperphosphatemia	‣ 1.42	0	0
	Normal	0.79 – 1.42	9	32.14
	Mild	0.6 – 0.79	11	39.29
	Moderate	0.3 – 0.59	8	28.57
	Severe	< 0.3	0	0
Binary categorization	Hypophosphatemia	< 0.79	19	67.86
	Normal	‣ 0.79	9	32.14

statistically significant difference was observed (Wilcoxon $P = 0.40$).

Post-treatment phosphate levels were compared between patients who received one (N = 14) vs two (N = 14) intravenous doses of FCM. A two-sample t-test on log-transformed data (due to improved distribution) revealed significantly lower phosphate levels in the two-dose group (t (26) = 2.22, $P = 0.035$, mean diff = 0.27, 95% CI: 0.02–0.51). The groups were comparable across most baseline characteristics, except for the hemoglobin and leukocyte count (Table 3). A multivariate linear regression, controlling for these baseline differences, found the overall model was not statistically significant (F (3,24) = 1.56, overall $P = 0.2247$) and none of the predictors showed a significant effect (number of applications $P = 0.154$, Hg $P = 0.7817$, Lp = 0.9385, VIF < 1.9). To further investigate the independent predictors of post-treatment log-transformed phosphate levels, multiple linear regression analyses were conducted. The final model included baseline creatinine, baseline phosphate and baseline hemoglobin. This model was highly statistically significant (F (3, 24) = 8.5, $P = 0.0005$, adj R2 = 0.4545) and all predictors reached statistical significance. Creatinine was a negative predictor (β = -0.0067, $P = 0.0121$), while baseline phosphate (β = 0.84, $P = 0.0065$) and baseline hemoglobin (β = 0.0087, $P = 0.0279$) were positive predictors of post-treatment phosphate levels. Phosphate was log-transformed; regression

Table 3. Baseline characteristics and post-treatment phosphate levels by the number of intravenous ferric carboxymaltose applications

Characteristic	1 IV applications	2 IV applications	*P* value
Age (years), Mean	60.57 ± 17.29	61.00 ± 18.14	0.9495
Female sex	12 (85.71)	12 (85.71)	1.000
IV iron rationale: Gastroenteritis	10 (71.43)	7(50.00)	0.440
Baseline Hemoglobine, g/L	97.34 ± 10.46	83.34 ± 11.95	0.0028
Baseline Leukocytes, x10^9/L	7.86 ± 2.58	5.95 ± 2.03	0.0383
Baseline Platelets, x10^9/L	332.1 ± 82.97	317.4 ± 154.1	0.7545
Baseline ferritin, µg/L	11.20 (7.90-20.50)	10.0 (7.00 – 14.70)	0.5656
Baseline calcium, mmol/L	2.29 ± 0.12	2.58 ± 0.12	0.5365
Baseline phosphate, mmol/L	1.22 ± 0.16	1.15 ± 0.19	0.2701
Baseline creatinine, µmol/L	69.64 ± 19.87	74.71 ± 21.47	0.5223
Baseline vitamin D, nmol/L	60.0 (19.0 -75.0)	33.5 (27.0-74.0)	0.9085
Post-treatment phosphate, mmol/L	0.79 (0.61-1.12)	0.69 (0.57-0.79)	0.035*

Continuous variables are presented as mean ± standard deviation or as median (IQR) for skewed data. Categorical variables are presented as percentages (N). Test used: Student's t-test for continuous normally distributed variables, the Wilcoxon rank-sum test (Mann–Whitney U test) for continuous non-normally distributed data and Fisher's exact test for categorical variables. *For post-treatment phosphate levels, the *P* value was derived from a two-sample t-test on log-transformed data due to improved data distribution.

Table 4. Multivariate linear regression model predicting post-treatment phosphate levels (N = 28). Dependent variable: log-transformed post-treatment phosphate levels.

Variable	Parameter estimate	Standard error	t value	Pr > |t|	Variance inflation	95% Confidence limits	
Intercept	-1.60778	0.52235	-3.08	0.0052	0	-2.68585	-0.52971
creatinine	-0.00671	0.00247	-2.71	0.0121	1.09508	-0.01181	-0.00161
phosphate_1	0.84362	0.28324	2.98	0.0065	1.07472	0.25904	1.42820
Hg 1	0.00871	0.00372	2.34	0.0279	1.01992	0.00103	0.016
Model statistics	**Value**						
F-static (df)	8.50 (3,24)						
Overall *P*-value	0.0005						
R^2	0.5152						
Adjusted R^2	0.4545						

coefficients indicate approximate percent changes per unit predictor. Detailed results are presented in Table 4. Although a model that included the number of intravenous applications along with phosphate and creatinine also yielded a statistically significant overall fit ($P = 0.0013$; adjusted $R^2 = 0.4098$), the variable number of applications was not significant at the conventional threshold ($P = 0.0842$).

The baseline values of vitamin D (shown separately in the groups of patients with one and two applications of FCM) did not differ between patients receiving one vs two applications (median 60.0 vs 33.5 nmol/L, $P = 0.9085$ (Table 3). In a univariate analysis, baseline vitamin D was not associated with post-treatment phosphate levels (Spearman's $\rho = 0.118$, $P = 0.548$), nor with the change in phosphate (Δ phosphate; Spearman's $\rho = -0.253$, $P = 0.194$). When baseline vitamin D was added as a main effect to the primary multivariate model predicting log-transformed post-treatment phosphate levels, vitamin D did not show an association with the outcome $P = 0.169$.

Discussion

Although the latest generation of intravenous iron preparations such as FCM allow for a quick and safe correction of iron deficit in one or two applications in patients with iron deficiency anemia, hypophosphatemia is recognized as a delayed adverse effect[12-19]. The overall incidence and clinical significance of this side effect are not entirely known because of inconsistent reporting.

The results of our study showed that hemoglobin and iron parameters improved as expected after FCM administration. Hypophosphatemia (defined as serum phosphate < 0.79 mmol/L) was observed in 67.86% of the patients in this cohort. It is important to emphasize that most post-treatment hypophosphatemia values were mild and moderate (39.29% and 28.57%, respectively) and that all the patients were asymptomatic. Despite the high rate of incidence, literature data suggest that hypophosphatemia is self-limited and asymptomatic in the majority of patients[19]. No values in our study were observed in the severe hypophosphatemia category.

Meta-analysis data in the literature have identified preserved renal function, the severity of iron deficiency and the dose of FCM as predictors of hypophosphatemia[3]. In our study, the univariate analysis indicated a significant difference in phosphate levels between the one and two dose groups; this association did not remain significant in multivariate regression after adjustment. Clinical practice often dictates giving more doses to patients with severe anemia. In this analysis, the observed univariate difference in phosphate levels likely reflects the more severe baseline clinical status that influenced the dosing decision, rather than an independent effect of the number of applications itself.

The independent significance of baseline hemoglobin in the final model is particularly noteworthy. While the role of hemoglobin in the initial model may have been more tied to confounding treatment assignment, its direct predictive power in the final model highlights its deeper association with phosphate metabolism in our study cohort, regardless of treatment dose. These findings suggest that baseline biochemical and hematological parameters may have a greater predictive contribution to post-treatment phosphate levels than the number of administered doses itself. In the final model, higher baseline hemoglobin was associated with higher post-treatment phosphate levels. Higher baseline phosphate also predicted higher post-treatment levels.

Conversely to the available data, higher creatinine levels were linked with lower post-treatment phosphate levels. Mean baseline creatinine was 72.18 ± 20.47 µmol/L, suggesting that the cohort in our study predominantly consisted of individuals with preserved or only mildly impaired renal function. Maybe these contrary findings in our study can be justified by excluding patients with severely impaired renal function. The small size of our sample should also be considered when interpreting these findings. Aside from sample size, the main limitations of our study were its retrospective observational nature and only two measurements of phosphate levels with no repetitive measurements in a longer follow-up period. This was done under the presumption that the lowest levels would be recorded two weeks after FCM application[19].

The prevention of hypophosphatemia as an unwanted side effect remains unclear. Considering the data, we believe that the different degrees of hypophosphatemia (< 0.3 mmol/L, 0.3–0.59 mmol/L, 0.6–0.79 mmol/L) should be recorded during follow-up visits and must become standard practice in reporting this side effect due to the increased risk for complications which correlates with the severity of hypophosphatemia[20-23].

The suggestion is that patients treated with FCM should be informed of this possible adverse effect and seek medical help in cases of deterioration of fatigue, myalgias or bone pain as clinical signs of hypophosphatemia. It would be reasonable to monitor serum phosphate levels, especially in patients treated with repetitive administrations of FCM and in those with existing risk factors as mentioned before.

Conclusion

In conclusion, mild asymptomatic hypophosphatemia is a common laboratory finding after the administration of FCM. Patients on long-term iron replacement therapy and those with lower baseline hemoglobin levels may be at higher risk. We suggest to monitor phosphate levels in these circumstances. The clinical decision on the type of the parenteral iron formulation should be assessed on an individual basis, taking into account existing comorbidities and risk factors.

DISCLOSURE OF CONFLICT OF INTEREST The authors declare no conflict of interest.

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SAŽETAK

Rana učinkovitost i hipofosfatemija nakon terapije željezovom karboksimaltozom u bolesnika s anemijom zbog manjka željeza: iskustvo jednog centra

Hana Matijaca, Klara Brčić, Klara Grgorinić, Ivan Krečak i Petar Gaćina

Cilj ovog rada bio je istražiti učinkovitost korekcije sideropenične anemije visokodoznim parenteralnim pripravkom željezove karboksimaltoze (FCM) te odrediti učestalost i razinu hipofosfatemije kao nuspojave. Retrospektivno su analizirani podaci 28 bolesnika liječenih u Kliničkom bolničkom centru Sestre milosrdnice jednom ili dvije aplikacije FCM-a u pojedinačnoj dozi od 1000 mg ovisno o težini anemije. Uključni kriteriji bili su vrijednost hemoglobina < 110 g/L, Fe saturacije < 20 %, feritina < 30 µg/L te vrijednosti fosfata > 0,79 mmol/L. Parametri koji su analizirani prije i dva tjedna nakon aplikacije FCM-a su kompletna krvna slika, ferogram, feritin i vrijednosti kalcija i fosfata, dok su razine vitamina D i kreatinina određeni samo jednom prije aplikacije. Nakon parenteralne nadoknade željeza došlo je do očekivane korekcije vrijednosti hemoglobina i deficita željeza. Razlika u vrijednosti fosfata prije i poslije aplikacije FCM-a bila je statistički značajna. Novonastala, asimptomatska hipofosfatemija blagog i srednjeg stupnja zabilježena je u 67,86 % ispitanika. U ovom istraživanju početne vrijednosti hemoglobina i fosfata bile su prediktivni čimbenici stupnja hipofosfatemije nakon aplikacije FCM-a.

KLJUČNE RIJEČI

Anemija zbog manjka željeza; Sideropenija; Željezova karboksimaltoza; Hipofosfatemija