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Effects of ferric carboxymaltose on hypophosphatemia in patients with iron deficiency anemia: A single-center retrospective study

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Abstract

A retrospective analysis was conducted to assess hypophosphatemia and associated hormonal alterations in patients with iron deficiency anemia treated with ferric carboxymaltose (FCM). Overall, 163 patients were evaluated. Hematological and biochemical parameters, including phosphorus, calcium, magnesium, parathyroid hormone (PTH), and 1,25-dihydroxy vitamin D, were evaluated before and after FCM administration. For the purposes of this study, serum phosphorus levels below 2.5 mg/dL were considered indicative of hypophosphatemia, whereas levels below 1 mg/dL were classified as severe hypophosphatemia. Of the patients, 20.9% were male (n = 34) and 79.1% were female (n = 129). The median age was 56 years (25th–75th percentile: 45–71). Fifty-four patients (33%) received 500 mg and 109 patients (67%) received 1000 mg of FCM. Serum phosphorus levels measured on day 14 after treatment were significantly lower compared to baseline values (3.4 ± 0.6 vs. 2.4 ± 0.9; $p < 0.001$). Hypophosphatemia developed in 91 patients (55.8%). While serum phosphorus levels decreased significantly, PTH levels increased (72.7 [50.7–105.3] vs. 84.4 [60.7–122.6]; $p < 0.001$), and 1,25-dihydroxy vitamin D levels showed a significant decline (35.8 ± 15.5 to 26.7 ± 16.2). A significantly higher risk of hypophosphatemia was observed among patients receiving 1000 mg FCM compared with those receiving 500 mg (3.7-fold increase, $p = 0.001$). Moreover, the rate of hypophosphatemia detected in our study population was comparable to previously published data. Although FCM provides rapid improvement in hematological parameters, it is associated with transient, dose-dependent hypophosphatemia. Hypophosphatemia occurred in 55.8% of patients and was significantly more frequent in patients receiving 1000 mg ferric carboxymaltose.

Keywords: Iron deficiency anemia, ferric carboxymaltose, 1.25-dihydroxy vitamin D, hypophosphatemia, parathyroid hormone

Introduction

Globally, more than 25% of individuals are impacted by anemia, and iron deficiency is responsible for about half of this prevalence. Anemia remains an important health concern worldwide, with a disproportionate impact on women of reproductive age, pregnant women, and preschool-aged children [1]. The prevention and treatment of iron deficiency remain major public health goals, especially in women, children, and individuals living in low-income countries. Challenges in the management of iron deficiency include identifying and addressing the underlying cause and selecting an iron replacement therapy that meets the patient's needs [2]. World Health Organization (WHO) reports indicate that the prevalence of anemia among women of reproductive age in Türkiye is approximately 29% [3].

Among individuals without inflammation, values of serum ferritin below 30 ng/mL and transferrin saturation below 20% were considered indicative of iron deficiency. The etiology of iron deficiency should be investigated and addressed accordingly. Oral iron therapy (iron sulfate 325 mg daily or every other day) is generally the first-line treatment [4]. Parenteral iron therapy should be considered in individuals who do not tolerate oral supplementation, exhibit impaired iron absorption, have chronic inflammatory diseases, continue to lose blood, or are in advanced stages of pregnancy (second or third trimester) [4]. Oral iron is generally prescribed as the initial treatment; however, gastrointestinal adverse effects, limited bioavailability, and poor

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compliance frequently restrict its use [5].

Ferric carboxymaltose (FCM) is an intravenous iron compound designed for rapid delivery of high iron doses, with a favorable safety profile and minimal risk of anaphylaxis [6].

Transient hypophosphatemia is a known adverse effect of intravenous iron therapy and has been reported to occur more frequently and persist longer with FCM compared to other intravenous iron formulations. A temporary increase in fibroblast growth factor-23 (FGF-23), attributed to the molecular properties of FCM, is considered a key mechanism underlying hypophosphatemia. FGF-23 elevation leads to enhanced urinary phosphate loss and reduced production of 1.25-dihydroxyvitamin D [7,8]. Although hypophosphatemia is often asymptomatic, repeated high-dose FCM administration has been associated with serious complications such as osteomalacia and fractures [9].

We retrospectively analyzed hypophosphatemia rates, related biochemical and hormonal changes, and the association between these findings and FCM dosage in patients with iron deficiency anemia receiving FCM therapy.

Material and Methods

We conducted a retrospective chart review of patients with iron deficiency anemia (IDA) treated with intravenous FCM at the Hematology Outpatient Clinic of Düzce Atatürk State Hospital between December 1, 2024, and July 1, 2025. A total of 163 patients were included.

Patients aged ≥ 18 years with a diagnosis of IDA who received intravenous FCM treatment, had available serum phosphate levels before treatment and at the second week after treatment, and had at least four weeks of follow-up were included. Individuals with advanced chronic kidney disease (estimated glomerular filtration rate (eGFR) GFR below 30 mL/min/1.73 m², corresponding to stages 4–5), parathyroid disorders, metabolic bone disease related to vitamin D deficiency, use of phosphate binders, use of medications affecting serum phosphate levels (e.g., tenofovir, pamidronate), or incomplete clinical or laboratory data were excluded.

Ferric carboxymaltose was administered intravenously as a single infusion of either 500 mg or 1000 mg according to clinical requirements. Demographic data (age, sex), hemoglobin, hematocrit, ferritin, transferrin saturation, serum phosphorus levels (before treatment and at week 2), serum creatinine, calcium, magnesium, eGFR, and 1.25-dihydroxy vitamin D levels were recorded from patient files before treatment and on day 14 after treatment. All data were anonymized prior to analysis.

Based on the WHO criteria, anemia was defined as a hemoglobin level below 13 g/dL in men aged 15 years and older, below 12 g/dL in non-pregnant women aged 15 years and older, and below 11 g/dL in pregnant women [10].

Transferrin saturation (TS) was calculated using the following formula: (serum iron level / total iron-binding capacity) ×100 [11]. Hypophosphatemia was considered present when serum phosphorus levels were below 2.5 mg/dL, while levels below 1 mg/dL were classified as severe hypophosphatemia [12].

We conducted a retrospective observational study at a single center. Ethical approval was obtained from the Düzce University Non-Interventional Health Research Ethics Committee (August 25, 2025; approval no. 2025/167). The study was performed in compliance with the principles outlined in the Declaration of Helsinki.

Statistical Analysis

Categorical data were summarised as numbers and percentages, whereas numerical variables were summarised as mean ± standard deviation or as median values with the interquartile range (25th–75th percentile), as appropriate. The Kolmogorov–Smirnov test was utilised to evaluate the normality of the numerical variables. Comparisons between two independent groups were performed using the Student's t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. In instances where repeated measurements were compared, the paired-samples t-test was employed when the parametric assumptions were met, whereas the Wilcoxon signed-rank test was utilised when these assumptions were violated. The potential of baseline laboratory parameters obtained prior to ferric carboxymaltose administration and ferric carboxymaltose dose to predict hypophosphatemia was evaluated. Initially, univariate logistic regression analyses were performed and variables with a p-value less than 0.25 were considered potential risk factors.The aforementioned variables were entered into a multiple logistic regression model using the "enter" method with the objective of identifying independent predictors of hypophosphatemia. The performance of the model was evaluated using the Nagelkerke R^2 statistic. A p value below 0.05 in two-tailed tests was accepted as indicating statistical significance. Statistical analyses were conducted using IBM SPSS Statistics software (version 20.0; IBM Corp., Armonk, NY, USA).

Results

The analysis comprised 163 patients. Of the patients, 129 (79.1%) were female and 34 (20.9%) were male. The median age of the total 163 patients was 56 years (25th–75th percentile: 45–71). Fifty-four patients (33%) received 500 mg, and 109 patients (67%) received 1000 mg doses of ferric carboxymaltose (FCM).

As demonstrated in Table 1, substantial alterations were detected in multiple laboratory parameters subsequent to ferric carboxymaltose treatment. Statistical analysis revealed a significant increase in both haemoglobin and hematocrit levels post-treatment (both $p < 0.001$). Iron-related parameters, including ferritin and transferrin saturation, also demonstrated significant increases in comparison with baseline values ($p < 0.001$ for both). Conversely, serum phosphorus and calcium levels exhibited a significant decrease following treatment ($p < 0.001$ for both). Following the administration of ferric carboxymaltose, a significant increase was observed in parathyroid hormone levels, whilst 1.25-dihydroxy vitamin D levels decreased (both $p < 0.001$). A modest yet statistically significant increase was observed in alkaline phosphatase levels ($p = 0.002$). Minimal but statistically significant changes were observed in magnesium and creatinine levels after treatment ($p = 0.004$ and $p = 0.002$, respectively).

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Table 1. Comparison of laboratory parameters before and after ferric carboxymaltose treatment

Laboratory parameters	Before	After	P value
Hemoglobin (g/dL)	10.8 ± 1.9	11.7 ± 1.5	< 0.001
Hematocrit (%)	34.2 ± 5.1	36.8 ± 4.3	< 0.001
Ferritin (ng/mL)	17.6 (9.6 − 26.9)	371.4 (222.6 − 596)	< 0.001
Transferrin saturation (%)	10 (6 − 16)	31 (22 − 39)	< 0.001
Phosphorus (mg/dL)	3.4 ± 0.6	2.4 ± 0.9	< 0.001
PTH (pg/mL)	72.7 (50.7 − 105.3)	84.4 (60.7 − 122.6)	< 0.001
1,25 dihydroxyvitamin D (pg/mL)	35.8 ± 15.5	26.7 ± 16.2	< 0.001
Calcium (mg/dL)	8.9 ± 0.5	8.7 ± 0.5	< 0.001
Magnesium (mg/dL)	1.90 ± 0.2	1.94 ± 0.2	0.004
ALP (U/L)	79 (61.5 − 99.5)	83 (63 − 104.5)	0.002
Creatinine (mg/dL)	0.70 (0.59 − 0.89)	0.70 (0.59 − 0.87)	0.002

PTH: Parathyroid hormone, ALP: Alkaline phosphatase; In the dependent groups t-test, the values are presented as the mean ± standard deviation; In the Wilcoxon signed-rank test, the values are presented as the median (25th–75th percentile)

As demonstrated in Table 2, hypophosphatemia was observed in 91 patients (55.8%) on day 14 after ferric carboxymaltose treatment. Patients who experienced hypophosphatemia had received significantly higher cumulative doses of ferric carboxymaltose than those without hypophosphatemia ($p < 0.001$). In contrast, age and sex distributions did not differ significantly between the groups. Baseline serum phosphorus and calcium levels were significantly lower in patients who developed hypophosphatemia than in those who did not (both $p < 0.001$). No statistically significant differences were found between the groups in terms of hemoglobin, hematocrit, ferritin, transferrin saturation, parathyroid hormone, vitamin D metabolites, magnesium, alkaline phosphatase, or creatinine (all $p > 0.05$). Clinical records were reviewed for symptoms related to hypophosphatemia. No severe or clinically significant symptoms attributable to hypophosphatemia were documented in the patient records. In addition, no patient required phosphate or vitamin D replacement therapy during follow-up.

Table 2. Comparison of laboratory parameters according to hypophosphatemia status

Variables		Not hypophosphatemia	Hypophosphatemia	p value
Age		54 (43 − 66)	60 (45 − 74)	0.115
Gender	**Male**	12 (16.7)	22 (24.2)	0.328
	Female	60 (83.3)	69 (75.8)	
FCM dose	**500**	36 (50)	18 (19.8)	< 0.001
	1000	36 (50)	73 (80.2)	
Baseline parameters	**Hemoglobin (g/dL)**	10.7 ± 2.1	10.8 ± 1.7	0.594
	Hematocrit (%)	34.4 (30.4 − 38.4)	33.5 (30.6 − 37.8)	0.941
	Ferritin (ng/mL)	15.3 (8.5 − 25.5)	18.6 (10.6 − 30)	0.243
	Transferrin saturation (%)	10 (6 − 16)	11.8 ± 7.4 10 (6 − 15)	0.982
	Phosphorus (mg/dL)	3.6 ± 0.5	3.2 ± 0.6	< 0.001
	PTH (pg/mL)	74.1 (49.5 − 97.2)	69.3 (50.7 − 109.8)	0.626
	1,25-dihydroxyvitamin D (pg/mL)	34 ± 15.7	36.8 ± 15.4	0.393
	Calcium (mg/dL)	9 ± 0.4	8.8 ± 0.5	0.001
	Magnesium (mg/dL)	1.9 ± 0.2	1.9 ± 0.2	0.887
	ALP (U/L)	78 (60.8 − 88.5)	85 (63 − 105)	0.103
	Creatinine (mg/dL)	0.6 (0.6 − 0.9)	0.7 (0.6 − 0.9)	0.082

FCM: Ferric carboxymaltose, PTH: Parathyroid hormone, ALP: Alkaline phosphatase; In the independent samples t-test, values are presented as mean ± standard deviation; In the Mann–Whitney U test, values are presented as median (25th–75th percentile)

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As demonstrated in Table 3, the administration of ferric carboxymaltose and the baseline serum phosphorus level were found to be independently associated with the development of hypophosphatemia. In the adjusted analysis, patients receiving 1000 mg ferric carboxymaltose had an approximately 3.7-fold higher likelihood of developing hypophosphatemia compared with those receiving 500mg (OR = 3.68;95% CI: 1.69-8.02; p = 0.001), indicating a clear dose-dependent relationship between ferric carboxymaltose exposure and hypophosphatemia risk.

Conversely, higher baseline serum phosphorus levels were independently associated with a reduced risk of hypophosphatemia (OR = 0.30; 95% CI: 0.15-0.61; p = 0.001). In the multiple logistic regression model, the baseline variables of age, serum calcium, alkaline phosphatase, and ferritin levels did not retain statistical significance.

Table 3. Univariate and multiple logistic regression analysis of risk factors for hypophosphatemia

Variable	Univariate		Multiple	
	OR (95% CI)	p	OR (95% CI)	p
Age (years)	1.02 (0.996 − 1.036)	0.109	1.01 (0.991 − 1.036)	0.265
Ferric carboxymaltose dose (ref: 500mg)	4.06 (2.03 − 8.10)	< 0.001	3.68 (1.69 − 8.02)	0.001
Calcium (mg/dL)	0.33 (0.163 − 0.670)	0.002	0.59 (0.26 − 1.33)	0.215
Alkaline phosphatase (U/L)	1.01 (0.999 − 1.019)	0.082	1.01 (0.99 − 1.02)	0.363
Phosphorus (mg/dL)	0.28 (0.143 − 0.534)	< 0.001	0.30 (0.15 − 0.61)	0.001
Hemoglobin (g/dL)	1.05 (0.886 − 1.234)	0.591	-	-
Hematocrit (%)	1.01 (0.945 − 1.069)	0.883	-	-
Ferritin (ng/mL)	1.01 (0.989 − 1.023)	0.517	-	-
Transferrin saturation (%)	0.99 (0.966 − 1.012)	0.365	-	-
Parathyroid hormone (pg/mL)	1.01 (0.996 − 1.017)	0.258	-	-
1,25-dihydroxy vitamin D (pg/mL)	1.01 (0.985 − 1.040)	0.390	-	-
Magnesium (mg/dL)	0.90 (0.200 − 4.017)	0.886	-	-
Creatinine (mg/dL)	1.41 (0.445 − 4.474)	0.560	-	-

OR: Odds ratio, CI: Confidence interval; Multiple logistic regression was performed using the "enter" method; Model performance: Nagelkerke R^2 = 0.272

As demonstrated in Table 4, the frequency of hypophosphatemia differed significantly between ferric carboxymaltose dose groups (p < 0.001), with a higher proportion observed in patients receiving 1000 mg. Hypophosphatemia developed in 59.6% of patients receiving 1000 mg FCM (65/109), whereas this rate was 31.5% in patients receiving 500 mg FCM (17/54). In addition, eight of the nine patients with severe hypophosphatemia (< 1 mg/dL) were in the 1000 mg dose group.

Table 4. Distribution of hypophosphatemia status according to ferric carboxymaltose dose

FCM dose	Hypophosphatemia status			p
	Normal	Hypophosphatemia	Severe hypophosphatemia	
500 mg (n = 54)	36 (66.7)	17 (31.5)	1 (1.8)	< 0.001
1000 mg (n = 109)	36 (33.0)	65 (59.6)	8 (7.3)	

FCM: Ferric carboxymaltose; Severe hypophosphatemia was defined as serum phosphorus < 1.0 mg/dL; Values are presented as n (%), calculated as row percentages within each dose group

Discussion

In this study, we demonstrated that FCM treatment leads to a dose-dependent and significant reduction in serum phosphorus levels in addition to hematological improvement. According to data obtained from a total of 163 patients, hypophosphatemia developed in 55.8% (n = 91) of patients. This rate is consistent with the 40–70% range reported in the literature. However, the most striking finding is the dose relationship: hypophosphatemia was observed in 59.6% of the 109 patients receiving 1000 mg FCM, whereas this rate was significantly lower in those receiving 500 mg. The odds ratio (OR) of 3.7 obtained from our logistic regression analysis confirms that high-dose treatment independently increases the risk of hypophosphatemia and that this adverse effect is not a "rare finding" but rather an expected outcome [13].

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Our findings are largely consistent with results reported in the literature. In a single-center retrospective study conducted by Terzi Demirsoy et al., the incidence of hypophosphatemia after FCM treatment was reported as 67.4%, and the rate of hypophosphatemia was shown to be significantly higher, particularly in patients receiving 1000 mg FCM. In our study, the incidence of hypophosphatemia after FCM treatment was found to be 55.8%. In both studies, low baseline phosphorus levels and high FCM dose were identified as independent risk factors for the development of hypophosphatemia. Similarly, in our study, the risk of hypophosphatemia was approximately 3.7-fold higher in patients receiving 1000 mg FCM compared to those receiving 500 mg, and baseline phosphorus level was shown to be a protective factor [14].

In some studies reported in the literature, the higher rates of hypophosphatemia observed after FCM treatment have been attributed to differences in patient populations, dosing regimens, and timing of phosphorus measurements. While Terzi Demirsoy et al. reported that a baseline phosphorus level ≤ 3.4 mg/dL increased the risk of hypophosphatemia by approximately ninefold, the lower odds ratio observed in our study may be related to a larger patient population and stricter exclusion criteria. Nevertheless, a common finding in both studies is that high-dose FCM administration is a strong risk factor for hypophosphatemia [14].

The pathophysiological mechanism underlying this condition caused by FCM occurs directly through the hormone FGF-23. Under normal conditions, iron deficiency increases FGF-23 production while allowing its cleavage; however, the carboxymaltose shell of FCM inhibits this cleavage, leading to pathologically elevated levels of intact FGF-23 [8]. In a study conducted by Schouten et al., serum phosphorus levels decreased from 3.4 mg/dL to 1.8 mg/dL within one week after IV iron administration, accompanied by a reduction of more than 80% in active vitamin D levels. In our study, the significant decrease in active vitamin D levels after treatment (from 35.8 to 26.7 pg/mL) and the secondary increase in parathyroid hormone (PTH) levels (from 72.7 to 84.4 pg/mL) represent the clinical reflections of this complex hormonal interaction affecting mineral homeostasis. Severe hypophosphatemia may lead to clinically significant complications, including muscle weakness, respiratory failure, cardiac dysfunction, hemolysis, and neurological symptoms. In rare cases, profound hypophosphatemia may require urgent medical intervention and phosphate replacement therapy. Therefore, awareness of this potential complication is important, particularly in patients receiving high-dose intravenous iron therapy [12,15].

As emphasized by Van Doren and Auerbach, there are marked differences among modern IV iron formulations in terms of hypophosphatemia risk; FCM carries a much higher risk compared to preparations such as ferric derisomaltose or iron dextran. In our study, eight of the nine cases of severe hypophosphatemia occurred in the 1000 mg dose group, and patients with low baseline phosphorus levels were found to be at higher risk ($p < 0.001$), confirming that this represents a clinically critical adverse effect [13].

Overall, our findings demonstrate that hypophosphatemia following ferric carboxymaltose administration is common and shows a clear dose-dependent pattern. Patients receiving 1000 mg FCM had a significantly higher risk of developing hypophosphatemia compared with those receiving 500 mg. These findings highlight the importance of considering baseline phosphorus levels and monitoring mineral metabolism in patients receiving higher doses of intravenous iron.

Limitations

This study has several limitations. First, its retrospective and single-center design may limit the generalizability of the findings. Second, serum phosphate levels were measured only at baseline and on day 14 after ferric carboxymaltose administration; therefore, the exact timing of the nadir phosphate level and the persistence of hypophosphatemia could not be determined. Serial phosphate measurements would be required to better characterize the temporal course of hypophosphatemia after treatment. Third, clinical symptoms related to hypophosphatemia may have been underreported because of the retrospective nature of the study.

Conclusion

This study showed that ferric carboxymaltose treatment leads to rapid and effective improvement in laboratory parameters in individuals with iron deficiency anemia, while also resulting in a dose-dependent and substantial decline in serum phosphorus levels. Data obtained from a total of 163 patients indicate that hypophosphatemia following FCM treatment is not merely a transient laboratory abnormality but represents a systemic disturbance of mineral metabolism characterized by suppression of active vitamin D synthesis secondary to increased FGF-23 levels and the development of secondary hyperparathyroidism.

The most important implication of our study is the direct association between hypophosphatemia risk and the administered iron dose. The 59.6% incidence of hypophosphatemia and the 3.7-fold increased risk (OR) observed in the 109 patients receiving 1000 mg FCM highlight the sensitivity of mineral balance to high-dose treatment. In addition, the higher risk of developing severe hypophosphatemia among patients with low baseline phosphorus levels indicates that this patient group requires special attention.

In conclusion, although FCM therapy represents one of the most important components of the “Iron Revolution,” clinicians are advised to monitor phosphorus and vitamin D levels on day 14 after treatment, particularly in high-risk patient groups and in those receiving high doses such as 1000 mg. This approach is of critical importance for preventing long-term complications such as osteomalacia and muscle weakness.

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Ethical Approval
This study was approved by the Düzce University Non-Interventional Clinical Research Ethics Committee (Decision No: 2025/167, Date: August 25, 2025). The study was conducted in accordance with the Declaration of Helsinki.

Patient Consent
The requirement for informed consent was waived by the ethics committee due to the retrospective nature of the study.

Conflict of Interest
The authors confirm the absence of any conflicts associated with this work.

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References

1. Pobee RA, Setorglo J, Klevor M, Murray-Kolb LE. The prevalence of anemia and iron deficiency among pregnant Ghanaian women, a longitudinal study. PLoS One. 2021;16:e0248754.
2. De Franceschi L, Iolascon A, Taher A, Cappellini MD. Clinical management of iron deficiency anemia in adults: systemic review on advances in diagnosis and treatment. Eur J Intern Med. 2017;42:16-23.
3. World Health Organization. The global prevalence of anaemia in 2011. Geneva: World Health Organization; 2015.
4. Auerbach M, DeLoughery TG, Tirnauer JS. Iron deficiency in adults: a review. JAMA. 2025;333:1813-23.
5. Moore RA, Gaskell H, Rose P, Allan J. Meta-analysis of efficacy and safety of intravenous ferric carboxymaltose (Ferinject) from clinical trial reports and published trial data. BMC Blood Disord. 2011;11:4.
6. Barish CF, Koch T, Butcher A, et al. Safety and efficacy of intravenous ferric carboxymaltose (750 mg) in the treatment of iron deficiency anemia: two randomized, controlled trials. Anemia. 2012;2012:172104.
7. Ifie E, Oyibo SO, Joshi H, Akintade OO. Symptomatic hypophosphataemia after intravenous iron therapy: an underrated adverse reaction. Endocrinol Diabetes Metab Case Rep. 2019;2019:19-0065.
8. Schouten BJ, Hunt PJ, Livesey JH, et al. FGF23 elevation and hypophosphatemia after intravenous iron polymaltose: a prospective study. J Clin Endocrinol Metab. 2009;94:2332-7.
9. Shimada T, Mizutani S, Muto T, et al. Cloning and characterization of FGF23 as a causative factor of tumor-induced osteomalacia. Proc Natl Acad Sci U S A. 2001;98:6500-5.
10. McLean E, Cogswell M, Egli I, et al. Worldwide prevalence of anaemia, WHO vitamin and mineral nutrition information system, 1993-2005. Public Health Nutr. 2009 ;12:444-54.
11. World Health Organization. International statistical classification of diseases and related health problems. 10th rev, 5th ed. Geneva: World Health Organization; 2016.
12. Subramanian R, Khardori R. Severe hypophosphatemia. Pathophysiologic implications, clinical presentations, and treatment. Medicine (Baltimore). 2000;79:1-8.
13. Van Doren L, Auerbach M. IV iron formulations and use in adults. Hematology Am Soc Hematol Educ Program. 2023;2023:622-9.
14. Terzi Demirsoy E. Incidence of hypophosphatemia after ferric carboxymaltose treatment: single center experience. Namik Kemal Med J. 2021;9:201-6.
15. Schouten BJ, Doogue MP, Soule SG, Hunt PJ. Iron polymaltose-induced FGF23 elevation complicated by hypophosphataemic osteomalacia. Ann Clin Biochem. 2009;46:167-9.