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Beyond Iron: Unmasking Hypophosphatemia in a Young Female Following Intravenous Iron Transfusion: A Case Report

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Abstract

Intravenous iron transfusions, commonly used to treat iron deficiency anaemia, are typically considered safe. However, this case report underscores a rare but noteworthy complication: Hypophosphatemia in a young woman after receiving this treatment. The case report details her clinical presentation, laboratory results, management, and outcome, highlighting the necessity for vigilance and proactive monitoring in similar situations.

Keywords: Hypophosphatemia, Intravenous Iron Transfusion.

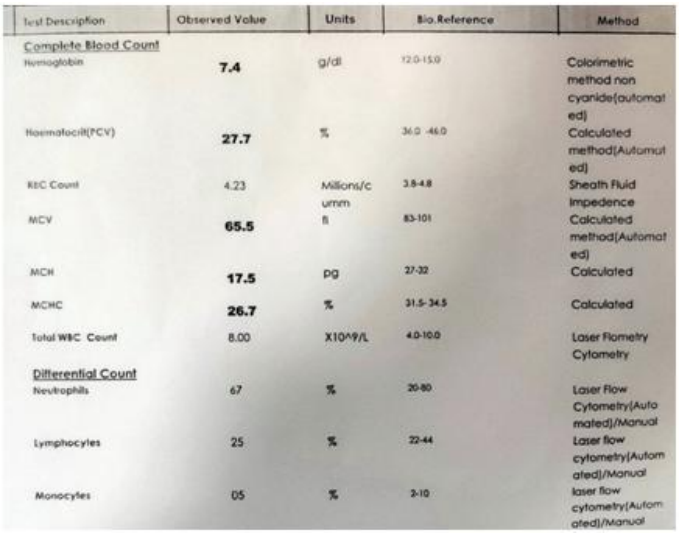
Introduction

Iron deficiency is a significant cause of anemia in patients with chronic kidney disease, inflammatory bowel disease, intestinal malabsorption syndromes, and women with heavy menstrual bleeding. However, not all cases involve anemia. Even without anemia, iron deficiency can cause symptoms like fatigue, irritability, apathy, and depression. Consequently, iron replacement therapy is often started while investigating the underlying cause. Oral iron supplements are typically the first choice for treating iron deficiency, but they frequently cause gastrointestinal side effects, have low bioavailability, and often lead to poor adherence. Therefore, the parenteral route is commonly preferred. Iron infusions, such as ferric carboxymaltose, can cause hypophosphatemia, which is usually transient and asymptomatic. This report discusses a rare case of hypophosphatemia in a young woman following intravenous iron therapy.

Case Presentation

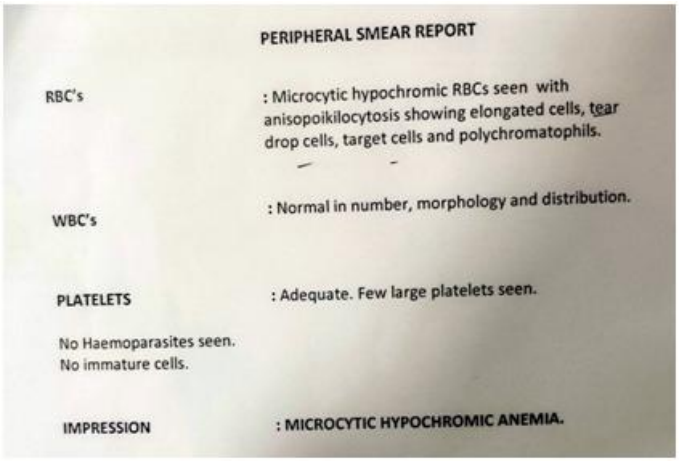
A 30-year-old woman presented with generalized fatigue, body aches, and palpitations, with no other significant complaints. Upon examination, pallor was noted. Systemic examination was unremarkable. Laboratory tests revealed a hemoglobin level of 7.4 mg/dL (see Figure 1). Peripheral smear and iron studies confirmed a picture consistent with iron deficiency anemia (see Figures 2 and 3). The patient received two doses of ferric carboxymaltose over a two-weeks period. One week after the last infusion, she returned with complaints of severe bone pain and fatigue. Laboratory investigations showed improvement in hemoglobin levels to 10.5 g/dL from the pre-infusion level of 7.4 g/dL, but serum phosphate was low at 1.8 mg/dL (reference range: 2.5-4.5 mg/dL).

Levels of serum calcium, magnesium, and parathyroid hormone were within normal limits. Based on the symptoms and low phosphate levels, hypophosphatemia secondary to intravenous iron transfusions was diagnosed. Further intravenous iron therapy was discontinued, and the patient was started on oral phosphate supplements while encouraged to increase intake of phosphate-rich foods. Within two weeks, her serum phosphate levels normalized to 3.2 mg/dL, and her symptoms gradually resolved. The patient is currently being followed up.



Test Description	Observed Value	Units	Bio.Reference	Method
Complete Blood Count				
Hemoglobin	7.4	g/dl	12.0-15.0	Colorimetric method non cyanide(automated)
Haematocit(PCV)	27.7	%	36.0 -46.0	Calculated method(Automated)
RBC Count	4.23	Millions/cumm	3.8-4.8	Sheath Fluid Impedence
MCV	65.5	fl	83-101	Calculated method(Automated)
MCH	17.5	pg	27-32	Calculated
MCHC	26.7	%	31.5- 34.5	Calculated
Total WBC Count	8.00	X10^9/L	4.0-10.0	Laser Flometry Cytometry
Differential Count				
Neutrophils	67	%	20-80	Laser Flow Cytometry(Automated)/Manual
Lymphocytes	25	%	22-44	Laser flow cytometry(Automated)/Manual
Monocytes	05	%	2-10	laser flow cytometry(Automated)/Manual

Figure 1 : Showing decreased haemoglobin



PERIPHERAL SMEAR REPORT

RBC's	: Microcytic hypochromic RBCs seen with anisopoikilocytosis showing elongated cells, tear drop cells, target cells and polychromatophils.
WBC's	: Normal in number, morphology and distribution.
PLATELETS	: Adequate. Few large platelets seen.
No Haemoparasites seen. No immature cells.	
IMPRESSION	: MICROCYTIC HYPOCHROMIC ANEMIA.

Figure 2 : Perpheral smear shows microcytic hypochromic anemia

SERUM	FERRITIN	6.95	ng/dl	Child : 7-140 ng/ml Adult Male : 20-250 ng/ml Female :10-120 ng/ml	CLIA

Figure 3: Decrease serum ferritin

Discussion

This case involves a patient who developed symptomatic hypophosphatemia within two weeks of receiving intravenous ferric carboxymaltose for severe iron deficiency. Ferric carboxymaltose infusion therapy leads to the renal wasting of phosphate. It's been proposed that this iron compound elevates serum levels of fibroblast growth factor 23 (FGF23) by inhibiting FGF23 degradation. FGF23 is a peptide secreted by both osteoclasts and osteoblasts in response to increased phosphate levels. Its role is to regulate phosphate reabsorption in the renal tubules, maintaining phosphate balance. Consequently, elevated serum FGF23 levels result in hypophosphatemia by inappropriately reducing phosphate reabsorption in the proximal renal tubules, even when serum phosphate levels remain low. The clinical signs of elevated FGF23 levels and subsequent hypophosphatemia vary and are commonly observed in patients with genetic defects affecting phosphate regulation and those with tumor-induced osteomalacia. Weakness in proximal muscles, dental issues, bone pain, and osteomalacia are typical symptoms of these conditions. Treatment should be tailored based on the severity and clinical presentation of hypophosphatemia, utilizing either oral or intravenous preparations.

Conclusion

Hypophosphatemia may develop after intravenous iron infusion and should be kept in mind when patients exhibit corresponding symptoms. Healthcare providers should regularly check phosphate levels in patients undergoing intravenous iron therapy, particularly those with a predisposition to hypophosphatemia. Prompt

identification and proper treatment are crucial to averting complications linked with hypophosphatemia.

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