Case Report

Abrupt total iron binding capacity elevation in microcytic hypochromic anemia

Konda Siva Krishna[1], Yenduri Hari Krishna Vignesh[2], Shaik Abdul Shukoor Waseem[2], Aravapalli Sravan Kumar[2], Satheesh S Gottipatti[3], Sirnivas Babu Puttagunta[4]

From [1]Assistant Professor, Department of Pharmacy Practice, [2]Student, Department of Pharmacy, [3]Dean of Academics, Department of Pharm d, [4]Principal, Department of Pharmaceutics, Vignan Pharmacy Collage, Guntur, Andhra Pradesh, India

ABSTRACT

Iron deficiency is a leading global cause of anemia, responsible for approximately 841,000 deaths annually, with Africa and parts of Asia bearing 71% of the mortality burden. Key risk factors include heavy menstrual bleeding, pregnancy, and the post-partum period. Management typically involves oral iron supplementation, dietary changes, and intravenous iron therapy for severe cases. This report presents the case of a 48-year-old woman diagnosed with iron deficiency anemia and Vitamin B12 deficiency. She exhibited microcytic hypochromic anemia confirmed by a peripheral smear, which revealed characteristic red blood cell morphology. The case underscores the importance of early detection and management of iron and Vitamin B12 deficiencies to prevent complications. It also highlights the need to address healthcare disparities that limit access to diagnostic and treatment options in underserved regions.

Key words: Hemoglobin, Microcytic hypochromic anemia, Morphology, Total iron binding capacity

Anemia is characterized by a reduction in the circulating red blood cell (RBC) mass below normal levels, making it a widespread condition globally. RBCs contain hemoglobin (Hgb), a protein composed of four polypeptide chains and a heme ring with iron in its reduced form, essential for oxygen transport. Anemia often results from insufficient dietary iron intake or poor absorption, impairing Hgb synthesis and reducing the oxygen-carrying capacity of RBCs [1]. Microcytic hypochromic anemia is a common hematological disorder characterized by reduced RBC size and Hgb content. It is often associated with iron deficiency, chronic diseases, or genetic disorders such as thalassemia. Long-term untreated or poorly managed anemia can lead to severe complications, significantly affecting an individual's quality of life.

This case report presents a woman with a long-standing history of microcytic hypochromic anemia, highlighting the progression of her condition and the impact of treatment adherence on disease management.

CASE REPORT

A 48-year-old woman complained of fatigue, pallor, shortness of breath, and tachycardia. Lung auscultation reveals the crackles sound and there are no signs of splenomegaly or hepatomegaly on abdominal palpitations.

The patient was referred to a physician, where the routine clinical examination of complete blood picture and further investigation reveals abnormal low values (Table 1). All the primary and secondary investigation reveals the confirmatory diagnosis of microcytic hypochromic anemia with idiopathic abnormally increased total iron binding capacity (TIBC), with decreased liver functioning.

The patient was treated with 2 units of blood transfusion, further maintenance therapy with methylcarbylamine 1,500 mcg for every 3 days a week for 6 weeks ferrous ascorbate equivalent to elemental iron 100 mg folic acid 500 mcg, and zinc sulphate 61.8 mg (LIVOGEN -XT) 2 times a day for 30 days and DEXORANGE syrup 10 mL twice in a day. They further suggested checking of complete blood count (CBC) every month for 1 year.

DISCUSSION

Anemia develops due to a complex interplay of nutritional, environmental, and genetic factors. The intermediate determinants, such as food insecurity, inadequate childcare, limited healthcare access, poor nutrition education, and lack of sanitation, contribute to the onset of anemia. Insufficient dietary intake of essential nutrients, such as iron, Vitamin B12, and folate,

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Correspondence to: Konda Siva Krishna, Vignan Pharmacy College Vadlamudi Post, Chebrolu Mandal, Guntur – 522213, Andhra Pradesh, India. E-mail: kondasivakrishna999@gmail.com

Table 1: Blood investigations of the patient

Name of investigation	Results
Hemoglobin	5.6 g/dL
MCV	61.5 fL
MCH	18.3 pg
MCHC	29.7 g/dL
Red cell width	21.3%
Vitamin B 12	<100.0 pg/mL
Serum iron	20.4 µg/dL
Reticulocyte count	3.5%
Bilirubin total	1.1 mg/dL
Bilirubin direct	0.4 mg/dL
AST	13.0
Total iron binding capacity	457.0 µg/dL

MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, AST: Aspartate aminotransferase

leads to impaired RBC production. In addition, chronic illnesses and infections trigger inflammatory responses that interfere with iron metabolism and erythropoiesis. Poor access to healthcare services further exacerbates the condition by delaying diagnosis and treatment, increasing the risk of severe anemia over time [1]. The immediate determinants directly impact RBC production and survival, ultimately leading to anemia. Nutritional deficiencies, chronic inflammation, and genetic Hgb disorders such as sickle cell disease and thalassemia result in ineffective erythropoiesis and increased RBC destruction. These factors lead to either decreased RBC production or accelerated loss, significantly lowering Hgb levels. Addressing anemia requires a multifaceted approach, including improved nutritional intake, better healthcare accessibility, and enhanced public awareness to mitigate both the underlying causes and their long-term consequences.

Nutritional anemia can result from various micronutrient deficiencies, primarily iron deficiency, as well as inadequate levels of essential vitamins and minerals. Key vitamin deficiencies associated with anemia include Vitamin A, riboflavin (B2), pyridoxine (B6), folate (B9), cobalamin (B12), Vitamin C, Vitamin D, and Vitamin E. The common causes of nutritional anemia include low dietary intake of these micronutrients, poor bioavailability, or the presence of absorption inhibitors in the diet, increased nutritional requirements during periods of rapid growth such as infancy, adolescence, and pregnancy, and excessive nutrient losses. Addressing these factors through proper dietary intake and supplementation can help prevent and manage nutritional anemia effectively [2].

Rapid growth in infancy or adolescence, pregnancy, erythropoietin (EPO) therapy, chronic blood loss, menses, acute blood loss, blood donation, phlebotomy as a treatment for polycythemia vera, Inadequate diet, malabsorption from disease (sprue, Crohn's disease), malabsorption from surgery (gastrectomy and some forms of bariatric surgery), acute or chronic inflammation [3].

Laboratory iron studies include TIBC with a normal value of 300–360 µg/dL. The normal value of serum iron and serum ferritin ranges from 50–150 µg/dL to 15–30 µg/dL, respectively [4]. Red cell protoporphyrin levels are an intermediate molecule in the pathway of heme synthesis, whenever there is an impairment in heme synthesis, this gets accumulated in the red cells. The normal value is 30 µg/dL and in iron deficiency, the value increases to 100 µg/dL. Transferrin receptor protein levels in serum with a normal value of 4–9 µg/L [5].

The differential diagnosis of anemia by mean corpuscular volume (MCV) categorizes anemia into microcytic (MCV <80), normocytic (MCV 80–100), and macrocytic (MCV >95 or 100). For microcytic anemia, potential causes include iron deficiency, thalassemia, chronic inflammation, and rare causes. If the MCV is <80, check iron levels, TIBC, ferritin, and transferrin saturation. "Classic" iron deficiency is indicated by low iron, high TIBC, transferrin saturation below 20%, ferritin below 15 or 20, high red cell distribution width (RDW), elevated platelets, and high soluble transferrin receptor. If iron deficiency is not the cause, check EPO levels and consider Hgb electrophoresis.

For normocytic anemia, possible causes are acute bleeding, hemolysis, chronic inflammation, chronic kidney disease, and concomitant micro- and macrocytic conditions. In cases of normocytic anemia (MCV 80–100), a reticulocyte count should be performed. Macrocytic anemia can be caused by Vitamin B12/folate deficiency, drugs, alcohol, liver/thyroid disease, myelodysplastic syndrome (MDS), reticulocytosis, or lab artifacts. For macrocytic anemia (MCV >95 or 100), inquire about alcohol use and check RDW, B12, folate, thyroid-stimulating hormone, peripheral smear, and liver function tests. Round cells may indicate liver or thyroid issues, oval cells suggest B12/folate deficiency, and normal cells may point to a lab artifact. If the workup is negative, consider a bone marrow biopsy to rule out MDS. Anemia is defined as Hgb levels below 12 g/dL in females and below 13 g/dL in males. A CBC should be performed, focusing on MCV, RDW, and assessing other cell lines [6].

Symptoms of anemia can manifest in various ways throughout the body. Central symptoms include fatigue, dizziness, and fainting. The eyes may exhibit yellowing, while the skin might appear pale, cold, or yellow. Respiratory symptoms can include shortness of breath. Muscular weakness may also be present. Changes in stool color can indicate intestinal issues. Anemia can affect blood vessels, leading to low blood pressure. Heart-related symptoms can include palpitations, a rapid heart rate, chest pain, angina, and, in severe cases, heart attacks. The spleen may also become enlarged. In severe cases of anemia, some symptoms, such as chest pain, angina, and heart attack, are highlighted in red [6].

The treatment must focus on the cause of iron deficiency. For most of the causes, such as pregnant women, growing children and adolescents, patients with infrequent episodes of bleeding, and those with inadequate dietary intake of iron, oral iron therapy will be sufficient. After identifying the causes and obtaining the diagnosis, the therapy will start. The therapy includes red cell transfusion, oral iron replacement therapy, and parenteral iron therapy [7].

Oral Iron Replacement Therapy

Indications

Indications for intravenous (IV) ferric carboxymaltose administration include intolerance or non-compliance with oral iron, malabsorption, celiac disease with insufficient absorption, history of certain surgeries (gastrectomy, gastrojejunostomy, and/or bariatric surgery), clinically active inflammatory bowel disease, unresolved bleeding, pregnancy with Hgb levels below 10.0 g/dL, end-stage renal disease anemia treated with EPO, and moderate to severe anemia (Hb 8.0–10.9 g/dL or Hb <8.0 g/dL, respectively) with significant symptoms. The dosage for IV ferric carboxymaltose is 1,000–2,000 mg elemental iron. IV iron replacement is indicated when oral iron is not tolerated or absorbed adequately. It is also used in conditions like inflammatory bowel disease, where iron absorption may be impaired. In addition, it is used in pregnant women with low Hgb levels and in patients with end-stage renal disease [8].

Parenteral iron therapy

Indications

The primary clinical indications for IV iron therapy include intolerance or non-compliance to oral iron supplementation. IV iron is also recommended in cases of acquired or hereditary disorders that impair intestinal iron absorption or hinder iron release from macrophages. It is particularly indicated for patients with severe iron deficiency anemia, characterized by Hgb levels below 9 g/dL, especially when caused by ongoing or uncontrolled blood loss or increased iron demands. Functional iron deficiency, notably when associated with the use of erythropoiesis-stimulating agents, is another important indication [9].

CONCLUSION

This case emphasizes the importance of early intervention, continuous monitoring, and adherence to treatment in preventing the progression of chronic anemia.

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REFERENCES

1. Abd-Elmawgood EA, Hassan MH, Mobark DH, Rashwan NI. Iron- refractory Iron deficiency anemia: Review article. SVU Int J Med Sci 2024;7:9-17.
2. Kawabata H. Diagnosis and treatment of iron deficiency anemia. Rinsho Ketsueki 2024;65:503-13.
3. Patel DR, Patne AB, Sharma M, Vidja PJ. Prevalence and hematological patterns of geriatric anemia: A prospective analysis. CME J Geriatr Med 2024;16:45-8.
4. Bhasin A, Rao MY. Characteristics of anemia in elderly: A hospital based study in South India. Indian J Hematol Blood Transfus 2011;27:26-32.
5. Kilungu AM, Thadei B, Nganga M. A rare case of Iron deficiency anemia manifesting with xylophagia. Tanzan J Health Res 2025;25:1911-4.
6. Sharma P, Bhatia P, Singh M, Das R, Attri SV, Ghara N, *et al*. Genomics of iron refractory iron deficiency anemia phenotype reveals a spectrum of novel pathogenic biallelic and monoallelic TMPRSS6 variants and rare overlapping disorders. Gene 2024;895:147981.
7. Naidoo KL, Munian A, Neethling B, Rapiti N. Identification and management of iron deficiency anaemia in hospitalised children in Durban, South Africa. South Afr Med J 2025;115:22-8.
8. Aroor S, Mundkur SC, Kumar S, Handattu K, Samuel PC. Refractory microcytic hypochromic anemia with type I diabetes mellitus and reversible cardiac hemochromatosis in congenital hypotransferrinemia. Pediatr Hematol Oncol J 2024;9:37-9.
9. Gupta A. Anemia of chronic disorders. In: Decision Making Through Problem Based Learning in Hematology: A Step-by-Step Approach in Patients with Anemia. Berlin: Springer Nature; 2024. p. 63-75.

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