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Perspective

Role of Ferritin and Hemoglobin Markers in Management of Hemolytic Anemia

Nancy Faeser*

Department of Health Science, University of Goethe, Frankfurt, Germany

ABOUT THE STUDY

The premature lysis of red blood cells characterizes hemolytic anemia, which can be chronic or fatal. Any normocytic or macrocytic anemia should include it in the differential diagnosis. In the reticuloendothelial system, hemolysis can occur intravenously, extravascularly, or both. Poor deformability leads to trapping and phagocytosis, antibody-mediated destruction *via* phagocytosis or direct complement activation, fragmentation due to microthrombi or direct mechanical trauma, oxidation, or direct cellular destruction. Acute anemia, jaundice, hematuria, dyspnea, exhaustion, tachycardia, and potentially hypotension are symptoms that patients with hemolysis may exhibit. Reticulocytosis, elevated lactate dehydrogenase, elevated amounts of unconjugated bilirubin, and decreased levels of haptoglobin are all laboratory test results that indicate hemolysis. By further separating immune from nonimmune causes, the direct antiglobulin test. To find aberrant red blood cell morphologies when hemolysis is present, a peripheral blood smear should be done. Hemolytic disorders are divided into extrinsic nonimmune causes, immune-mediated anemia's, immune-mediated hemolytic illnesses, membranopathies, enzymopathies, and hemoglobinopathies. Infections, systemic disorders, direct trauma, thrombotic microangiopathies, and oxidative assaults are examples of extrinsic nonimmune causes. Numerous drug interactions might result in hemolytic anemia. A hemolytic anemia should be taken into consideration if anemia develops suddenly or if there is considerable hyperbilirubinemia during the newborn period.

Hemoglobin marker

In hemolytic disorders, haemoglobin is the best marker of clinical severity. When the condition is mild (Hb>10 g/dL), its levels may be near to normal or significantly lower when it is moderate (Hb 8-10 g/dL), severe (Hb 6-8 g/dL), or extremely severe (Hb 6 g/dL). Hemoglobin values at diagnosis were the most significant predictor of prognosis, related with the risk of death and the need for multiple therapy lines, in a recent large retrospective study of 308 cases of primary Autoimmune Hemolytic Aemia (AIHA). In RBC enzymopathies involving the Pentose Phosphate (PP) shunt, such as Glucose-6-Phosphate-Dehydrogenase (G6PD) deficit, as well as in autoimmune hemolytic forms involving complement activation and Paroxysmal Nocturnal Hemoglobinuria (PNH), an acute onset is more frequently occured.

Ferritin marker

Ferritin is an intracellular protein that stores iron and releases it when needed, serving as a buffer against iron deficiency and overload. It could serve as a proximate indicator of the quantity of iron present throughout the body. Numerous chronic hemolytic diseases, including congenital dyserythropoietic anemia, chronic cold agglutinin disease, membrane abnormalities and enzymopathies, cause a rise in ferritin levels. It has been postulated that anemia itself is a potent stimulation for iron absorption in the stomach, and that iron produced by inadequate erythropoiesis and extravascular hemolysis is not easily removed, despite the fact that the particular mechanism causing its rise has not been examined. When receiving eculizumab therapy, patients with PNH may exhibit either higher ferritin values due to ongoing extravascular hemolysis (personal observation) or decreased ferritin values due to hemosiderinuria and iron loss. Acute phase protein ferritin is elevated in a number of metabolic and inflammatory disorders (e.g., chronic and acute infections, hepatitis, and tumors). Therefore, having both of these disorders present at the same time as chronic hemolysis may result in elevated ferritin levels. Additionally, iron excess is seen in patients with hereditary hemochromatosis and after transfusions.

CONCLUSION

It may be necessary to perform immediate interventions, such as blood transfusions, plasmapheresis, or diuresis, depending on how serious the condition is. If there is active bleeding, blood transfusions are always the core of treatment for severe anemia.

Correspondence to: Nancy Faeser, Department of Health Science, University of Goethe, Frankfurt, Germany, E-mail: faesergoethe@edu.com

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But depending on the underlying cause, the treatment will always differ. If the underlying reason is originally unknown, a direct antiglobulin (Coombs) test can be utilized to distinguish between an immunological and non-immune cause of hemolysis.