Anemias

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KEYWORDS

- Anemia • Hemoglobin • Hematocrit • Autoimmune hemolytic anemia (AIHA)
- Sickle cell anemia (SCA) • Sickle cell disease (SCD) • Iron deficiency • B_{12} deficiency

KEY POINTS

- Anemias present significant expense and challenges to the US health care system.
- The focus of future research is primarily genetic research and associated risk factors that predispose patients to anemias.
- It is imperative that health care providers are made aware of various types of anemia and the appropriate management options.
- Research is ongoing regarding various aspects of anemia.

INTRODUCTION

The healthcare industry in the United States (US) is faced with many complex illnesses. Among those complex illnesses exist a variety of anemias. Anemia is a condition characterized by a decreased number of circulating red blood cells (RBCs) and/or hemoglobin. This article discusses various types of anemia, clinical characteristics, and evidence-based management strategies.

Erythrocytes (RBCs) facilitate circulation of oxygen from the lungs to vital organs. The average, healthy adult needs a large number of RBCs to fulfill this role, approximately 5 million RBCs per microliter of blood. Anemia can be defined as either a reduced number of circulating RBCs, reduced hemoglobin concentration, or reduced hematocrit (HCT).[1] Anemia is commonly found worldwide, resulting from numerous causes.

This article provides an overview of RBC production, definitions of anemia, clinical presentation, appropriate diagnostic laboratory evaluation, and methods utilized to determine causes of anemia. Anemia is a complex subject matter, however, the article focuses on 4 common types of anemia: (1) iron-deficient anemia, (2) vitamin B_{12}–deficient anemia, (3) sickle cell anemia (SCA), and (4) hemolytic anemia.

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RBC PRODUCTION

The production of RBCs in the bone marrow (BM) is stimulated by a hormone released from the kidneys called erythropoietin (EPO). While in the BM, RBCs must grow and differentiate from erythroid progenitors into reticulocytes and eventually into mature RBCs. Contained within each RBC is a protein that is able to link to oxygen and then release it in tissue capillaries. RBCs circulate in the blood for approximately 120 days and are then removed from circulation by macrophages.[1]

In order to maintain a balance between production and loss of RBCs, in a steady state the BM must produce 50,000 reticulocytes/μL of whole blood each day.[1] However, in the event of an increased loss of blood (eg, acute hemorrhage, gastrointestinal [GI] bleed, or hemolytic anemia), and the bone marrow does not effectively compensate with increased RBC production to meet the demands, the patient will inevitably develop anemia.

CATEGORIZING ANEMIA

The explanations for anemia are numerous; therefore, a methodological approach for determining the cause of anemia should be considered by clinicians, especially in complicated cases of anemia. Two approaches are utilized in determining the causality of anemia: the kinetic approach and the morphologic approach.

KINETIC APPROACH

The kinetic approach examines the causative mechanism that produces the decrease in hemoglobin and is divided into 3 categories: (1) increased loss of RBCs, (2) increased destruction of RBCs, and (3) decreased production of RBCs.[2]

Increased loss of RBCs may result from a chronic or acute hemorrhage. Blood loss can be obvious in cases like traumatic injuries, heavy menstruation, hematemesis, gross hematuria, or melena stools. However, occult blood loss is less obvious in cases such as slow-bleeding ulcers or GI cancers and can only be detected with further diagnostic testing.

Causes of increased destruction of RBCs include; hemolytic anemias that are either inherited (eg, sickle cell disease [SCD], thalassemia) or acquired (eg, autoimmune hemolytic anemia, malaria, splenomegaly, hemolytic uremic syndrome) (**Table 1**).

Decreased production of RBCs can be caused by lack of essential vitamins or hormones needed for normal erythropoiesis or by bone marrow disease. Iron, vitamin B_{12}, and folic acid deficiencies are classic examples of nutrient deficiencies that cause anemia. Hormones are also important in regulating hematopoiesis. Patients with chronic renal failure can develop anemia if the kidneys do not produce the erythropoietin levels that are needed to increase erythropoiesis (see **Table 1**). Other hormone deficiencies, such as hypothyroidism and hypogonadism, can also lead to anemia.[3] Decreased RBC production may also result from bone marrow suppression from chemotherapy, radiation therapy, or from disease within the bone marrow (eg, myelodysplastic syndrome, aplastic anemia, tumor infiltration).[4]

MORPHOLOGIC APPROACH

The morphologic approach categorizes anemia by RBC size using the mean corpuscular volume (MCV). The morphologic approach has 3 categories: (1) microcytic anemia, in which the MCV is less than 80 fL; (2) normocytic anemia, in which the MCV is within the normal range of 80 to100 fL; and (3) macrocytic anemia, in which the MCV is greater than 100 fL (**Table 2**).[5]