Anemia

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KEYWORDS

• Anemia • Emergency Department • Evaluation • Management

KEY POINTS

- Patients with anemia are frequently encountered in the emergency department, and emergency physicians often play an important role in the evaluation and management of anemia.
- After diagnosing anemia based on a low hemoglobin, hematocrit, or red blood cell (RBC) count, the RBC indices and peripheral smear should be evaluated.
- The initial treatment of anemia depends on the clinical status of patients.
- The decision to initiate blood transfusion is not always straightforward, and it is not a decision that should be taken lightly.

INTRODUCTION

Patients with anemia are frequently encountered in the emergency department (ED), and emergency physicians (EPs) often play an important role in the evaluation and management of anemia. Some of these patients may have chief complaints directly related to their anemia, and others may be asymptomatic. Although many patients have findings consistent with anemia on routine laboratory tests, only a small percentage will require acute intervention. An understanding of the broader types of anemia as well as how to manage such patients is important in the day-to-day practice of an EP, as the presence of anemia will impact treatment plans for a wide variety of other disorders. This article reviews the evaluation and management of adult patients presenting to the ED with anemia.

BACKGROUND

Definition

Anemia is defined as a condition in which the body has a decreased amount of circulating erythrocytes, or red blood cells (RBCs). It can also be defined as a decreased hemoglobin concentration or RBC mass compared with age-matched controls.[1] As

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with almost all human laboratory assays, *normal value* is a statistical term used to define a range within which 95% of the population's values fall.[2] The World Health Organization (WHO) defines anemia as a hemoglobin less than 13 g/dL in adult men and less than 12 g/dL in non-pregnant adult women.[3] However, these values were chosen somewhat arbitrarily; most laboratories define anemia as the lowest 2.5% of the distribution of hemoglobin values from a normal, healthy population.[4]

Anatomy

Erythropoiesis

Erythrocytes originate in the bone marrow as hematopoietic progenitor and precursor cells. After several cell divisions, mature RBCs emerge as discoid, pliable anucleate cells, each containing 4 hemoglobin molecules. An erythrocyte typically survives for 100 to 120 days before undergoing apoptosis (programmed cell death).[5] Erythropoiesis, or the process of RBC production, occurs in a regulated fashion under the control of the hormone erythropoietin (EPO). EPO is a glycoprotein, secreted from peritubular cells within the kidney when renal cells detect decreased oxygen in circulation available for metabolism.[1,6] Successful erythropoiesis depends on 4 factors: a stimulus for erythrocyte production, the ability of precursor cells in the bone marrow to respond to the stimulus, the presence of essential nutrients required for erythrocyte synthesis, and the life span of the erythrocyte.[7]

Erythropoiesis should be stimulated in response to most forms of anemia, but it takes 3 to 7 days for new RBCs to appear in the blood.[5]

Hemoglobin

Hemoglobin is a tetramer made up of 2 pairs of polypeptide (globin) chains, with each chain containing an iron-containing heme complex for oxygen binding. The structure of hemoglobin is under both genetic and environmental influence.[4]

Various forms of hemoglobin are known to exist. In adults, hemoglobin A and A2 are the major and minor forms of hemoglobin, respectively. Hemoglobin F, present in utero, should make up less than 1% to 2% of adult circulating hemoglobin but may be present in higher quantities in the setting of other hemoglobin variants.

Under genetic influence, other forms of hemoglobin may make up the minority or most of the circulating hemoglobin, affecting the overall RBC oxygen-carrying capacity. Hemoglobin S is the predominant hemoglobin in sickle cell disease. Other hemoglobin variants also include hemoglobin C and E as well as thalassemia.[4] Hemoglobin variants generally have altered oxygen affinity, a shorter life span, and are more unstable leading to increased hemolysis.

Production abnormalities

Abnormalities in the production of erythrocytes can be caused by insufficient cofactors, such as vitamin B12 and folate, or can be caused by genetic abnormalities, such as congenital hemoglobinopathies or membranopathies. *Hemoglobinopathies* are abnormalities within the globin chains, as described earlier. *Membranopathies* are abnormalities in the membrane of the RBC; hereditary spherocytosis and elliptocytosis are 2 examples.

Cause

Acute anemia

Anemia can be classified in several different ways. For the EP, the most important initial questions for classification is whether the anemia is acute or chronic. This classification can be identified based on clinical presentation as well as laboratory investigations. In the ED, the common causes of acute anemia include hemorrhage