Acute Leukemia

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KEYWORDS

• Acute leukemia • Acute myelogenous leukemia • Acute lymphoblastic leukemia
• Emergency providers

KEY POINTS

- To obtain a basic understanding of the pathophysiology, classification, and treatment of acute leukemias.
- To integrate an understanding of the epidemiology and myriad presentations of acute leukemia.
- To learn to recognize and treat the life-threatening presentations of acute leukemia in the emergency department (ED) setting.
- To know the common complications of treatment and emergent management of these complications.

INTRODUCTION

The American Cancer Society estimated that 13,800 cases of acute myelogenous leukemia (AML) and 6000 cases of acute lymphoblastic leukemia (ALL) were diagnosed in the United States in 2012.[1,2] Like many other chronic diseases, emergency providers (EPs) increasingly are treating more patients who have or previously had an acute leukemia. Furthermore, the life-threatening complications and complexities of its treatment make an understanding of leukemia essential. Physician-related delays in diagnosis of leukemia have been shown to contribute to poor outcomes and higher mortality associated with the disease in low-income nations.[3] In developed countries, delay in diagnosis of pediatric cancers is a leading cause of malpractice claims.[4] But what large determinant studies have shown is that children who present to an ED with symptoms of leukemia have less delay in treatment initiation than those who first present to their general practitioner.[5,6] Understanding leukemia and its complications in the ED will improve patient care and outcomes.

PATHOPHYSIOLOGY

Acute leukemia results from a series of mutational events that take place during the complex process of hematopoiesis. All pluripotent cells in the bone marrow proliferate

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into 2 major cell lineages: the myeloid cells, which include granulocytes, erythrocytes, megakaryocytes, and monocytes; and the lymphoid cells, which include the B- and T-lymphocytes (Fig. 1). Myeloid cells proliferate into their mature end cells within the bone marrow, whereas the lymphoid precursors migrate to the lymphoid organs (eg, lymph nodes, spleen, and thymus) to complete maturation. Although a detailed discussion of pathophysiology and genetics of leukemia is beyond the scope of this article, EPs should know that both AML and ALL arise from multiple genetic mutations that allow both unchecked proliferation and abnormal maturation. This preferential multiplication of leukemic cells leads to decreased production of normal cells.[7] Leukemias are most commonly diagnosed on a smear of peripheral blood demonstrating the abnormal leukocytes.[8] Mutations can lead to abnormality in any step in the cell maturation process, which is why leukemia, especially the myeloid type, is such a

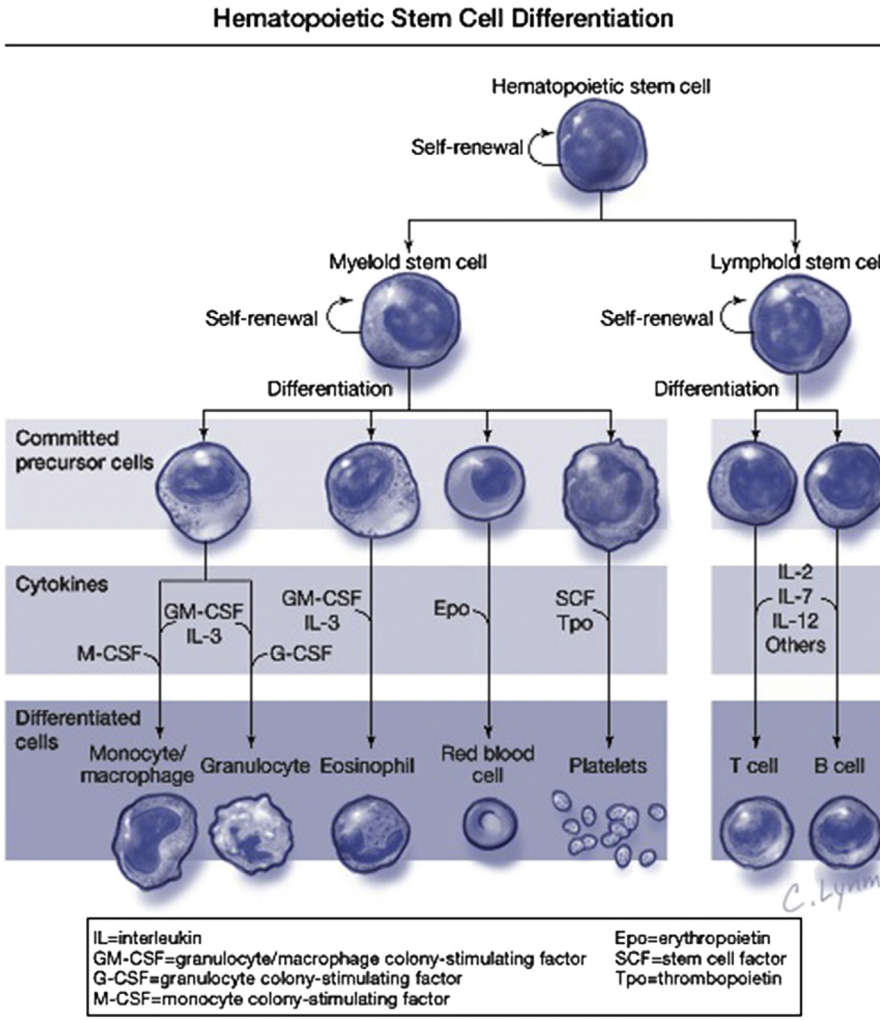

Fig. 1. Normal hematopoeisis. (*From* Cassio Lynn for the Albert Lasker Award for Basic Medical Research 2005. Available at: http://www.cassio-lynm.com. Accessed December 16, 2013; with permission.)