

Leukostasis

Management to prevent crisis in acute leukemia

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BACKGROUND: Hyperleukocytosis, a peripheral white blood cell count greater than $100,000/\text{mm}^3$, is most commonly seen in patients with newly diagnosed or relapsed acute lymphoblastic leukemia and acute myeloid leukemia. Leukostasis is a reduction in blood flow related to hyperviscosity. Hyperleukocytosis, causing leukostasis, is an oncologic emergency and requires an exacting assessment and rapid response with appropriate intervention to prevent morbidity and mortality in the first week after diagnosis.

OBJECTIVES: The objectives of this article are to equip oncology nurse to identify patients with hyperleukocytosis and to provide nursing interventions that will ensure safe, quality care.

METHODS: A case study is used to demonstrate key concepts that are critical in early assessment, identification, and treatment of patients with leukostasis.

FINDINGS: Oncology nurses well versed in the pathophysiology, clinical presentation, and management of leukostasis can make a significant contribution to the safe management of patients with cancer.

KEYWORDS

hyperleukocytosis; leukostasis; acute leukemia; leukapheresis; hyperviscosity

DIGITAL OBJECT IDENTIFIER

10.1188/17.CJON.E267-E271

HYPERLEUKOCYTOSIS IS A PERIPHERAL WHITE BLOOD CELL (WBC) COUNT greater than $100,000/\text{mm}^3$ and is most commonly seen in patients with hematologic malignancies, particularly acute lymphoblastic leukemia and acute myeloid leukemia (Ruggiero, Rizzo, Amato, & Riccardi, 2016). In untreated acute myeloid leukemia, 5%–20% of patients present with hyperleukocytosis (Villgran et al., 2016). This laboratory abnormality is caused by leukemic cell proliferation (Rölliig & Ehninger, 2015). Hyperleukocytosis is an oncologic emergency, and its management mandates intensive supportive care and interventions for rapid cytoreduction.

Leukostasis is a reduction in blood flow related to hyperviscosity from hyperleukocytosis. Two main theories have been proposed to explain the pathophysiology of leukostasis in acute leukemia. The first centers on the idea that with a higher than normal degree of viscosity, stasis can occur in microvasculature, resulting in organ damage (Ali, Mirrakhimov, Abboud, & Cashen, 2016; Jain, Bansal, & Marwaha, 2013; Ruggiero et al., 2016; Schiffer, 2016; Shiber & Fines, 2011; Stucki et al., 2001). If the leukocrit, or fractional volume of leukocytes, is greater than 12–15 ml/dl, a significant rise in blood viscosity can occur (Ali et al., 2016). The leukocrit is the volume percentage of leukocytes in whole blood, which is twice as high in leukemic myeloblasts than in the leukemic lymphoblasts. This may be one reason for a higher incidence of leukostasis in acute myeloid leukemia than in acute lymphoblastic leukemia (Ali et al., 2016; Ruggiero et al., 2016; Shiber & Fines, 2011). The large myeloblasts that create sludge in smaller vessels also lead to organ and vascular damage (Jain et al., 2013). Leukemic blasts are less deformable than mature leukocytes, which may account for the higher prevalence of leukostasis in acute leukemias compared to chronic leukemias (Ali et al., 2016).

The second theory involves the adhesion properties of cells. Under certain circumstances, leukemic cells can promote their own adhesion to endothelium. Stucki et al. (2001) discovered that when leukemic cells secrete cytokines, which change the adhesion molecule activation on the endothelial cells, the cells can regulate their own adhesion to endothelium. Blast-secreted cytokines can worsen leukostasis. Leukostasis can be a result of the adhesive interactions that occur when damaged endothelium is present in the blood vessel and leukemic blasts (Ali et al., 2016; Jain et al., 2013; Ruggiero et al., 2016; Stucki et al., 2001).

Clinical Presentation

Patients with leukemia and a WBC count of greater than $100,000/\text{mm}^3$ are diagnosed with hyperleukocytosis. Some of these patients will not develop leukostasis. Clinical symptoms associated with this laboratory value indicate