Advances in Myelodysplastic Syndrome: Nursing Implications of Azacitidine

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Myelodysplastic syndrome (MDS) is a group of clonal hematopoietic stem cell disorders characterized by ineffective hematopoiesis, leading to bone marrow failure and peripheral blood cytopenias. MDS is difficult to diagnose because of the absence of symptoms in the early stage of the disease; it often is discovered accidentally during routine physical examinations or blood tests. The U.S. Food and Drug Administration approved azacitidine (Vidaza®, Pharmion Corporation, Boulder, CO) for the treatment of MDS. Prior to the approval of azacitidine, no approved therapies were available for the treatment of MDS. Azacitidine is believed to exert its anticancer effects by induction of hypomethylation and cytotoxicity. In clinical studies, the most common adverse events during treatment with azacitidine included nausea, anemia, thrombocytopenia, vomiting, pyrexia, leukopenia, diarrhea, fatigue, injection-site erythema, constipation, neutropenia, and ecchymosis. To ensure proper treatment with azacitidine, nurses should have an understanding of dosage and administration guidelines, commonly observed adverse events, monitoring and care of adverse events, and monitoring of laboratory tests. Having a comprehensive understanding of MDS, its underlying disease characteristics, and current treatments will enable oncology nurses to provide optimal patient care.

Myelodysplastic syndrome (MDS) is a group of clonal hematopoietic stem cell disorders associated with abnormalities of cellular differentiation and maturation. It leads to varying degrees of bone marrow failure and peripheral blood cytopenias (Perry, Maghfoor, & Dorr, 1999) that contribute to potentially serious morbidity (e.g., transfusion-dependent anemia, bleeding manifestations) and mortality (e.g., death from infection with neutropenia), plus the additional risk of leukemic transformation. MDS is characterized by ineffective hematopoiesis that arises from accelerated apoptotic death of affected multipotent hematopoietic progenitor cells and their progeny (List & Kurtin, 2003). Chromosomal abnormalities involving genes that control hematopoiesis can lead to MDS (Silverman, 2001). These genetic events affect myeloid cell maturation and differentiation that can lead to alteration in cytokine regulation and response to cytokines (Silverman, 2001). Overproduction of proapoptotic cytokines may contribute to excessive apoptotic cell death in MDS (Faderl & Kantarjian, 2004). As a result, anemia, thrombocytopenia, and neutropenia can present individually or simultaneously (Cain, Hood-Barnes, & Spangler, 1991).

Several terms used in the past for MDS include preleukemia, smoldering acute leukemia, and hematopoietic dysplasia (Utley, 1996). The historical terms were somewhat erroneous because many patients died from bone marrow failure without developing leukemia (Silverman, 2003). In 1976, the French-American-British (FAB) Study Group adopted the more accurately descriptive and appropriate designation of MDS. In MDS, bone marrow loses its ability to produce normal cells and, instead, produces dysplastic cells; thus, the diseased cells are called myelodysplastic cells.

Myelodysplastic Syndrome: Clinical Findings

MDS is an umbrella term for a range of disorders that often have little in common with one another in terms of natural history, prognosis, or treatment. MDS can be primary (arising de novo) or secondary (following exposure to chemotherapeutic agents or ionizing radiation). The majority of cases (70%–80%) occur de novo, whereas 20%–30% of cases are secondary (Kantarjian & Estey, 2001). Genetic and congenital anomalies are known to contribute to primary MDS, whereas smoking and exposure to petroleum, benzene, solvents, ionizing radiation, and chemotherapy agents are factors that may increase the risk of developing secondary or treatment-related MDS (American Cancer Society, 2004).

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