

Myelodysplastic Syndromes in Older Adults

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Myelodysplastic syndromes are a collection of disorders that affect the hematopoietic development of myeloid cells in the bone marrow. Although this disorder is curable by way of allogeneic stem cell transplantation, advanced age, limited donor availability, and multiple comorbidities often exclude patients from curative treatment. Developments using the drugs lenalidomide, decitabine, and azacitidine have offered treatment options to patients ineligible for transplantation. Nurses remain instrumental in the administration, patient monitoring, and patient education associated with these new therapies.

Myelodysplastic syndrome (MDS) does not describe a single disease but rather a collection of disorders that affect the hematopoietic development of myeloid cells in the bone marrow (see Figure 1). Characterized by cytopenias and various genetic anomalies, MDS occurs most frequently in older adults. About 90% of all cases occur in people older than age 60, with 38% of all cases occurring in adults older than age 80 (Rollison et al., 2009). Once thought to be merely a preleukemic syndrome, MDS now is recognized as a serious malignancy. Although 30% of patients with MDS may develop leukemia, more will die because of MDS alone (Ososki & O'Riley, 2007). Cure is possible with an allogeneic stem cell transplantation (about 30%–50% rate) (Litzow et al., 2010); however, a patient's age or various comorbidities often preclude them from this type of intense treatment. Despite this, advancements in epigenetics, immunomodulatory agents, and other novel anticancer drugs can improve duration and quality of life.

Like many diseases, the origin of MDS often is unknown. However, those who have received chemotherapy in the past carry a significant risk for developing MDS in the future. Usually this form of MDS arises 5–10 years after initial exposure to an alkylating chemotherapy, although topoisomerase inhibitors place patients at risk for developing MDS between 1–5 years after exposure (Nguyen, 2009). Common chemotherapeutic agents that put patients at risk for MDS are melphalan, cyclophosphamide, busulfan,

chlorambucil, carboplatin, etoposide, doxorubicin, and daunorubicin (Nguyen, 2009). Patients with treatment-related MDS are more likely to be younger than the typical patient with MDS, are more likely to have comorbidities related to their previous disease, and have a poorer prognosis compared to those with de novo MDS (Nguyen, 2009).

De novo (literally meaning “anew” or “afresh”) MDS occurs in previously healthy individuals with no prior exposure to chemotherapeutic agents. Little is known about what causes MDS to arise, but research has found several common risk factors: obesity, smoking, and exposure to radiation and organic chemical solvents (Du, Fryzek, Sekeres, & Taioli, 2009; Ma et al., 2009). Although a slight increase in risk exists among people with a family history of hematopoietic cancers (Strom, Gu, Gruschus, Pierce, & Estey, 2005), MDS is not believed to be an inherited condition.

Early symptoms of MDS can be vague, but may include any symptoms related to the various cytopenias associated with MDS. The patient may experience frequent bruising, infections, or may bleed easily. The patient also may experience symptoms related to anemia, such as increased fatigability and subsequent decrease in daily activities (Nguyen, 2009). MDS also

may be, incidentally, diagnosed as part of routine blood work or as part of a workup for an unrelated medical condition (Ososki & O'Riley, 2007). This blood work would show a “presentation of an unexplained anemia, leucopenia, or thrombocytopenia in an older adult” (Stone, 2009, p. 6296).

A number of tests are required to confirm an MDS diagnosis. A complete blood count is crucial to determine the type, scope, and character of the patient's cytopenias. A manual differential examination by a pathologist looks at the type of abnormal white cells in the peripheral blood. The type and quality of these abnormal cells (i.e., blasts, ringed sideroblasts, monocytes, or dysplastic neutrophils) determines how the MDS is classified (Nguyen, 2009).

Other testing includes a bone marrow biopsy, cytogenetics, and a fluorescence in situ hybridization (known as FISH) analysis to evaluate for specific genetic abnormalities. The most common are 5q deletion, monosomy 7 or 7q deletion, trisomy 8, or JAK2 mutation (Stone, 2009). Complex cytogenetics occur when the cells have multiple chromosomal abnormalities. Additional testing is done to complement and guide treatment of MDS. This includes complete metabolic and hepatic panels. A type and cross is

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Digital Object Identifier: 10.1188/10.CJON.545-547