

On being up to date and linked in

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We are mindful of our role in providing our readers with quality research- and literature-based articles about emerging therapies and diagnostic and palliative approaches that will have a positive impact on how they practice. So far this year, we have brought you articles on current therapies for metastatic melanoma and hairy cell leukemia as well as updates on managing chronic myelogenous leukemia, the late effects of cancer therapies, and most recently, small renal tumors. In this month's issue, on page 236, we bring you yet another of these comprehensive Reviews, this time by Estephan and Tiu on therapeutic approaches in myelodysplastic syndromes.

MDS is caused by dysfunctional blood cell activity that manifests as chronic cytopenias in the form of anemia, thrombocytopenia, or neutropenia and can sometimes progress to acute myeloid leukemia (AML). It is a multifaceted condition in that it is genetically and morphologically heterogeneous and can vary significantly in its natural history and prognosis. The MDS patient population tends to be older, with most patients being diagnosed at older than 60 years and a median age of early- to mid-70s, which means there are more comorbidities to contend with in treating the disease. Needless to say, this disease complexity and clinical variability present diagnostic and therapeutic challenges for the practicing oncologist. In addition, from 2001 to 2008, the age-adjusted incidence of MDS in the United States increased from 3.6 to 4.6 cases per 100,000 population, slightly higher than that of AML, and since MDS has a better survival rate than AML and the population is aging, MDS now has the greater prevalence.

Most cases of MDS are de novo and generally easier to treat than the secondary MDS caused by chemotherapy (about 10% of cases) or those that originate from environmental exposure to chemicals such as tobacco smoke or pesticides, or metals such as lead or mercury. The authors point out that allogeneic hematopoietic cell transplantation is the only "potentially curative" treatment

for MDS, though 3 drugs – lenalidomide, 5-azacitidine, and decitabine – have been approved as MDS therapies by the US Food and Drug Administration and are effective as disease-modifying agents in relieving the cytopenias, achieving cytogenetic remission, and reducing bone marrow blasts. 5-azacitidine has also been shown to improve overall survival in some patients. Numerous factors such as patient age and comorbidities, donor availability, and patient/physician preference mean that relatively

few patients (about 5%) can benefit from the allo-HCT, so that the remaining 3 approved agents have been the more widely used therapies. Fortunately, with the identification of genetic mutations, epigenetic mechanisms, and immunoregulatory pathways, we now have a better understanding of the pathogenesis of MDS. Genetic mutations have made diagnosis easier in cases that might formerly have been more difficult to discern. Molecular mutations can also help refine the disease's risk stratification, and therefore shed light on the prognosis and possibly even the patient's response to therapy. Can much improved

patient outcomes be far behind?

If you missed going to the annual meeting of the American Society of Clinical Oncology this year, turn to page 259, for some articles by the news team for our sister publication, *THE ONCOLOGY REPORT*. On page 263, Marlana Orloff, an oncology fellow, writes about attending her first ASCO meeting and in the process provides an interesting insight into the state of our specialty at the moment. As always, remember to visit our redesigned site at www.jcso-online.com, where you can find my podcasts on the home page as well as the click through to the 2014 digital issues of *THE JOURNAL OF COMMUNITY AND SUPPORTIVE ONCOLOGY* and instructions for downloading the JCSO app. It's quick, easy, and user friendly. And remember too, that you can find all of the 2014 articles in PubMed and PDFs of our articles can be downloaded on our site.

