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New opportunities in a personalized approach to the preleukemic phase of myelodysplastic syndrome and acute myelogenous leukemia

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Although not all patients with myelodysplastic syndrome (MDS) evolve into acute myelogenous leukemia (AML), it is generally believed that these patients if they survive long enough will eventually transform and was in the past named as a preleukemic syndrome. Basic knowledge regarding the molecular mechanism of the evolution of MDS into AML, development of epigenetic and immunomodulatory agents in its management may give us opportunities of better management of the disease as well as opportunities to prevention of its evolution to a fatal condition given the right circumstances. Continued improvement in classification and prognostication by inclusion of new data including cytogenetics and molecular markers, we are now able to tailor specific treatment for subgroups of patients who share similar diagnostic labels but differ in pathogenesis as indicated by their molecular markers leading to a more specific and personal approach to their management. A specific example in MDS is the presence of a cytogenetic abnormality ie. del5q which is responsive to a specific immunomodulatory agent called lenalidomide (an analogue of thalidomide). This syndrome which for the past decade had no standard therapy that prolonged survival has now shown a doubling overall survival with azacytidine, a demethylating agent. Meanwhile in AML, specific cytogenetic abnormalities have led to a completely different approach such as using a retinoid and arsenic trioxide for acute promyelocytic leukemia (APL) with the specific cytogenetic abnormality of a translocation 15;17. We also now identified AML patients with cytogenetic abnormalities of the core binding factor such as inverted 16 as a good prognostic marker and treated specifically with standard induction and high dose cytosine arabinoside consolidation. The intermediate group in AML with normal cytogenetics is a mixture of good and bad prognostic patients and with the help of molecular markers such as FLT3/ITD and NPM markers, we are able to tease out the good from the bad and plan out a more specific approach to their management. We an also identify up front patients who will not respond to our available therapies and should be prepared early for possible hematopoietic stem cell transplantation. The latter has evolved so that we can now push this form of treatment to include some of our elderly patients using less aggressive non-myeloablative approaches and using graft versus leukemia effects to our advantage.

Biography

Emmanuel C Besa completed his MD from the University of the Philippines, College of Medicine, finished his postdoctoral studies from the University of Pennsylvania at Presbyterian Medical Center in Hematology and Oncology. He joined the faculty of the Medical College of Pennsylvania as an assistant professor and promoted to full professor in 1994 and tenure in 1995. The institution evolved into the Drexel University College of Medicine by 2004. He moved to Thomas Jefferson University as Professor of Medicine and Medical Oncology, Kimmel Cancer Center as part of the Division of Hematologic Malignancies and Hematopoietic Stem Cell Transplantation program. He was an active member as part of the Educational Committee of the American Society of Hematology and has designed case teaching which is available at the ASH website. He was awarded the MDS Center of Excellence by the MDS Foundation in 2000 at Drexel and 2005 at Jefferson. He recently retired from his academic position in June 30, 2013 but continues to conduct CME lectures and is active as the Hematology Editor of Medscape Emedicine, an online medical text which is peer reviewed.

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