Acute Myeloid Leukemia: A Classification and Treatment Update

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Of the nearly 31,000 patients diagnosed with leukemia each year in the United States, one-third are diagnosed with acute myeloid leukemia (AML) (Jemal et al., 2003). With appropriate induction therapy, 50%–70% of adult patients achieve a complete remission. About 25% of all patients remain disease free without further treatment. Currently, the ultimate prognosis of this condition is associated intimately with a variety of molecular and cytogenetic abnormalities. In the 1980s, the new identification of chromosomal aberrations associated with AML increased understanding of the pathophysiology of the condition, and different syndromes with distinct clinical manifestations and prognoses were outlined (Berger et al., 1985, 1987; Keating et al., 1987; Larson et al., 1984). Despite advances in the understanding of this disease, however, several other aspects remain unclear.

A new classification and more sophisticated treatment strategies were developed and implemented during the 1990s. The addition of retinoic acid more than doubled the survival of patients with acute promyelocytic leukemia (APL), and monoclonal antibodies and their combination with radionuclides or toxins are promising approaches in different subsets of patients (Cortes & Kantarjian, 2000; Soignet et al., 2001).

Approximately 11,000 Americans will be diagnosed with acute myeloid leukemia (AML) in 2003, and about 75% ultimately will die from the disease. Despite significant advances in understanding biologic, molecular, and cytogenetic aspects of this malignancy, several other areas remain poorly understood. During the 1990s, significant advances in the characterization of this condition have shown that AML affects elderly patients more frequently. Treatment of patients in this age group poses a greater challenge partly because of increased tumor resistance and the presence of multiple medical comorbidities that may contraindicate therapy. New therapeutic approaches are promising and have renewed enthusiasm and optimism among patients and healthcare providers. Future treatment strategies for patients with AML most likely will include combinations of biologic agents with defined molecular targets (e.g., monoclonal antibodies, retinoids, hypomethylating agents, tyrosine kinase inhibitors).

Key Words: acute myeloid leukemia, treatment, classification, new therapies

This article reviews the initial presentation, classification, prognostic factors, and current therapies available for adult patients with AML.

Case Study

Mr. S, a 36 year-old man, noticed generalized weakness in September 1980. His medical history showed only a mitral valve prolapse and no occurrence of cancer in his family. During 1973 and 1974, he was exposed to benzene in controlled environments as a professional aquaculturist. Physical findings included a wide-split second heart sound without murmurs, lymphadenopathy, hepatosplenomegaly, ecchymoses, or active bleeding. A complete blood count showed 3,600/mm³ leukocytes with 44% neutrophils, 51% lymphocytes, 3% monocytes, 2% eosinophils, and 178,000/mm³ platelets. Two months later, a few myeloid blasts were noted in his peripheral blood; a bone marrow aspiration revealed more than 30% myeloblasts, monoblasts, and promonocytes with more than 20% non-erythroid mononuclear cells consistent with acute myelomonocytic leukemia. Auer rods were present.

Mr. S was admitted to Memorial Sloan-Kettering Cancer Center (MSKCC) for induction chemotherapy with daunorubicin, cytarabine, and 6-thioguanine (DAT) in December 1980. On admission, a bone marrow aspirate revealed 45% blasts and monocytes. After a first course of induction, a

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