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

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**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 monocytoid 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