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Commentary

An Overview on Pathogenesis and Therapeutic Advancements of Acute Non-Lymphocytic Leukemia

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DESCRIPTION

Acute Myeloid Leukemia (AML) is the most common type of acute leukemia, and its incidence rises with age. Although the cause is usually unknown, it can develop after exposure to genotoxic agents or after an antecedent hematologic disorder (eg, marrow failure syndrome). The disorder begins in a malignantly transformed multipotential hematopoietic stem cell that undergoes a series of genomic alterations before manifesting clinically. AML is a remarkably complex malignancy with significant genetic, epigenetic, and phenotypic heterogeneity.

AML's molecular and cytogenetic abnormalities include mutations in critical genes involved in normal cell development, survival, proliferation, and maturation, adding to the difficulty of targeting these pathways without causing toxicity. Furthermore, multiple malignant clones coexist in almost every AML patient. Each of these subclones is distinguished by a distinct set of genetic and epigenetic abnormalities, and their responses to treatment may vary. The complexity of the molecular, cellular, and clonal structure may explain why progress in AML treatment has been extraordinarily difficult. As a result, progress in understanding the pathobiology of AML has far outpaced progress in translating our knowledge into better treatment.

With current treatments, approximately 35% to 40% of patients under the age of 60 may achieve long-term survival. However, there is a wide variation in outcome among genetically distinct subsets of the disease, with some subtypes having a notoriously poor outcome. Furthermore, the overall prognosis in older patients (>60 years) has remained highly unsatisfactory. As a result, there is a critical unmet need for therapeutic advancements. It is assumed that successful treatment must effectively eradicate the leukemic stem cell and its subclones so that residual disease cannot act as a source of recurrence. Describe the astonishing variety of gene mutations discovered in AML as drivers of disease development and progression they discuss the interrelationships between identified genetic mutations and their biological significance, focusing on the therapeutic implications. AML pathogenesis may be aided by epigenetic alterations reflecting chromatin changes that affect gene expression in addition to DNA mutations. Our current understanding of epigenetic alterations in disease pathogenesis, as well as their potential value for treatment development epigenetic changes have the potential to significantly disrupt and perturb a wide range of key functional intracellular pathways. Because epigenetic changes are potentially reversible, they appear particularly amenable to therapeutic interventions and provide appealing avenues for targeted treatment development. In AML, therapeutic progress has been slow. Current treatment is still largely based on the traditional cornerstones of combination chemotherapy and the appropriate use of stem cell transplantation. What is the current clinical management framework for an AML patient? Although the promise of a fully developed personalized treatment approach has yet to be fulfilled, clinicians require the best possible information to guide the optimal treatment approach in an individual patient. The option of an allogeneic or autologous stem cell transplant has become an integral part of the current comprehensive treatment strategy. New conditioning regimens and transplant sources, as well as donor options, have become available. What type of transplant is best for a specific patient, taking into account leukemia-specific and patient-related factors? These issues are addressed critically and clearly in terms of AML clinical management and the utility of stem cell transplantation in the front-line treatment of AML. New conditioning regimens and transplant sources, as well as donor options, have become available. What type of transplant is best for a specific patient, taking into account leukemia-specific and patient-related factors.

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