Pediatric Acute Myeloid Leukemia (AML) Classifications

Pediatrics & Therapeutics

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DESCRIPTION

Acute leukemia is the most common hematological malignancies in children, with Acute Lymphoblastic Leukemia (ALL) accounting for 80% of cases and Acute Myeloid Leukemia (AML) for 15% to 20%. AML affects 1.5 per 100,000 people annually in infants, 0.9 per 100,000 people in children ages 1-4, and 0.4 per 100,000 people in children ages 5 to 9, before progressively rising in incidence as people become older, reaching 16.2 per 100,000 people over the age of 65. AML typically develops from scratch in children, and its underlying cause is unknown. Myelodysplastic Syndrome (MDS) frequently precedes AML in adult and elderly patients, although it is uncommon for AML in children to be brought on by the clonal expansion of pre-leukemic myeloproliferative illnesses like MDS Juvenile Myelomonocytic Leukemia (JMML). AML or development as a secondary malignancy is more likely in those with germline defects, such as those with Fanconi anemia or Bloom syndrome. 15-20 percent of all juvenile acute leukemia is pediatric Acute Myeloid Leukemia (AML). Due to enhanced supportive care, greater risk stratification, and increased chemotherapy, survival rates have improved over the previous few decades to around 70%. The majority of children who develop AML do so as a de novo entity, although small percentages do so as a secondary malignancy. Due to its heterogeneity in morphology, immunophenotype, and cytogenetics, paediatric AML distinguishes a number of subtypes, including molecular alterations of the leukemic cells derived from Peripheral Blood (PB) or Bone Marrow (BM), and occasionally from solid tumor masses known as chloromas or leukemic skin infiltrations, both of which contain AML cells. The World Health Organization (WHO) classification is now used for classification, which is also based on morphology, cytochemistry, immunophenotyping, karyotyping, and molecular analysis. Pediatric AML patients who get treatment experience complete remission rates of over 90% and long-term survival rates of at least 70% (defined as 5% blasts in BM and no leukemic cells in PB or elsewhere). The escalation of Gung University, Taoyuan, Taiwan chemotherapy, the addition of novel drugs, enhanced allogeneic Stem Cell Transplantation (allo-SCT), better risk group-specific therapy, enhanced salvage upon recurrence, and developments

therapy, enhanced salvage upon recurrence, and developments in supportive care have all contributed to an increase in survival. These days, risk-group classification is based on the degree of Measurable-Residual Disease (MRD) following therapy and the cytogenetic (including molecular) abnormalities in the leukemic cells. Intensive chemotherapy with anthracyclines and cytarabine is used as part of the treatment, and in some genetically high-risk cases or delayed responders, stem cell transplantation is also used. Approximately 30% of juvenile AML patients will typically experience relapse, while 5%-10% of kids will pass either from the consequences of their disease or the adverse effects of their treatment. A comprehensive understanding of the genetic anomalies and aberrant processes involved in leukemogenesis is necessary for targeted therapy, which may improve anti-leukemic efficacy and reduce treatment-related morbidity and death. The patient's early treatment response is reflected in the MRD, and this seems to be a potent prognostic indicator. Measurement of MRD is done via DNA or RNA-based Polymerase Chain Reaction (PCR) analysis of leukemia-specific transcripts, cytometric analysis of aberrant flow multidimensional immunophenotypes, and more recently Next-Generation Sequencing (NGS).

The current survival rate for pediatric AML is around 70%, and current treatment has reached a therapeutic plateau. Due to toxicity, further treatment intensification is not possible. The multiple prognostically significant non-randomly related molecular and cytogenetic abnormalities that have been identified recently serve as an example of the heterogeneity of AML. Nevertheless, many of the contributing factors to leukemogenesis are still unknown. In the near future, the use of new tools, particularly next generation sequencing, will help us better understand the genetic landscape of AML and open the way for the creation of more focused and individualized treatments. Collaboration across nations is essential to achieving these objectives for a condition as rare as pediatric AML.

Citation: Tsung-Chieh Y (2022) Pediatric Acute Myeloid Leukemia (AML) Classifications. Pediatr Ther. S5:002.

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Commentary

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Received: 06-Jun-2022; Manuscript No. PTCR-22-18326; Editor assigned: 08-Jun-2022; PreQC. No. PTCR-22-18326 (PQ); Reviewed: 22-Jun-2022; QC. No. PTCR-22-18326; Revised: 29-Jun-2022; Manuscript No. PTCR-22-18326 (R); Published: 07-Jul-2022, DOI: 10.35841/2161-0665.22.S5.002.