HEMATOLOGY, TRANSFUSION AND CELL THERAPY

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Adult Hematology Abstract Categories

Acute Leukemias

OP 01

TREATMENT OUTCOMES AND FACTORS AFFECTING SURVIVAL IN PEDIATRIC ACUTE MYELOID LEUKEMIA

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Objective: This study aims to analyze the clinical and genetic characteristics of pediatric AML patients, evaluate treatment responses and survival under AML-BFM 2004 and 2012 protocols, identify factors affecting survival, and compare findings with international data to provide context for results. **Methodology:** In this study, 49 pediatric patients under 18 years of age diagnosed with AML at a tertiary university hospital in Turkey between January 2010 and December 2023 were retrospectively reviewed. Demographic data, clinical findings at initial diagnosis, use of leukapheresis, administration of cytoreductive therapy prior to induction, induction response, relapse status, outcomes of stem cell transplantation, and current status (alive/deceased) of these patients were evaluated. Age at diagnosis was stratified into three groups: <2 years, 2–13 years, and ≥14 years. Hemoglobin level (g/dL), leukocyte count (x10^9/L), platelet count (x10^9/L), and blast percentages in bone marrow aspirate and peripheral smear (%) at diagnosis were recorded. Leukocyte count was categorized as $<50 \times 10^9$/L or $\geq 50 \times 10^9$/L.Patients were treated according to the AML-Berlin–Frankfurt–Munster 2004 and AML-BFM 2012 protocols. In the AML-BFM 2004 protocol, patients are stratified into standard-risk (SR) and high-risk (HR) groups based on their treatment response after induction and genetic features at the time of initial diagnosis. The AML-BFM 2012 protocol also stratifies risk as SR, intermediate-risk (IR), and HR. In addition, patients were compared in two groups: AML M3 (acute promyelocytic leukemia) and non-AML M3 (AML M1, M2, M4-7, biphenotypic, undifferentiated type).The AML-BFM 2004 protocol was used between 2010 and 2015, and the AML-BFM 2012 protocol was used between 2016 and 2023. Treatment efficacy was assessed at the morphological level using bone marrow aspiration at the end of chemotherapy blocks. Complete remission was defined as normocellular marrow with ≤5% blasts, peripheral neutrophil count $\geq 1 \times 10^9$/L, platelet count $\geq 80 \times 10^9$/L, absence of extramedullary disease, and no blasts in the central nervous system. **Statistical Analysis:** Data were expressed as mean ± SD or median (IQR) based on normality. Intergroup comparisons were performed using Chi-square, Fisher's exact, and t-tests. Non-normally distributed data were analyzed using the Mann–Whitney U test. Survival rates were calculated using the Kaplan–Meier method. Receiver operating characteristic (ROC) curve analysis evaluated prognostic prediction performance. Statistical analyses were conducted using IBM SPSS Statistics Data Editor. A p-value <0.05 was considered statistically significant. **Results:** The median age was 12 years, with the most frequent age group being 2–13 years. Twenty-five patients (51%) were female. The most frequent morphological subtype was AML M3 in 16 cases (32.7%). Risk stratification classified 28 patients (57.2%) as SR, 6 (12.2%) as IR, and 15 (30.6%) as HR. The most frequent genetic mutation observed was t(15;17) in 16 patients (32.6%), followed by t(8;21) in 9 patients (18.3%). Seventeen patients (34.7%) were treated under the AML-BFM 2004 protocol, and 32 under the AML-BFM 2012 protocol. Leukapheresis was performed in 8 (16.3%), with all leukapheresis patients achieving a response. Complete remission (bone marrow blasts <5%) after induction was achieved in 40 patients (81.6%). Six patients underwent bone marrow transplantation (BMT), of whom 3 (50%) died due to post-transplant relapse. Thirteen patients (26.5%) experienced relapse: One in the AML M3 group and 12 (24.5%) in the non-AML M3 group. Twelve relapsed patients (92.3%) died. Among BMT recipients, one patient underwent transplantation for HR disease without prior relapse; however, later died due to post-transplant relapse. Overall, 19 patients (38.8%) died. Survival rates were significantly lower in patients with leukocyte counts $\geq 50 \times 10^9$/L (p=0.002). ROC curve analysis revealed an area under the curve (AUC) of 0.744 for leukocyte count ($p < 0.005$), with a cut-off value $>42{,}850 \times 10^9$ /L. Lower hemoglobin levels at diagnosis were associated with reduced survival (p=0.030). The mean overall survival (OS) for AML-M3 patients was 113 months ±%6.8 months (95% CI: 99.3-126.3), whereas the

2531-1379/

median OS for non-AML M3 patients was 28±%17.2 months (95% CI: 0.00-61.8) (p=0.002). Survival duration was shorter in IR and HR groups than the SR group.Cumulative survival rates at 1 and 5 years were significantly longer in AML-M3 patients than non-AML M3 patients. For AML-M3, cumulative survival was 94%±%6 at both time points. In non-AML M3 patients, 1-year and 5-year cumulative survival rates were 73% and 43%, respectively (p = 0.002). In relapsed patients, median OS after relapse was 3±0.8 months (95% CI:1.23-4.76) (p=0.000). No significant difference in survival rates was observed between the AML-BFM 2004 and AML-BFM 2012 protocols. **Conclusion:** In the present study, key factors influencing survival in pediatric AML included risk group, age at diagnosis, induction response at diagnosis, and time-to-relapse. Among relapsed patients, the initial risk group also affected survival. Leukapheresis had no impact on survival. Mortality remains high in non-AML M3 cases. Further research is required to develop genetically defined treatment subgroups in pediatric AML. We recommend more stringent risk stratification for IR patients under the AML-BFM 2012 protocol, and advocate for larger studies aimed at creating standardized, personalized treatment protocols for all patients.

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OP 02

TP53-Deleted Mixed Phenotype Acute Leukemia with Widespread Nodal Disease: Complete Remission after HyperCVAD plus Azacitidine

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Introduction: Mixed phenotype acute leukemia (MPAL) is rare and clinically aggressive, particularly when accompanied by TP53 deletion and complex karyotype. Nodal presentations can mimic lymphoma, delaying definitive therapy. We report a young woman with MPAL (B/Myeloid) and extensive nodal involvement who achieved complete remission (CR) with HyperCVAD plus azacitidine. **Methods:** Single-patient case review of prospectively collected data. Diagnostic work-up included complete blood counts, bone marrow (BM) aspirate/biopsy with immunohistochemistry (IHC), multiparameter flow cytometry, cytogenetics/FISH, PCR panel for recurrent fusions, and FDG PET-CT. Treatment consisted of HyperCVAD combined with azacitidine. Response was assessed morphologically, by PET-CT, and by minimal residual disease (MRD) testing. **Results:** A 37-year-old woman presented with fatigue, bilateral cervical and axillary lymphadenopathy, and pancytopenia. BM was normo−to-hypersellular (cellularity ∼50 −65%) with blast proliferation; reticulin 0−1/4. IHC showed CD34+, CD117+, CD33+, heterogeneous CD3 and rare TdT; PAX5 was positive in marrow sections, while CD20, MPO, and CD13 were negative. Excisional axillary-node pathology revealed blast infiltration (CD34+, CD117+, CD33+, CD3+, CD5+, CD10+, BCL2+, Ki-67 ∼30%; PAX5 and MPO negative), supporting leukemic involvement. Flow cytometry identified a 53% blast population expressing CD33, HLA-DR, and aberrant CD7, negative for CD19, CD10, surface CD3, and MPO—consistent with **MPAL (B/Myeloid)** in the aggregate clinicopathologic context.Cytogenetics demonstrated **complex hyperdiploidy (85−92 chromosomes)** with **trisomy 8** and **tetrasomy 10**; FISH detected **TP53 (17p) deletion**. TEL/AML1, PML/RARA, BCR/ABL, AML/ETO were negative by FISH; PCR for BCR-ABL, PML-RARA, and FLT3 was negative. Baseline PET-CT showed **widespread FDG-avid nodal disease** (cervical, axillary, mediastinal, abdominal, retroperitoneal; SUVmax ∼4 −10) without visceral uptake. First-line **HyperCVAD plus azacitidine** was administered with standard supportive care. End-of-treatment evaluation demonstrated **morphologic CR**, **MRD negativity**, and **metabolic complete response** by PET-CT. The patient remained in remission on early surveillance. **Discussion:** This case highlights three practice points. (1) **Nodal MPAL can masquerade as lymphoma**; integrated BM, node histology, flow, and molecular profiling are essential to prevent misclassification and treatment delay. (2) **TP53 deletion with complex karyotype** portends high risk; nonetheless, **HyperCVAD plus azacitidine** achieved deep response, suggesting potential synergy of epigenetic priming with intensive chemotherapy in adverse-genetic MPAL. (3) Discordant lineage signals (e.g., PAX5 IHC positivity with B-lineage markers absent on flow, and MPO negativity despite myeloid antigen expression) illustrate real-world diagnostic ambiguity in MPAL and the need to rely on the totality of evidence rather than any single assay. **Conclusion:** In TP53-deleted, complex-karyotype **MPAL with extensive nodal disease**, **HyperCVAD plus azacitidine** induced **MRD-negative CR** with metabolic clearance. This experience supports considering epigenetic-augmented intensive regimens in high-risk MPAL and underscores the diagnostic value of coordinated marrow−node evaluation.

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OP 03

Dasatinib-Induced Progressive Enterocolitis Mimicking Inflammatory Bowel Disease in a Patient with Chronic Myeloid Leukemia: A Case Report

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Introduction: Dasatinib is a potent second-generation tyrosine kinase inhibitor widely used in chronic myeloid leukemia (CML) treatment, particularly in patients intolerant to imatinib. While generally well-tolerated, dasatinib can cause various adverse effects including pleural effusions, cytopenias, and gastrointestinal symptoms. However, progressive enterocolitis resembling inflammatory bowel disease (IBD) is rarely reported and poses diagnostic challenges due to clinical and endoscopic similarities to IBD. **Case Report:** A 71-year-old female with a 20-year history of achalasia was diagnosed with chronic myeloid leukemia in 2021 following evaluation