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*abstract*

## **KDM6A: An Emerging Biomarker for Prediction of Treatment Response and Relapse in Pediatric Acute Myeloid Leukemia**

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DOI: 10.69690/ODMJ-018-0425-2403



SIOP Asia, 2025, Saudi Arabia

## KDM6A: An Emerging Biomarker for Prediction of Treatment Response and Relapse in Pediatric Acute Myeloid Leukemia

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**Introduction:** Despite advancements in diagnostic techniques and the development of newer chemotherapeutic agents, outcomes for acute myeloid leukemia (AML) remain dismal. KDM6A, a histone H3K27me3 demethylase, is a tumor suppressor gene with potential implications in AML. This study aimed to investigate the role of KDM6A in pediatric AML, exploring its association with treatment response and relapse.

**Methodology:** In this prospective study, bone marrow aspirates or peripheral blood samples were collected from children (age  $\leq 18$  years) at three time points: diagnosis, remission, and relapse. Total RNA was extracted from the samples and subsequently reverse-transcribed into complementary DNA (cDNA) for quantitative expression analysis of KDM6A. The dynamic expression patterns of KDM6A were evaluated using quantitative real-time PCR (qRT-PCR).

**Results:** A total of 81 patients with AML were enrolled in the study. The most common symptoms observed were fever (78.8%), pallor (54.7%), fatigue (36.7%), and loss of appetite (45.5%).

The most common cell surface markers detected were CD33 (100%), CD38 (91.67%), CD117 (87.80%), and CD13 (75%). The expression was downregulated at the diagnosis ( $n=81$ ) compared to the control group ( $n=15$ ) ( $p<0.01$ ). KDM6A was upregulated at remission compared to diagnosis ( $p=0.001$ ) ( $n=54$ ), whereas its expression decreased on relapse of the disease in paired samples in comparison to remission ( $p<0.01$ ) ( $n=10$ ). The KDM6A expression during remission was comparable to the control sample with no significant difference but was highly downregulated at diagnosis ( $p<0.001$ ).

**Conclusion:** The differential expression of KDM6A observed exclusively at diagnosis and relapse highlights its potential role in the pathogenesis of pediatric AML. Given the poor prognosis of AML, incorporating KDM6A as a novel disease marker is crucial for enhancing risk stratification and identifying targeted therapeutic strategies to improve patient outcomes.