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Assessment of CCAAT/enhancer binding protein α gene methylation in acute myeloid leukemia patients in Egypt: Relation to response to induction therapy

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Background: Global gene promoter studies, as well as gene-specific approaches, have revealed that aberrant promoter methylation is a common event in AML. One such gene, CCAAT/enhancer binding protein α (C/EBPα), is a key transcription factor involved in the regulation of cell proliferation and differentiation in a variety of cell types, particularly in the hematopoietic system. Because of the pharmacologic reversibility of epigenetic changes by drugs, such as the DNA-demethylating agent, epigenetic therapy seems prominently among novel leukemia treatment strategies.

Purpose: The present work is a cohort study that aimed at assessing the frequency of promoter methylation of the CEBPα gene in 70 cytogenetically normal, newly diagnosed AML Egyptian patients. Furthermore, the relation between the methylation status of the CEBPα gene and the outcome of standard induction therapy on day 28 was evaluated.

Patients & Methods: Purified genomic DNA samples from the 70 newly diagnosed AML patients were subjected to bisulfate modification before methylation-specific polymerase chain reaction was performed to assess the CEBPα gene methylation status. Clinical and laboratory assessment of patients after 28 days of induction of standard therapy was performed.

Results & Conclusion: Fifty-four percent of AML samples showed CEBPα gene methylation at the promotor region. Positive methylation was seen associated with blast expression of T-cell markers and blast counts in peripheral and bone marrow samples; but did not privilege a particular FAB classification subtype or relate to age and gender. The positive CEBPα gene methylation status was seen acceptable to predict AML patients with resistant clone who did not respond to standard induction therapy and blast clearance at day 28 (95% CI: 0.466–0.985, p= 0.005). Assessment ofCEBPα gene methylation in AML patients might lead to refined prognostic stratification and suggest differentially tailored treatmentbased on its methylation status.

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