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The expressions of myeloid differentiation and non-lineage specific differentiation antigens among FAB subtypes of acute myeloid leukemia in Iraqi patients

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ABSTRACT

Background. Acute myeloid leukemia (AML) is a complex malignant disorder showing various subtypes with myeloid differentiation and non-lineage specific differentiation antigens (CD markers) expressions.

Aim. The aim of this study is to evaluate the expression of myeloid and lymphoid antigens in relation to different French-American-British classification (FAB subtypes).

Method. Three hundred and forty-four acute myeloid leukemia patients diagnosed based on blast percentage underwent bone marrow aspirate or whole blood collection, and flow cytometry was used to identify blast cells.

Results. The expressions of myeloid differentiation antigens in AML patients were commonly observed; CD33 (73.3%), CD13 (68.3%), CD117 (67.4%), CD64 (54.6%), and cMPO (52.9%) in AML cases, with a significant difference in positivity (p-value < 0.05) among FAB classes (M0, M1, M2, M3, M4, M5, and M7). The expression of non-myeloid-associated antigen HLA-DR (34.01%) and CD34 (42.15%) showed high prevalence among FAB subtypes of AML. Regarding CD36 (13.3%), CD35 (13.08%), IREM2 (9.9%), and CD123 (9.01%), the expression was lower than HLA-DR and CD34, with no expression of CD38 in AML patients. A significant difference in expression and distribution of CD markers among AML subclasses was found (p<0.05).

Conclusion. A large number of AML cases showed CD33 (73.3%), CD13 (68.3%), and CD117 (67.4%) expressions; these markers give two scores in diagnosis and highly distribution in AML subtype. Although no other leukemia subtypes were found to be linked to AML instances, AML blasts exhibited abnormal lymphoid characteristics, whereby the most prevailing lymphoid marker that was aberrantly expressed in AML was CD7. T-cell markers were more prevalent than B-cell markers.

Keywords: AML, leukemia, Iraqi patients, CD marker, AML specific antigen, AML FAB subtype

INTRODUCTION

Acute myeloid leukemia (AML) is divided according to the World Health Organization (WHO) into subtypes from M0 to M7, each with distinctive clinical and prognostic consequences, based on their morphological and cytochemical characteristics [1]. Cluster of differentiation (CD) marker-based immu-

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