Evaluation of galectins gene expression in acute myeloid leukemia patients

A Thesis Submitted in Partial Fulfiment of the Requirements for the Award of the Degree of

Master of Science

By

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2020

ACKNOWLEDGMENT

First and foremost, praises and thanks to the God, by whose grace this work had been completed.

I would like to express my deep and sincere appreciation to prof. Dr. Nagwa Hassan Ali, Professor of cytogenetics, Department of Zoology, Faculty of Science, Ain Shams University, for her professional guidance, encouragement and careful monitoring through different stages of this research. I am extremely grateful for everything she has taught me.

I would also like to thank Prof. Dr. Magda Mahmoud Assem, Professor of clinical pathology, National Cancer Institute, Cairo University, for giving me the opportunity to do research. Her energy, vision and constant motivation have also inspired me.

I am extremely grateful for Dr. Reham Hassan Helwa, Assistant Professor of molecular cancer biology, Department of Zoology, Faculty of Science, Ain Shams University. It was a great pleasure and honor to work and study under her guidance. She has taught me the methodology and statistics to perform the research and to present the research works as clearly as possible. You have done so much that I can't find words to express my gratitude.

I appreciate help and encouragement that I have received from my colleagues of my department during the hard time.

Finally, Iam gratefully to my parents for their love and supporting me to complete this research successfully.

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Abbreviations

AML	Acute myeloid leukemia
APL	Acute promyelocytic leukemia
APCs	Antigen presenting cells
BCL2	B-cell lymphoma 2
BFGF	basic fibroblast growth factor Bone marrow
BM	Bone marrow
CD	Cluster of differentiate
COX	Cyclooxygenase
CRC	Colorectal cancer
CRD	Carbohydrate recognition domain
CR	Complete remission
CT	Cyclic threshold
DCs	Dendritic cells
DEPC	Diethyl pyrocarbonate
DFS	Disease-free survival
ECM	Extracellular matrix
EGFR	epidermal growth factor receptor
ELN	European leukemia net
ERK	Extracellular signal-regulated kinases
FAB	French–American–British
FLT3	Fetal liver tyrosine kinase 3
GAPDH	Glyceraldehyde 3-phosphate dehydrogenase
GSK3	Glycogen synthase kinase 3
HGF	Hepatocyte Growth Factor
HIF	Hypoxia-inducible factors
HIPK2	Homeodomain interacting protein kinase 2

HSCs	Human stem cells
IFN-γ	Interferon gamma
IL-10	Interleukin 10
IPT	Immunophenotyping
JM	Juxta-membrane
LSCs	Leukemia stem cells
MAPK	Mitogen-activated protein kinase
Mcl-1	myeloid cell leukemia 1
MHC	major histocompatibility complex
MMP	Matrix metallopeptidases
MSCs	Mesenchymal stem cells
MUC1	Mucin 1
NK	Natural killer cell
OS	Overall survival
PB	Peripheral blood
PDGF	Platelet-derived growth factor receptors
PDL-1	Programmed death-ligand 1
qRT-PCR	quantitative reverse transcription Polymerase chain reaction
RNA	Ribonucleic acid
RT-PCR	Reverse transcription Polymerase chain reaction
STAT3	Signal transducer and activator of transcription 3
TCR	T-cell receptor
TIM3	T-cell immunoglobulin mucin 3
Th1	T-helper cell 1
Th17	T-helper cell 17
TNF-α	tumor necrosis factor
VEGF	Vascular endothelial growth factor
WHO	World health organization

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Abstract

Introduction:

Acute myeloid leukemia (AML) is a malignant hematopoietic disease characterized by an overproduction of immature myeloid cells, (myeloblasts) in the bone marrow in which precursors of blood cells are blocked in an early stage of maturation. AML is a disease of older adults with median range 68 years. Galectins family are animal lectins which affect a wide range of cellular functions. Many galectins are repeatedly reported in several physiological changes and diseases including cancer. In AML, there is a big focus on galectins-3 and -9, but not the other galectins.

Patients and Methods: Bone marrow (BM) and corresponding Peripheral blood (PB) were collected from recently diagnosed 45 adult patients with *de novo* acute myeloid leukemia, present in National Cancer Institute (NCI), Cairo University (CU). Our study was carried out to investigate the regulation of galectins expression in the bone marrow and corresponding peripheral blood samples of AML diagnosed patients and correlating them to clinicopathological data.

Results: Our results discuss the dysregulation of several galectins in AML patients. Upregulation of galectin-1 has shown a significant correlation to monocytic AML, as it was more upregulated in M4 and M5 (p=0.006 and p=0.015 in bone marrow and peripheral blood respectively), as well as positive CD4, CD11c, and CD64. The same finding was encountered with galectin-2 where its overexpression was also a sign of monocytic AML. The other galectins are statistically significant with many clinicopathological features indicating their clinical significance correlation to monocytic AML. Galectin-3 is almost downregulated in opposite way to the previous studies. The expression of MHC class II is significantly associated with overall survival (OS) advantage (p<0.001).

Conclusion: Galectin-1 and -2 could be used as markers for monocytic AML. MHC class II could be a good prognostic factor.

Keywords: galectins/AML/bone marrow/peripheral blood/ qRT-PCR.