

Differential expression of autophagy-related genes is associated with clinical outcomes of patients with acute myelocytic leukemia

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Abstract:

Autophagy is a catabolic process that allows cells to maintain homeostasis and survival under stressful conditions. Accumulating evidence suggests that autophagy contributes in the initiation and progression of cancer as well as resistance to chemotherapy. Little is known about the clinical significance of autophagy and its relationship with therapeutic outcomes in patients with acute myelocyctic leukemia (AML). Here we report the differential gene expression pattern of some autophagy-related genes (ATGs) that have an operative function during different steps of autophagy in AML patients. These key autophgic markers (ATG4, ATG5, BECN1/ATG6, ATG7, MAP1LC3/ATG8, ATG12 and SQSTM1/p62) were transcriptionally analyzed in PBMCs samples from 43 AML patients compared with healthy volunteers by q-PCR. In general, the gene expression levels ATGs were significantly higher in PB-MCs from AML patients compared with healthy individuals. We also analyzed bone marrow (BM) samples newly diagnosed AMLs before and after treatment with standard chemotherapy. In contrast with PBMCs, the level of autophagy markers was low in BM samples from diagnosed AML patients compared with controls. Follow-up results indicated that autophagy level was significantly changed after treatment of AML patients, and this was associated with the clinical outcomes. In fact, high expression of ATGs in BM samples from AML patients was



associated with a higher risk of relapse. However, the expression levels of ATGs remained down-regulated in the remission population of AML, even lower than those observed in the BM samples from diagnosed AML patients. These results may further our knowledge of involvement of autophagy during pathogenesis and treatment of AML, and thus may have therapeutic implications in future studies.

Biography:

Ayla Fakhimadnadi is a professor at Tehran University of Medical Sciences, Iran.

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