Analysis of multiple Genetic and biochemical markers in patients with Esophageal Squamous Cell Carcinoma and evaluation their applicability in prognosis

Shahla Mohammad Ganji

National Institute of Genetic Engineering and Biotechnology (NIGEB), P.O. Box 14155/161, Tehran, Iran, Tel: +98-21-44 580 301-9 Ext. 4464, Mobile: +98-912 621 93 23, Fax: +98-21-44 580 399, E-mail: shahlamg@yahoo.com

==========

Esophageal cancer ranks among the top 10 most frequent cancers worldwide and 6th most frequent cancers in Iran. The aim of this research was investigation of multiple Genetic and biochemical markers in patients with Esophageal Squamous Cell Carcinoma and evaluation their applicability in prognosis. On the other hand, we know that hypermethylation of promoters is among mechanisms that leads to inactivation of genes involved in growth, development of cells and tissues along with cellular reproduction. Regarding to this fact that there is a close relationship between cancers and cell division, this phenomenon could have important impact on a better understanding of the biology, etiology and molecular mechanism beyond cancers and achievement of molecular markers. There are some important genes involved in regulation of cell cycle including; p14, p15, p16, p18, p21, and p27. For these reasons, we obtained 44 tumor tissue samples from patients with SCCE, 19 adjacent tissues, 50 whole bloods and 40 serums from 72 patients with SCCE whom attendant to the hospital to underwent the surgery. (Methylation Specific Polymerase Chain Reaction) MSPCR were done by specific primers for above genes. Also, the Quantitative Real time PCR by Taqman method was done for p16. On the other hands, the expression of these genes was done by RT-PCR. The results showed 30%, 41%, 9%, and 6.8% methylation for p16, p15, p14, and p27 genes respectively. For p18 and p21 there were not observed any methylation. Moreover, the methylation for p14+p15+p16 genes together, was 4.5%. Any of normal tissue samples didn't show methylation for above genes. These results are similar to Chinese and Italian patients. Also we assessed the functionality of Alpha-1 antutrypsin (AAT), as an acute phase protein as well as association of AAT phenotype and genotypes Z and S with SCCE. The results indicate that mean range of trypsin inhibitory capacity (TIC) and AAT nephlometry in patients are significantly different ($P<0.05$) from healthy control subjects. Moreover, 97.3% of SCCE patients were homozygote for the normal allele of protease inhibitor MM (PiMM), and only 2.7% MS heterozygote. The results of other researchers supports the elevation of AAT in seums of patients while there is not any similar results for phenotyping and genotyping of AAT in SCCE. By doing the statistical analysis we tested whether few factors that increase the risk of developing esophageal cancer could also affect aberrant DNA methylation and the results showed the association between them.