Abstract:

Background and objectives:

Cancer is the third most common cause of death in Iran and 30,000 Iranians lose their lives each year due to cancer. Esophageal cancer is the eighth most common cancer and the sixth cause of death from cancer worldwide and is reported to include more than 80% of all death cases in developing countries. *P21* protein is encoded by WAF1/CIP1, a tumor suppressor gene located on chromosome 2.6*P21*. This protein is known as one of the proteins related to cell cycle;*P21* stimulation is a common mechanism of growth inhibition in different physiological conditions.

Materials and methods:

The present case-control study randomly selected15 paraffin-embedded esophageal cancer tissue and 15 paraffin-embedded normal esophageal samples, collected from different medical centers of Zahedan and Kashan. RT-qPCRreactions were performed with three repetitions for *P21* gene and internal control (GAPDH) by Livac method 2-ΔΔCT for all samples and was analyzed using SPSS software.

Results:

The results indicatedno significant difference in*P21* gene expression between patients and controls ($p>0.05$). Also, no significant difference was observed in*P21* gene expression between males and females ($p>0.05$).

Discussion:

No significant differences were observed in *P21* gene expression. *P21*protein exists in low amount in most cells being reproduced. As protein *P53*is activated by DNA fractures or intermediate replicators,it isconnected to motifs in promoter CDKN1A (the gene coding *P21* protein) and increases transcription of CDKN1A gene sharply. But the transcription of CDKN1A gene are impaired in cancer cells, producedduring*P53*mutationsand thus fail to limit the progression of cell cycle in response to DNA damage and the incomplete cloned chromosome. The intracellular levels of this protein increases in cancerous tissue due to *P53* gene mutations and increased half-life of the mutant form of the protein, but cannot express *P21* gene due to lack of normal function.Regarding the activation pathways of *P21*(dependenton*P53* and independent of*P53*),increase in the expression of *P53*, D-cycline, MYC, and Ras causes lack or reduction in *P21* expression. Biological and network adjustment activity of *P21* complex makes predictions about its function to make cancer treatment possible.

No relationship between the expression of $P21^{WAF1}$ and clinical pathologic parameters was found regarding sex and age (Liu *et al* ., 2006)

Key words: Esophageal cancer, paraffin-embedded tissue, gene expression, *P21*gene, RT-qPCR.

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P21 tumor suppressor gene expression evaluation in esophagus cancer patients

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