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P68. FGFR1 SER777 MUTATION AND BLADDER CANCER

Asuman ÖZGÖZ*[1], Hale ŞAMLI[2], Murat ŞAMLI[3], Deniz DINÇEL[2], Ümit İNCE[4], Ahmet ŞAHIN[3]
[1]Kastamonu University, Fazıl Boyner School of Health, Kastamonu, Department of Nutrition and Dietetics, Kastamonu, TÜRKİYE
[2]Uludag University, School of Veterinary Medicine, Department of Genetics, Bursa, TÜRKİYE
[3]Acıbadem University, School of Medicine, Department of Urology, Istanbul, TÜRKİYE
[4]Acıbadem University, School of Medicine, Department of Pathology, Istanbul, TÜRKİYE

Bladder cancer is the fourth most frequent cancer type among men and eighth most frequent among women in western countries. As fibroblast growth factor receptors (FGFRs), receptor tyrosine kinases are related to many cancers and different pathological situations. Therefore FGFRs' regulatory mechanisms are needed to be understood and are in current interest. The four closely related FGFR receptors (FGFR1 to FGFR4) have important duties in proliferation, differentiation, migration, and survival of cells.

Because FGFR1 C-terminal including amino acids 755 to 822 has many serines; it is suggested that FGFR signaling might be regulated via specific serine phosphorylation and wild-type FGFR1 was detected to be phosphorylated by active ERK1 and ERK2 forms but FGFR1 critical serine mutated proteins at the S777A and S777D, were not. FGFR1 ser 777 was shown to be mutated in a few number of research that has been carried out in the area of cancer . But these mutations was shown only in cell lines and and not by directly sequencing or controlling of the mutation as nucleotide change. In our study, FGFR1 ser 777 mutation was studied by sequence analysis method in 62 cases with bladder cancer and in 30 healthy controls.

In the study we performed, there was any FGFR1 ser 777 mutation neither in case nor in the bladder cancer group. It may be concluded that ser 777 is not mutated in bladder cancer.