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Angiogenesis and anti-angiogenesis marker of bladder cancer before and after treatment

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Aim: Cancer is one of the most widespread causes of death in the world. It spreads through the body by metastasis and its ability to penetrate the blood and lymphatic vessels, and it has been established by research that this invasion is made easier by angiogenesis. VEGF (vascular endothelial growth factor) is an angiogenic factor which increases blood vessel permeability; it stimulates secretion of MMP (Matrix metalloproteinases), which is responsible for breakdown of the extracellular matrix, and thus eases metastasis and invasion. In patients with bladder cancer, intravesical chemo-immunotherapies are administered to reduce the risk of progression. In order to examine this mechanism more closely, we examined bladder cancer patients receiving routine treatment to find the differences in levels before and after treatment of the angiogenic and antiangiogenic markers VEGF, MMP, ES (endostatin) and TSP-1 (thrombospondin-1).

Material & Method: The study group consisted of 90 cases (bladder cancer, first treatment and second treatment), and the control group comprised 30 health workers in the same age range as the study group. Blood is taken routinely from all patients before and after the operation and at the end of intravesical treatment, and a small amount of this blood was used in the study. The blood collected was examined for levels of VEGF, MMP, ES and TSP-1 by Elisa test, and the differences in relation to bladder cancer were evaluated.

Results: Comparison between the control group and the case group was performed with Mann Whitney U test. When the control group was compared with group 1(bladder cancer) levels of VEGF and MMP were shown to be significantly raised ($p<0.05$), but differences in TSP-1 and ES were not statistically significant. In the comparison of the control group with group 2 (first treatment), VEGF and MMP were seen to be significantly reduced, but differences in TSP-1 and ES were not statistically significant. When the control group was compared with group 3 (second treatment), clear reductions in the levels of VEGF, MMP, TSP-1 and ES were observed. When group 1 and group 2 were compared, VEGF and MMP showed a significant reduction, but the difference in TSP-1 and ES levels was statistically insignificant. When group 1 and group 3 were compared, ES, TSP-1, VEGF and MMP were all significantly reduced. In the comparison between group 2 and group 3, differences in the levels of ES, TSP-1, VEGF and MMP were statistically insignificant ($p<0.05$).

Discussion: It was found that in bladder cancer patients, treatment gave rise to a reduction in the angiogenesis activators VEGF and MMP, but did not cause a significant change in the levels of the powerful angiogenesis inhibitors endostatin and TSP-1. It is felt that the reason for this lack of increase in the antiangiogenic factors ES and TSP-1 is connected to the length of adjuvant treatment given. We believe that ES and TSP-1 will increase in relation to the length of adjuvant treatment. We feel that in the light of this information, these markers can be used in following the diagnosis and treatment of bladder cancer.

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