Результати. При виконанні кореляційного аналізу констатовано сильний, позитивний кореляційний зв'язок між GS та PPFV-PVI, PPFV, PPFA-PAI (r = 0,77; 0,77; 0,75 відповідно), між PPFA, PPFT, PPFT-SCFTI – помірний (r = 0,74; 0,64; 0,59 відповідно).

Висновки. PPFP визначені на основі даних МРТ продемонстрували позитивні кореляційні взаємозв'язки з балом за шкалою Глісон у пацієнтів з РС cT_{1-2}. Подальші дослідження на більших вибірках пацієнтів необхідні з метою валідації рівнів PPFP та методики вимірювання останніх для практичного застосування.

PERSPECTIVES FOR THE CYTOKINE ENDOTHELIAL-MONOCYTE ACTIVATING POLYPEPTIDE II TO BE USED IN NEURO-ONCOLOGY

Shuba I. M.[1], Kornelyuk O. I.[2], Glavatskyi O. Ya.[1], Karpova I. S.[2], Lylo V. V.[2]
[1]*Kyiv, Ukraine, State Institution "Romodanov Institute Neurosurgery, National Academy of Medical Sciences of Ukraine"*
[2]*Kyiv, Ukraine, Institute of Molecular Biology and Genetics, National Academy of Sciences of Ukraine*

Background. Oncological diseases, including primary malignant gliomas, are one of the most important problems of medicine nowadays and the search for new non-toxic chemotherapeutic drugs that can supplement the treatment regimen is extremely relevant. Gliomas are the malignant tumors of the brain and that are most aggressive neoplasm. Gliomas are characterized by high infiltrativeness and an extremely high degree of vascularization. That is why it is advisable to use drugs with antiangiogenic properties. The cytokine endothelial-monocyte activating polypeptide II (EMAP II) has been shown to have these properties. Many studies have shown that one of the possible antiangiogenic mechanism of EMAP II action is the ability to inhibit the binding to of VEGF (vascular endothelial growth factor) to receptors VEGFR-1 and VEGFR-2. In addition, EMAP II has been shown to bind to α5β1-integrin on the surface of endothelial cells, preventing them from adhering to fibronectin. It causes inhibiting endothelial cell proliferation and migration, and consequently inhibits angiogenesis. The ability of EMAP II to stimulate apoptosis to was shown for endothelial cells.

Aim: to investigate if the cytokine EMAP II reveals cytotoxic effect on glioma cells.

Materials and Methods. The cell culture of the human glioma cell line U251MG and the primary culture of gliomas cell extracted from fragments of malignant gliomas tissue after surgical intervention were treated with different concentrations of EMAP II (1.0 pM – 10.0 μM). Cytotoxic effect was determined by MTT test after 24 h of incubation with EMAP II.

Results. The biphasic effect of EMAP II cytotoxicity in the range of low (600.0–700.0 pM) and high (2.0–10.0 μM) concentrations was observed on the cells of the U251GM line. On the primary cells culture we observed three concentration ranges with cytotoxic effects – 20.0–30.0 pM, 600.0–700.0 pM and 10.0 μM.

Conclusion. The cytotoxic effect was showed of the cytokine EMAP II on cells of glioma lines. We think the cytokine EMAP II may be a promising compound to be used in combination with anti-angiogenic and chemotherapeutic drugs, enhancing their effect.