Abstract

Angiogenesis is the process of creating new blood vessels based on the existing capillary network. This proces depends on pro – and anti – angiogenic factors, which in a physiological state are in balance with each other. In the neoplastic process, as a result of the "sw*itch on*" phenomenon, this balance is disturbed and proangiogenic factors (VEGF-A) predominate, which leads to the activation of angiogenesis. The circulating VEGF-A receptors sVEGFR1 and sVEGFR2 are considered endogenous angiogenesis inhibitors, however, their role in carcinogenesis is unclear.

The aim of the study was to assess the angiogenic potential by measuring the concentration of VEGF-A and its circulating receptors sVEGFR1 and sVEGFR2 in the blood plasma (issue controls) and tumor tissue of patients with intracranial neoplasms.

The study included 69 patients (45 women and 24 men) aged 20 to 80 (mean 61,6 years) treated surgically for intracranial tumors. Among the tested samples there was material from 21 patients with gliomas – a group of patients with low-stage gliomas (I and II – 5 people) and high-stage gliomas (III and IV – 16 people) was distinguished, 18 people with meningiomas (I grade) and from 30 patients with metastatic tumors (grade III and IV). The control group consisted of 30 healthy volunteers aged 20–55 (mean 51,3 years).

The material for the study was peripheral blood and a section of tumor tissue. The concentration of VEGF-A, sVEGFR1 and sVEGFR2 was determined in the tumor section obtained during surgery and in the blood plasma by ELISA. Protein concentration was also determined. The studied angiogenesis parameters were converted to 1 mg of protein and the sVEGFR1/VEGF-A and sVEGFR2/VEGF-A ratios were calculated.

Statistically significantly higher concentrations of VEGF-A and sVEGFR1 in the blood plasma of the patients were found in comparison to the control group. The concentration of sVEGFR2 in the blood plasma of patients was significantly lower. A statistically significantly higher concentration of VEGF-A was observed in tumor homogenates compared to plasma.

The following conclusions were drawn: 1. A high concentration of VEGF-A in the blood of patients and in the homogenates of intracranial tumors sindicates the stimulation of angiogenesis. 2. Many times than in the blood higher concentration of VEGF-A in tumor homogenates than in blood of patients indicates tumor cells as a source of VEGF-A.

3. Increased concentration of sVEGFR1 in blood plasma and tumor homogenate from patients is a compensatory mechanism triggered by high concentration of VEGF-A. 4. The reduced concentration of sVEGFR2 in the blood and in the tumor homogenate seems to be caused by its consumption in the process of inhibiting increased angiogenesis. 5. The analysis of VEGF-A concentrations of individual types of tumors (low/high–grade gliomas, meningiomas, metastatic tumors) indicates that angiogenesis is stimulated in all types of intracranial tumors studied, but with varying intensity. The highest proangiogenic activity was found in patients with high–grade gliomas and in patients with metastatic tumors. 6. The used sVEGFR1/VEGF-A and sVEGFR2/VEGF-A ratios allow to obtain additional information about the intra–body inhibition of angiogenesis dependent on circulating VEGF-A receptors.