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INTRATUMOURAL CISPLATIN IMPLANTATION IN BRAIN TUMOURS

M.Sramka, P.Hudoba, J.Halko, J.Drobník and P.Nadvorník
Research Institute of Medical Bionics, Institute of Clinical Oncology, Bratislava; Institute of Macromolecular Chemistry, Czechoslovak Academy of Sciences, Prague, Czechoslovakia.

This investigation was performed by computer tomography (CT), guided stereotaxic implantation of Cisplatin into brain tumours in 20 patients. The localization, volume and histological type of tumours were known before implantation. 5 to 30 mg of Cisplatin were administered in a biodegradable gel matrix in the form of cylinders (diameter: 2.8mm) seeded into solid tumours.

In six cases, classical resection of tumour was performed several weeks after Cisplatin implantation. In histological examination, profuse necrosis around the implants was observed. Platinum distribution in the brain was determined and a pharmacokinetic study was performed. In the case of metastatic melanoma, CT examination revealed substantial reduction of the size. With small tumours promising results were obtained. The use of the method is indicated in small and deep brain tumours which are inoperable with classical neuro-surgical treatment.

STE

CELLULAR MECHANISMS OF IMMUNOSUPPRESSION FOLLOWING RADIO- AND CHEMOTHERAPY IN CANCER PATIENTS

Jan A.Steffen
M.Skłodowska-Curie Institute of Oncology, Warsaw, Poland.

Studies on patients with carcinoma of the uterine cervix after radiotherapy and patients with Hodgkin's disease and carcinoma of the ovary after radio- and/or chemotherapy demonstrated long-lasting impairment of lymphocyte mitogen-responsiveness, persisting in cured patients when both modalities were combined. Following radiotherapy, the proliferative response of non-T cells in the presence of T-lymphocytes from healthy donors was impaired only a few weeks after treatment. Sub-sets of T cells and T-cell functions displayed different sensitivity and kinetics of renewal; suppressive interactions were abolished and T-cell cytotoxicity was effectively enhanced shortly after radiotherapy, while the ability of T cells to induce non-T lymphocyte proliferation was impaired for several months. These results are explained by different kinetics of <u>in vivo</u> renewal of lymphocyte sub-sets involved, reduced synthesis of interleukins and, following radiotherapy, also by a more selective damage of recirculating cells.

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PROVIRAL UNIT II OF ENDOGENOUS MMTV IS SELECTIVELY AMPLIFIED AND EXPRESSED IN C57Bl/10 MAMMARY TUMOURS INDUCED BY NON-VIRAL CARCINOGENS

J.Svec and E.Hlavay
Cancer Research Institute, Bratislava, Czechoslovakia.

Restriction analysis of DNA isolated from C57Bl/10 mammary adenocarcinoma induced by DMBA, estrogen, and prolactin revealed the presence of four Unit II proviral copies of endogenous MMTV. The amplified proviruses are hypomethylated and fully expressed. Their internal structure is slightly modified, since an additional EcoRI recognition site is present within the proviral genomic DNA. Sequences of cellular DNA, adjacent to the amplified Unit II proviruses show no homology to the integration domains (int-1 and int-2) common for exogenous MMTV.
