
The acute effect of a fractionated course of whole brain irradiation in patients with intracranial tumors were studied by PET. In the level of centrum semiovale of cerebral images, both regional cerebral blood flow and oxygen utilization of gray and white matter were measured by the 0-15 steady-state inhalation technique. In 12 cases with radiation therapy (RT) for brain tumors, rCBF, rOEF and rCMRO2 were compared with those in 6 cases having no brain tumors without cranial irradiation. The results revealed that both rCBF and rCMRO2 of RT group were slightly lower than those of non-irradiated group, but not significant. The slight elevation of rOEF was showed in the irradiated patients. In four patients followed by PET studies before and after RT, the images showed decreases in both rOEF and rCMRO2 after cranial irradiation in two. But rOEF was almost unchanged or slightly rose.


We examined four patients suspected radiation necrosis, with O-15 labelled gas steady state method and F-18 fluorodeoxyglucose method using positron emission tomography for the purpose of differentiation between radiation necrosis and recurrent tumor, and for the analysis of the pathogenesis of radiation necrosis.

In all the subjects, oxygen and glucose consumptions of the lesion with radiation necrosis decreased remarkably. Cerebral blood flow of the lesion also significantly decreased, but the decrease was less prominent than decrease of oxygen and glucose consumptions. From these observation, considering the fact that glucose consumption of malignant glioma is high, the decrease of glucose consumption of the lesion would be interpreted to be tissue necrosis rather than recurrent tumor. These results would indicate that primary mechanism causing radiation necrosis is a direct effect of radiation on neural tissue rather than radiation injury to the capillary endothelial cells.


For the purpose of analysis of therapeutic effect on regional circulation and metabolism in tumor tissue and in structurally normal brain tissue following radio-chemotherapy, we examined 13 patients with histologically proven cerebral gliomas with O-15 labelled gas steady state method, F-18 fluorodeoxyglucose method and positron emission tomography.

In tumor tissue, glucose consumption decreased remarkably after the therapy. While, in the structurally normal contralateral cortex, significant decrease of glucose consumption and extraction fractions of oxygen and glucose with slightly increased cerebral blood flow were observed within a month after the completion of the therapy. These results would be interpreted as the effect of the therapy on tumor tissue and as not only the result of decreased intracranial pressure but also of the side effects of the therapy on tissue metabolism.

THE STUDY OF CEREBRAL BLOOD FLOW AND METABOLISM BY PET AND MORPHOLOGICAL IMAGING BY MRI-CT IN DEMENTIA. T. Ujike, S. Kitamura, S. Kuroki, T. Kato, S. Sakamoto, T. Soeda, A. Terashi, M. Iio, M. Oshibuchi, T. Kanda, K. Iyoda. 2nd Dept. of Internal Medicine, Nippon Medical School, Tokyo, Dept. of Radiology, Nakano National Hospital, Dept. of Radiology, Hakujoikai Memorial Hospital, **Dept. of Internal Medicine, Hakujoikai Memorial Hospital, Tokyo.

Cerebral blood flow and oxygen metabolism and morphological changes were studied in twelve cases of senile dementia of Alzheimer type (SDAT), fifteen cases of multi-infarct dementia (MID) and five cases of aged normal subjects with positron emission tomography (PET) using O-15 and MRI-CT. CBF and CMRO2 were measured with steady state technique. Spin echo (SE) (repetition time 2000ms/echo time 60ms) and inversion recovery (IR) (1400/40) images were obtained with Hitachi G-10 (0.15 Tesla). IR images could reveal small lacunar infarcts in MID and cortical atrophy of front-temporo-parietal lobes in SDAT. SE images showed diffuse severe patchy white matter lesions in MID. In mild dementias of SDAT, the decreases of CBF and CMRO2 were most remarkable at temporal cortex, and in moderate SDAT, at temporoparietal and frontal cortices. In mild and moderate dementias of MID, the decreases of CBF and CMRO2 were most remarkable at frontal cortex. These results correlate the clinical symptoms and pathologies of dementias well.