DOSE DISTRIBUTIONS IN THE EYE FROM ^{106}RU APPLICATOR

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Beta irradiation with ^{106}Ru applicator is one of the methods of treatment of the tumours located in eye.

Method

In this method we give a dose of 60 Gy to the tumour top. The tumour top is located between 2 and 10 mm from eye surface and it is defined as the border of cancer infiltration. We use echogram to border localisation. From the source certificate we know that the dose rate is charged with a 30% determination error. We increased the calculated dose considering the error value so that at least 60 Gy dose to the tumour top was delivered.

The critical dose for the sclera is 1500 Gy and for the lens 15 Gy respectively. We calculated the maximal dose by the same procedure as the minimal dose through modifying dose rate by certificate error. We calculated doses in the sclera in three places: on the sclera's surface, in the middle of it (about 0.6 mm depth) and on the sclera's bottom edge which is the base of the tumour (about 1.2 mm depth).

Results

Nineteen patients were treated. In all cases the tumour top received at least 60 Gy. In 4 cases it was impossible to deliver a sufficient dose (minimum 60 Gy) at the tumour top without exceeding 1500 Gy in the sclera. It was because of the tumour size was too large (more than 8 mm). In these cases, there was a probability of exceeding 1500 Gy on the sclera's surface when we took the maximal dose rate option. In 4 cases the dose in the lens exceeded a critical dose but we have got the medical agreement to do that.

The important thing is to check the tumour size because the applicator should cover the cancer.

This method of treatment time calculation is fast and easy. We have written the computer program for that procedure which reduced the time of calculation and preparation of the protocol up to a few minutes.

SURGERY FOLLOWED BY IRRADIATION IN GLIOBLASTOMA MULTIFORME. A REPORT OF 28 CASES

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Treatment results in glioblastoma multiforme, irrespective of the management, are poor. Median survival in patients managed with surgery alone is 4 months and in those treated with surgery and adjuvant radiotherpy -9 months.

Twenty eight patients with glioblastoma multiforme were treated at the Department of Oncology and Radiotherapy, Medical University of Gdańsk between 1991 to 1995. There were females and 20 males and the median age was 58 years (range 18 to 75 years). In 22 cases (78%) diagnosis was confirmed by histology, and in the remaining six cases biopsy was not taken due to the deep localization of the tumour; in all these patients diagnosis was based on CT imaging. All patients were irradiated with cobalt unit and received conventional radiotherapy, 5 days a week, 1.8 Gy per fraction. The first part of treatment included whole brain irradiation (40 Gy) delivered through lateral parallel opposed fields. Thereafter in all instances a brain CT was done and in case of regression or stabilisation (23 pts), a boost dose of 15-22 Gy with reduced portals was delivered. Total dose delivered to the tumor bed was 55-62 Gy. Radiotherapy tolerance was satisfactory and there were no serious complications and interruptions of treatment.

Median local recurrence-free survival was 5.3 months, and a median survival - 9.9

months (range, 1.6 to 31.2 months). There was no correlation between survival and radiotherapy dose, sex, pretreatment WHO performance status and tumor localization.

Our results confirm poor prognosis in glioblastoma multiforme. New more effective therapeutic approaches are sorely needed in this tumour.

ANALYSIS OF MUTATIONS IN TUMOUR SUPRESSOR GENE P53 IN BREAST CANCER PATIENTS FROM POZNAN AREA

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The p53 is transcriptional factor that enhances the rate of transcription of six or seven known genes which play important role in cell cycle regulation. The human p53 protein contains 393 amino acids and has been divided structurally and functionally into four domains. The p53 gene and its protein product have been studied since it became clear that slightly more than 50% of human cancers contain mutations in this gene. A study of mutational spectrum at the p53 gene are localized predominantly in the DNA-binding domain of the protein (exons 4-9). The nature of this changes is most commonly a missense mutation in one allele followed by a reduction to homozygosity, producing a faulty protein. Deletions or chain termination mutations are more rarely.

Mutations in p53 gene have been also found in breast cancer in 30-40% of cases. Kind of these mutations suggest that enviromental mutagens may play important role in arising of this type of cancer.It is observed that in West Poland breast cancer occures more frequently then in other areas of the country; the highest numbers of cases are found in GreatPoland still now for unknown reasons. In this work 48 cases of breast cancer were studied. 12 different mutations in p53 were found. This mutations were then compared with datebase catalogs containing mutations in p53. Only 3 from 12 found mutations are the same as reported in datebase. Nine of them were not observed before what may suggest that specific mutational spectrum in patients with breast cancer from GreatPoland exists. Futher studies involving greater number of cases are needed to confirm this observation.

IS ACUTE MUCOSITIS DOSE LIMITING FOR ALTERED FRACTIONATED RADIOTHERAPY ?

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There is now a substantial number of studies on radiotherapy for head and neck cancer using altered fractionation schedules.

Accumulated dose/week (AD) vs incidence and severity of acute mucositis

In conventional radiotherapy given in 1.6-2.0 Gy fractions up to total dose of about 70 Gy, confluent mucositis (CM) is generally reached at day 22. The threshold for the CM appears to be around 20 Gy and the CM usually develops about 9 days after delivering that dose. However, some studies suggest that the onset of CM may depend on accumulated dose/week and the larger AD is the sooner CM is reached.All these observations suggest that the intensity of acute epithelial reactions, and likely other H-type-like tissues reflects the balance between the rate of cell killing by irradiation and the rate of regeneration of surviving stem cells. Once a critical level of survival cells has been attained, a certain type of clinical damage will develop at a rate only determined by the cellular kinetics of the tissue. When a peak in the CM is reached, further stem cell killing can not produce an increase in intensity of acute reactions, but could be manifest as prolonged time to heal the reactions.