images reviewed by one experienced radiologist. Median time of onset was 44 months (range 6 - 197). All patients had pain as the first symptom. The only independent predictive factor for developing osteoradionecrosis was pre-existent osteoporosis. Other risk factors, including higher age, postmenopausal status or steroide treatment, are all related to osteoporosis. We didn't find any significant treatment-related predictive factor for pelvic osteoradionecrosis.

Conclusion: Patients with osteoporosis are at highest risk for developing osteoradionecrotic fractures after pelvic radiotherapy. More studies are needed to find out other endogenous predictive factors (e.g.TGF-beta).

731 POSTER

Time-dose-response relationships in patients with head and neck squamous cell carcinomas treated by surgery and postoperative radiotherapy

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Purpose: To define the influence of the dose and time on the response to treatment in postoperatively irradiated head and neck cancer patients and to establish a good prediction of failure.

Methods and Materials: From January 1985 to December 1995, 214 patients with histologically proven head and neck squamous cell carcinomas were irradiated after radical surgery or single tumour resection according to surgical and histopathologic findings. The total doses given ranged between 50–75 Gy to the primary bed tumour and between 42–56 Gy to the neck with fraction sizes of 1.7–2 Gy/day. The median length of the time interval between surgery and radiotherapy, time of irradiation and total treatment time was 81 days, 59 days and 139 days, respectively. The end-point analysed was the local-regional tumour control rate at the primary tumour bed and neck for 5 years from the beginning of radiotherapy. Univariate and multivariate analyses were used to determine predictors of failure from among the following studied variables: i) clinical stage (T/N) of the patients; ii) tumour grade; iii) neck surgery; iv) tumour margins; v) histological tumour nodal extension; vi) chemotherapy; vii) normalised total dose; viii) time interval between surgery and radiotherapy; ix) time of irradiation; and x) total treatment time.

Results: The actuarial 5 years tumour control rate for the entire group was 72%, and 92% of the patients who achieved local control are currently alive without disease. Tumour control was inversely related to T stage (83% for T_2 vs. 57% for T_4) and the probability of local control within each stage was dependent on the N status ($\geq$71% for T_3-T_4/N_0 vs. 31–44% for T_3-T_4/N_1-N_3). Histological N status and tumour margins, but not tumour grade, impacted significantly on tumour control. When local control was analysed as a function of the dose to the primary, a nonsignificant negative dose-response relationship was found. The total treatment time was a significant prognostic factor and the time interval between surgery and irradiation proved to be an independent predictor of failure.

732 POSTER

Improving the quality of care in a rural radiation oncology center through use of telemedicine

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Purpose: In 1998, in a rural region of the United States, a telemedicine system was used to link a community radiation oncology center with an academic center of medical excellence in an effort to improve care.

Methods and Materials: A tele-medicine system was installed in both centers to allow remote consultation and review of treatment related radiographs. A 3-D compatible treatment planning system and practice management software allowed on-line review of treatment plans and data. A comprehensive quality assurance program was instituted. In December of 2000, utilization of the tele-radiography system and the trend of quality assurance indicators were assessed.

Results: The teleradiography system was used for peer review of all cases. This system was also used for sub-specialty consultation in the treatment of uncommon malignancies. The integrated treatment planning system allowed the simulation and treatment planning for complicated cases to be performed in the academic center and then the treatment was delivered in the community center. Quality assurance parameters showed a positive trend. Treatment accuracy, as measured by the deviation between the central axis simulation and portal films, was assessed. In 1998, 96% of port films (2,828 of 2,949) were within 0.5 cm and 1% (23 of 2,949) were greater than 1.0 cm. In 2000, 97% (4,111 of 4,245) were within 0.5 cm and 0% (17 of 4,245) were greater than 1.0 cm. Discrepancies between prescribed and treated field parameters improved significantly, from 0.9% (84 events/9,632 patient visits) to 0.3% (25 events/10,162 patient visits) between 1998 and 2000. Physicist chart reviews revealed deviations of 5-10% from prescribed parameters in 1.1% (15 of 1,375) and >10% in 1.2% (16 of 1,375) of charts reviewed in 1998 and deviations of 5-10% in 0.0% (0 of 2,096) and >10% 0.7% (15 of 2,096) in 2000.

Conclusions: Objectively, quality assurance indicators revealed a modest, but measurable, improvement following incorporation of the rural center into a regional oncology network. Subjectively, the teleconferencing system was useful in obtaining expert advice in treating less common malignancies as well providing on-going opportunities for continuing education.

733 POSTER

The concentration-dependency of the radiosensitising effect of gemcitabine and the influence of the rescue agent amifostine in vitro

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Gemcitabine (dFdC) is an active antitumour agent with radiosensitising properties. Since the combined use of dFdC and radiotherapy (RT) also results in an increased toxicity, there is a need for optimisation of this combined approach. Cytoprotective agents might be utilised to reduce these toxic effects. In that respect, amifostine (ami) is one of the most promising cytoprotectors that in vivo selectively protects normal tissues against radiation- or chemotherapy-induced toxicities. We studied the concentration-dependency of the radiosensitising effect of dFdC and the combination of dFdC/RT with ami in various cell lines.

H292 and A549, two lung cancer cell lines, ECV304, a bladder cancer cell line and CAL-27, a carcinoma cell line of the tongue were used in this study. The cells were treated with 0, 1, 2, 4, 6 and 8 nM dFdC for 24 hrs prior to RT. Ami (3.5 mM) and alkaline phosphatase (7.5 U/ml) were added 30 min prior to RT. Cells were irradiated at room temperature by 60Co over a dose range of 0-8 Gy. Cell survival was determined 7 days after RT by the sulforhodamine B test. ID50, radiation dose resulting in 50% cell kill was calculated from the survival curves, fitted according to the linear-quadratic model: survival=exp(-aD-bD2). The radiosensitising effect is reflected by the dose enhancement factor (DEF): ID50(-dFdC)/ID50(+dFdC). The protection factor (PF) was calculated by ID50(+ami)/ID50(-ami).

For ECV304 cells the DEFs varied from 1.39 to 2.98 after treatment with 1 to 6 nM dFdC. H292, A549 and CAL-27 seemed to be less sensitive for the radiosensitising effect of dFdC, with DEFs ranging from 1.05 to 2.67, 1.02 to 2.52 and 1.06 to 2.52 for 1 to 8 nM dFdC, resp. H292, A549 and CAL-27 cells were also less sensitive for the cytotoxic effect of dFdC: IC50 values (conc. causing 50% cell kill) were 8.0, 9.0 and 8.9 nM in H292, A549 and CAL-27 cells, respectively, while in ECV304 the IC50 was 3.1 nM. In combination with dFdC/RT ami clearly showed a protective effect. In H292 cells the PF of ami after treatment with 4 nM dFdC/RT was 1.64 and with 8 nM dFdC the PF was 1.86.

In conclusion, we observed a concentration- and cell line-dependent radiosensitising effect of dFdC in vitro, which seemed to correlate with the sensitivity of the cell line for the cytotoxic effect of dFdC. Ami clearly showed protective effects. Since these protective effects seem to occur selectively in normal tissues ami should be used to further optimise dFdC/RT combinations.

734 POSTER

Loss of alkaline phosphatase expression in breast carcinoma: Implications in the amifostine selective cytoprotection

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Introduction: Amifostine (WR-2721) is an importance cytoprotective agent widely used in clinical oncology to protect normal tissues against radiation and chemotherapy. This is an inactive compound that becomes dephosphorylated to an active thiol (WR-1065) by the enzyme alkaline phosphatase (AF), abundantly found in the normal endothelium. Although a direct evidence is missing, it is believed that amifostine selectively protects normal tissues and not the tumors, as AF is down-regulated in the tumoral vasculature.