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LR-group, and 34 patients were randomized to the Vv-group. No differences were detected between the two groups with respect to vasospasm. 30-day clinical follow-up assessments did not demonstrate any significant group differences neither with BI ($p=0,11$) nor with the 30-day mortality rate ($p=0,65$). However, results of GOS were significantly better in the LR-group ($p=0,018$).

Conclusion: Based on the data in this study there is no difference between the efficacy of the crystalloid-based and the colloid-based preventive 3HT.

Q/4 POSSIBLE ANTI-TUMORIGENIC USAGE OF ANGIOSTATIN IN ONCOTHERAPY

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Keywords: angiostatin, chimera, neovascularisation, anti-tumorigenesis, oncotherapy

Introduction: Angiostatin, a cleavage product of plasminogen kringle domains, is an endogenous angiogenesis inhibitor which can decrease tumorous growth. However, angiostatin is instable so therapeutic usage is complicated.

Our goal was to design an angiostatin-protease chimera which produces angiostatin in situ and in vivo after activation.

Methods: PCR amplified protease, angiostatin cDNA and elements needed for expression in bacterial and eukaryotic cells were ligated into an adequate vector in the same reading frame. The plasmid was used for transfecting different cell lines to achieve in vitro and in vivo production. Aorta ring assay served for monitoring endothelial proliferation in presence of transfected cells. BALB/c mice were injected with transfected and wild type 4T1 breast cancer cells to quantify tumor growth in time.

Results: The chimera was purified from E. Coli and its autocatalysis was observed using SDS-PAGE: the full chimera (80 kDa) appeared if isolation happened in presence of protease inhibitor. Endothelial

proliferation in hanging drop culture stopped when chimera expressing cells grew at the bottom. The aorta ring assay became negative on transfected cell lines. In vivo tumor progression was tested using 20 independent clone lines. Mice injected with transfected 4T1 cells only started growing cancerous tissue 10 days after infection and their tumor progression proceeded with slower rate compared to control animals. Metastases occurring in these mice showed smaller diameters as well.

Conclusion: Our chimera cleaves active angiostatin and develop its anti-tumorigenic effect as demonstrated both in vitro and in vivo experiments. Patent application is in process.

Q/5 BIOMARKER EXPRESSIONS OF HEAD AND NECK CARCINOMAS ARISING AT DIFFERENT ANATOMICAL LOCALIZATIONS

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Keywords: head, neck, cancer, biomarker, expression

AIMS: The objective of our study was to evaluate and to compare the biomarker expressions of head and neck squamous cell carcinomas (HNSCC) arising at different anatomical sites.

PATIENTS and METHODS: We enrolled 124 patients with HNSCC into our study. Among them, 7 had oral, 25 oropharyngeal, 13 hypopharyngeal and 79 had laryngeal cancer. We prepared Tissue Microarrays (TMA) made from the patients' tissue samples and used immunohistochemical staining for the detection of four conventional (Ki67, p53, EGFR, p16ink4) biomarkers and a relatively new (Collagen XVII/BP180) marker. The TMAs were digitalized, and the histological evaluation was performed on computers using a special software. We considered the expressions positive if more than 25% of the tumor cells were stained with the used biomarker. For statistical analysis, we performed Pearson's Chi-square tests.

RESULTS: The p16ink4 and Ki67 expression rates of the tumors in the supraglottic