Hyperoxia influences cancer growth and metastasis. A pilot experimental model

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Introduction: Perioperative care of cancer patients is under scrutiny. Among many factors promoting cancer recurrence and metastasis, high oxygen concentration exposure is underevaluated. While oxygen toxicity is documented in several circumstances, its implication in tumor cell growth and progression is poorly understood.
Objective: To characterize high oxygen concentration exposure effects on tumor progression using a breast cancer murine model.
Material and methods: A highly aggressive breast tumor cell line 4T1 (ATCC®) was injected in mammary gland in 8 week old females BALB/c mice. We divided the animals into 3 groups, each including 6 individuals: G1 – tumor bearing mice with no intervention post inoculation; G2 – primary tumor removal at 2 weeks post inoculation; G3 - primary tumor removal at 2 weeks post inoculation followed by 6 hours of 75% oxygen exposure. In all groups cancer evolution was assessed at 6 weeks by standard pathomorphological evaluation: specimens from the primary tumor, locally recurrent tumor and target organ metastasis were assessed by hematoxylin-eosin staining, and digital microscopy.
Results: Surgically removed primary tumors in G3 group had similar characteristics with those in G2 group and previously described models. At study endpoint, compared with both G2 and G1 groups, G3 animals showed significantly higher tumor burden: larger local recurrence and more metastasis (larger number and dimensions) in liver and lungs, associated with significantly enlarged spleen.
Conclusions: Short term (6 hours) high oxygen (75%) concentration exposure results in significantly more aggressive progression of a 4T1-BALB/c murine breast cancer model.
Key words: hyperoxia, cancer growth, metastasis.

Effects of different sevoflurane concentrations on Akt isoforms in normal and cancer breast cells. An experimental model

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Introduction: Multiple perioperative factors influence cancer patient evolution and outcome. Microenvironmental factors activate different gene programs that enable tumor cell to invade, survive and promote drug resistance and metastasis. The effects of anesthetic drugs on cancer progression is under scrutiny, but published data are controversial and the involved mechanisms unclear. Tumor development implies PI3K/AKT pathway activation. Akt isoforms (1,2,3) are frequently amplified in various malignant tumors and associated with malignant cell survival, proliferation and invasion. Their activation is often observed in human cancers and is associated with decreased survival rate.
Objective: Identification of Akt isoforms activated in sevoflurane exposed breast tumor cells.
Material and methods: Normal breast cells MCF10A (ATCC®) and breast cancer cells MDA-MB-231 (ATCC®) were cultured 2D (standard adhesive culture plastic plates) and 3D (matrigel). Study groups were exposed to different sevoflurane concentrations