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Differental loss E-cadherin in breast carcinomas

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Purpose: Invasion, eventually leading to metastasis, is presented as a result of a balance between the activation of two sets of genes, invasion promotor and invasion suppresor genes. E-cadherin is an invasion suppresor gene product. Our study try to analyse the E-cadherin expression in breast tumors, linked to histological type.

Methods: Tissue material from 35 breast lesions of untreated patients was included in this study. There were made parallel samples for paraffin-embadding and staining with H.E. For immunohistochemistry 5 μm thick cryostat sections were cut from the frozen tissue blocks. We used the MoAb L-cam Boehringer Mannheim, for the E-cadherin staining. Bound antibody was detected using SBC (Boehringer Mannheim).

Results: Histologically normal tissue structures present in breast tumors were studied. The antibody anti-L-cam stained the intercellular borders of the luminal cells of the interlobular ducts. E-cadherin was expressed in all cases of benign and malignant lesions with different grades of intensity. Our results show that the most intensive reactions were remarked at the benign level, and to low grade of malignancy (well differentiated tumors).

Conclusion: E-cadherin is expressed in the majority of breast tumors at different levels. We observed a good correlation between the loss of expression of E-cadherin and high grade of malignancy. A future extension of the study and larger correlation with histological grade, ER status and other oncogenes products can converg to a real test for malignancy evaluation.

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The TNF level in plasma and in primary tumor conditional medium in gastric cancer patients

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Purpose: The TNF is responsible for paraneoplastic symptoms in gastric cancer patients. We have investigated the TNF level in plasma and in primary tumor conditional medium in 21 gastric cancer patients and in 10 donors plasma.

Methods: The bioassay was performed. 16 patients with G4-G3 and in 5 patients with G1-G2 tumor grades. According to Bormann tyes: I type – 4 patients, I – 3, III – 4, IV – 10. Tumor location: upper portion of stomach – 4, middle portion – 6, lower portion – 11. Staging of process: II – 1 patient, III – 20 patients.

Results: The plasma TNF level in G4-G3 tumors was 1.8 ± 0.24 ng/ml, in G1-G2 – 0.514 ± 0.15 ng/ml, in donors plasma – 0.088 ± 0.014 ng/ml. The supernatant TNF level of G4-G3 tumors was 0.12 ± 0.008 ng/ml, and G1-G2 – 0.15 ± 0.02 ng/ml. Thus, we have found the corelation between the tumor grade, agressiveness of tumor process and TNF level in plasma.

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Apoptotic effects of taxol and radiation on the intestinal kript cells of swiss-albino mice

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Purpose: Taxol, a natural product isolated from the bark of the yew tree taxus brevifolia, exhibits significant antitumor activity against various experimental tumors of animals and humans. The drug induces nuclear fragmentation in cultured cells, a feature characteristic of apoptotic cell death. In this experiment, we investigated apoptotic effects of taxol and ionizing radiation on the intestinal kript cells of swiss-albino mice.

Materials & Methods: 170 Swiss-albino mice obtained from Test Animals Brooding Center of Uluda University were used for this experiment. They were 3–4 months old and weighing 20–25 gr. Each group consisted of 5 mice. Taxol was administered bolus IV through the tail vein. Radiation was given with a special device using linear accelerator (6 MV photon). Dose was calculated manually as Dmax dose. There were four groups in the study. First group (taxol alone, n = 15) received IV bolus taxol in three different doses (20, 30 and 40 mg/m^2). Radiation was administered in three different doses (0.25, 0.5 and 1 Gy) to mice in the second group (radiation alone, n = 15). Third group (radiation and taxol, n = 135) was taxol and radiation group. Mice were administered taxol intravenously (20, 30 and 40 mg/m^2) initially. They were irradiated after 12, 24 and 48 hours after taxol administration (0.25, 0.5 and 1 Gy). Fourth group was control group which did not receive either taxol or radiation. Mice were sacrificed 24 hours after taxol or radiation administration using ether anesthesia. Using light microscope, apoptotic and mitotic index were counted on jejunal kript cells of mice that were stained with hemotoxilein-eozin. Differences between groups were evaluated with student t-test statistically.

Results: Mean mitotic index was 2.80 in control group while it was 6.81 in taxol group, 8.79 in radiation group and 3.37 in radiation and taxol group. Although there was no significant difference between taxol alone and radiation alone groups and between control and taxol and radiation groups ($p > 0.05$), control and radiation and taxol groups were significantly different from either taxol alone or radiation alone groups ($p < 0.000$). Mean apoptotic index was 1.00 in control group while it was 19.44 in taxol group, 14.56 in radiation group and 5.78 in taxol and radiation group. Differences in apoptotic index between all groups reached significance ($p < 0.01$). Mean mitotic index was 4.60 in mice irradiated in 12 hours while it was 2.84 in 24 hours and 2.69 in 48 hours. Although there was no difference between 24 and 48 hours ($p > 0.05$), differences between 12 hours and 24 and 48 hours were statistically significant ($p < 0.05$). Apoptotic index was 3.43% in 12 hours, 9.96% in 24 hours and 3.92% in 48 hours. Differences between 24 hours and 12 and 48 hours were statistically significant ($p < 0.05$). There was no difference between 12 and 48 hours ($p > 0.05$).

Conclusion: Addition of taxol to radiation did not seem to be enhancing apoptotic effect of radiation in normal jejunal kript cells.

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Expression on tumors and its metatases of platelet endothelial cell adhesion (PECAM-1), a molecule involvement in transendothelial cell migration and angiogenesis

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Introducction: Platelet endothelial cell adhesion molecule-1 (PECAM-1) is a cell-cell adhesion molecule that is expressed on circulating platelets, on leukocytes, at the intercellular junctions of vascular endothelial cells. This is important molecule that mediate in process of transendothelial cell migrations in the inflammatory response and in formation of new vessels. Some investigators shows expression of PECAM-1, in some cellular tumoral lines, in vitro and they have hypothesed about the importance in the metastatic process.

Aim: To know if the human tumours and its metastases express PECAM-1.

Material and Methods: The tumour biopsies was collected of primary localisation and its metastases in 12 patients and it fixed in formalin and paraffin-embedded. We used the antibody anti CD31, H1/6, in the immunohistochemical staining.

Results: We found expression of PECAM-1 in more of 50% of the cells and ++/+++ or superior in colon, breast, and bladder urinary cancer, also in its metastases. We found expression nor in melanoma, ovarian, kidney and non small cell lung cancer, neither its metastases.

Conclusion: Some tumours and its metastases express PECAM-1. We believe that it will be very important to know which is the effect of regulation of this molecule in process of dissemination and tumoral angiogénesis, actually we are studying this.

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Fibronectin in cell adhesion. It's expression in breast tumors

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Purpose: A major cause of morbidity and death due to the cancer is the metastasis of cells from the primary tumor to distant sites where secondary tumors become established. Tumor cell adhesion to component of extracellular matrix and basement membranes is mediated by specific cell surface receptors that bind to extracellular adhesive proteins. The fibronectin-integrin system has provided a valuable model system for the study of the cellular and molecular mechanismes involved in cell adhesive steps in metastasis. We studied the fibronectin (FN) expression in 35 breast tumors (10 benign and 25 malignant) with different histological types.

Methods: We analysed tissue material from 35 breast tumors of untreated patients. We made parallel detection for histological type on paraffin-embed-