P8a

Serum trace elements and the oxidation status in bladder cancer

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Introduction: A relatively wide range of trace elements are known to play important roles in biological processes, including the oxidative processes. There is epidemiologic evidence of the role of oxidative reactions in cancer induction especially bladder cancer, and most of these reactions result from imbalance of trace elements in the cellular structure. Manganese (Mn) plays an important role in antioxidant defenses and forms part of a Mn-dependent superoxide dismutase enzyme. Iron (Fe) is an essential trace element that is crucial to normal cell functioning and its deficiency is associated with several disease states. On the other hand, Chromium (Cr) is a short-lived oxidative metal in the cells. Zinc (Zn) is another trace element that increases in various oxidative stress states and may play an important role in cancer etiology. In the present study, the concentration of Fe, Cu, Zn, Cr, Mn, malon-dialdehyde (MDA) as a biomarker of lipid peroxidation and total anti oxidant capacity (TAC) as an anti oxidant marker, were determined in the serum of patients with bladder cancer in comparison to healthy subjects.
Materials and Methods: This cross-sectional study was conducted on 51 patients with bladder cancer and 58 healthy volunteers after age, sex, and smoking habits were matched. After overnight fasting, samples were collected. The concentrations of Fe, Cu, Zn, Mn and Cr were measured by atomic absorption spectroscopy, plasma antioxidant status was evaluated using FRAP assay and MDA was measured according to procedure of Ohkawa et al. Comparisons were made using Student's t test.
Results: There was a significant increase in mean Cu and Cu/Zn serum level in bladder cancer patients compared to the control group (p value <0.001). In contrast, the serum Zn level in patients having bladder cancer was significantly lower than in the control group (p value <0.05). Moreover, the serum Fe level was significantly lower in the patients than the control group (p value <0.001). Serum concentration of MDA (p value <0.001) and Cr concentration (p value <0.05) were significantly increased in patients with bladder cancer. There was a significant decrease in serum concentration of Mn (p value < 0.001) and TAC (p value <0.001) of patients in comparison with healthy participants.
Conclusions: In the present study, a relationship was seen between the level of trace elements and the occurrence of bladder cancer, suggesting that an increase in the serum level of Cu and Cr and a decrease in the levels of Zn, Fe and Mn might be important causes of bladder cancer occurrence; however, defining such a cause-and-effect relationship needs several prospective studies to be done, which seems necessary with regard to the high prevalence of this cancer. In addition, regarding the findings of the study, we suggest that a diet rich in anti oxidants, Mn, Fe and Zn and low in Cr and Cu, and also controlling and lowering the ambient standard for Cr in the environment, may have some more protective role in preventing bladder cancer.

Lifestyle, Genetics

P9

Clinical features and prognosis of hereditary breast cancer

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Background: Breast cancer (BC) is the most prevalent malignant tumor in women in developed countries. According to the data of the National Cancer Register, in 2007 the morbidity rate in Ukraine was 61.2 per 100,000 women. Hereditary BC comprises 10–15% of overall BC incidence. The aim of our investigation is to identify clinical, morphological and immunohistochemical features of hereditary BC.
Methods: clinico-genealogical, morphological, immunohistochemical analysis and statistics.
Results: Based on clinico-genealogical analysis, the groups of the patients with hereditary and sporadic BC were delineated. The tumor associations in families of patients with hereditary BC were such as follows: mammary glands – 51.0%, ovary – 21.6%, gastric – 17.6%. It was established that in patients with hereditary BC, exogenous and endogenous risk factors (number of abortions, early menarche, age at pregnancy, number of births, duration of lactation period, smoking, breast injury, and body weight) are of relatively less importance in cancer development than in sporadic BC. Hereditary BC is more common in patients with dyshormonal hyperplasias of the breast. Based on the clinical features, hereditary BC is characterized by smaller size of tumours – 1.5 ± 0.6 cm, while being more malignant (high histological grade) and with worse prognosis. Hereditary BC is characterized by absence or low level of the expression of estrogen and progesterone receptors and BRCA1 protein. The overall survival in patients with hereditary BC was significantly poorer than in patients with sporadic BC (3-year survival rate, 72.42% and 88.06%, $p = 0.02$; 5-year survival rate, 52.6% and 80%, $p = 0.02$; 10-year survival rate, 15.8% and 26.7%, accordingly).
Conclusion: The results of complex examinations may be introduced to the practice of oncological clinics for delineating groups of patients with hereditary BC for their monitoring, which would be advantageous for early BC detection.

P10

Age-dependent derepression of transposons as common cause of ageing and cancer

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Apparently, a specific set of microRNAs and piRNAs expressing in stem cells can restore initial profile of their epigenetic markers through RNAi-directed DNA methylation, thus pluripotent immortal status of these cells is supported for ever and minimal level of the transposons activity is achieved. However, cell differentiation, starting with the most early stages, must be accompanied with repression of genes of some microRNAs from the primary set, otherwise these microRNAs would prevent expression of the stage-specific genes. As a result, differentiating cells can lose slowly the repressive chromatin markers with time, and this will excite the derepression of silent transposons and other mobile elements, therefore increase of DNA damage induced by them, and following activation of cell DNA repair system including mechanisms based on homologous recombination. In our opinion, these mechanisms cause not only the DNA repair, but also unauthorized recombinations