The Yorkshire Terrier, an eleven-year-old spayed female, was presented withaccidentally to have sublumbar mass was detected in ultrasonographic examination with mammary gland tumor. Black to reddish colored masses, located in the visceral peritoneum of sublumbar region, were observed on laparotomy with masectomy of right part. In the laparotomy, we observed reddish masses multifocally located in the serosal membrane of the large intestine. Histopathologic examination of the intestinal and the abdominal mass showed highly invasiveness into the muscle and metastasis of melanocytictumor cells through the blood vessels. Mammary glands showed abnormal hyperplasia of melanocytes and destruction of normal gland by tumor cells and infiltration of some lymphocytes in the pool of melanocytic cells. We have identified in malignant melanoma an angiotumoral complex in which tumor cells occupy a pericytic location along the microvessels with intravasation by immunohistochemistry for S100 protein and PKCα. Histologic findings in this dog was diagnosed as a angiotropic metastatic malignant melanoma.

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P#47

HER-2/neu Protein Expression in Canine Mammary Adenocarcinoma

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The objective of the present investigation was to study the expression of HER-2/neu(c-erbB-2) protein oncogene products in canine mammary neoplastic lesions, sections, of archived paraffin- embedded samples of 49 mammary gland tumors. We analyzed canine mammary gland tumor immunohistochemically using antibodies against human HER-2/neu (c-erbB-2), Epidermal Growth Factor Receptor(EGFR) and Activated Leukocyte Cell Adhesion Molecule (ALCAM/CD166). These Forty-nine tumors were divided into 2 groups: 22 benign (19 adenoma, 3 benign mixed tumors) and 27 malignant tumors (7 simple adenocarcinomas, 5 complex adenocarcinomas, 2 solid carcinoma, 4 sclerosing carcinoma, 5 malignant mixed tumors and 4 malignant myoepithelioma). Classified tumor group, we have attempt immunohistochemistry by using HER-2/neu (c-erbB-2) expression, Epidermal Growth Factor Receptor(EGFR) expression and Activated Leukocyte Cell Adhesion Molecule (ALCAM/CD166) expression on classified tumor group. These suggest that some of the biological and morphological characteristics of the tumor are associated in canine mammary

gland tumors, as also reported for human breast cancer. It was also possible to show that the immunoexpression of HER-2/neu (c-erbB-2) can be a factor in mammary carcinogenesis. This fact opens the possibility of using anti-HER-2/neu(c-erbB-2) antibodies in the diagnosis and treatment of canine mammary gland tumors such as in human case.

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P#48

Inhibition of Renal Fibrosis by ENA-A Resources

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Adriamycin (ADR), which is widely used in the treatment of various neoplastic conditions, exerts diverse side effects in several organs. Nephrotoxicity of adriamycin has been recently documented in a variety of animal species. The present study was designed to investigate the effect of ENA-A Resources, active alkaline mineral water on the ADR-induced fibrosis. This renal fibrosis rat model induced by adriamycin (ADR) had been reported, but it was not often in mice in intraperitoneal (I .P) approach. The study was carried out with male BALB/C mice. Test animals were divided into four groups of fifteen mice each as follows: Group I; control group (saline, I.P). Group II; ADR group (ADR, 5mg/kg body wt, I.P, twice a week). Group III; 5%ENA+ADR group (ADR, 5mg/kg body wt, I P, twice a week and 5% ENA-A Resources solution by drinking water). Group IV; 10%ENA+ADR group (ADR, 5mg/kg body wt, I.P, twice a week and 10% ENA-A Resources solution by drinking water). Intraperitoneal (I.P) injections of adriamycin (ADR) resulted in significant decrease of body weight in all the ADR-treated groups and death of the mice. Ten days after ADR treatment, there was a few histological changes observed. Glomeruli and tubular epithelial cell damaged, inflammatory cells infiltrated, interstitial fibroblasts proliferated, extracellular matrix and collagen deposited, and these histopathological changes became more and more worse went with the course of the experiment. The ENA-A Resources inhibited against ADR-induced renal fibrosis and delayed the life-span compared with ADR-injected mice. These data suggest that administration of ENA-A Resources is a promising approach in the treatment of nephrosis caused by ADR.

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