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ABSTRACTS OF THE IIAR CONFERENCES 1997

despite the intensive care. At autopsy there were no involvement of other sites than cardiac. Grossly were found three nodular lesions, two located in the left ventricule, the first of 23 mm, intramural in the lateral wall, the second of 20 mm, subepycardial and intramural in the posterior wall. The third lesion, intramural of 15 mm was located in the "crista terminalis" of right atrium. Histological and immunohistochemical examination of heart samples, including conduction system, demonstrated the neoplastic cells to be of B lymphocytic clone. A diagnosis of non Hodgkin lymphoma, diffuse large cell type (G, Working Formulation), diffuse centroblastic (Kiel) was made.Vascular embolism and neuronal-gangliar infiltration were seen. There was no evidence of acute myocardial infarction despite clinical signs and symptoms. The Authors believe that the cause of death of this patient was due in part to diffuse invasion of the myocardium by lymphoma cells, supported by the electrocardiographic pattern, in part to the involvement of senoatrial-node and cardiac intrinsic nervous system, causing conduction disturbances. The way of spread was haematogenous, lumen of coronary vessels was invaded by tumor, but also venous permeation and neuronal and gangliar infiltration were seen.

This study suggests that a cardiac involvement must be suspected in patients with lymphoma who present a cardiac symptomatology.

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p53 AND CYCLIN D1 EXPRESSION IN PAROTID GLAND CARCINOMAS

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Salivary grand carcinomas encompass a wide spectrum of histological entities. Defining the molecular abnormalities in such neoplasms might allow a better understanding of understanding of their clinical behaviour. The p53 and cyclin D1 genes play a central role in the regulation of the G1 phase of the cell cycle, and are frequently involved in human tumorigenesis. Point mutations of the p53 gene lead to an increase of the protein half-life, which allows its detection by the commercialy available antibodies. Likewise, recent studies have demondrated a strict correlation between cyclin D1 gene abnormalities (translocation and amplification), and overexpression of the cyclin D1 protein. To our best knowledge, the role of cyclin D1 has never been investigated in salivary gland tumors, while p53 inactivation has been related to a worse prognosis. We have therefore retrospectively investigated by immunohistochemistry the overexpression of the cyclin D1 and p53 genes in a series of 28 parotid gland carcinomas (11 mucoepidermoid, 8 adenocarcinomas NOS, 4 adenoid cystic, 2 squamous cell, 1 acinic cell, 1 salivary duct and 1 epithelial-myoepithelial). All the patients underwent total parotidectomy and 13 of them neck dissection. The patients with lymph node metastasis were treated by post-operative radiotherapy. The immunohistochemical analysis was performed by the ABC method with the antibodies DCS8 (for cyclin D1) and CM1 (for p53). The scoring system was the following: 0: unreactive cases and those expressing bss than 10% of immunoreactive (IR) cells; 1: 10 to 30% IR neoplastic cells; 2: 30 to 50% IR neopladic cells; 3: more than 50% IR neoplastic cells. The cases classified 2 and 3 were considered high expressors (HE), whereas those classified 0 and 1 as low expressors. Whatever the antibody used, the immunoreactivity was restricted to nucleus, and its intensity ranged from weak to strong in a given tumor. Overall, p53 was overexpressed in 12 cases (42.8%) and cyclin D1 in cases (21.4%). No significant association was found between p53 or cyclin D1 expression and the evaluated clinico-pathological parameters of tumor extension (T1-T2 vs T3-T4; p=.4 and p=1, respectively), cinical dage (I-II vs III-IV; p=.4 and p=1, respectively), lymph node (N0 vs N+; p=.2 and p=.8, respectively) and distant (M0 vs M+; p=1 and p=.3, respectively) metastases. Nevertheless, it is worth noting that all the patients who were high expressors of p53 died of their disease.

Our data suggest that cyclin D1 gene might be involved in the pathogenesis of salivary gland carcinoma, and confirm that high expression of p53 could be associated to more aggressive tumors.

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8701-BC BREAST CARCINOMA CELLS SHED MEMBRANE VESICLES IN RESPONSE TO EXTRACELLULAR SIGNALS

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Extracellular shedding of membrane vesicles is frequently described in tumors. Shedding mechanisms are not known but shed vesicles are suggested to participate on mechanisms by which tumoral cells acquire metastatic capability and evade immune surveillance (Taylor and Black, 1986; in: Dev. Biol. 3:33, Plenum Press NY). In this report we describe experiments ment to establish how membrane vesicle shedding is regulated.

In 8701-BC human breast carcinoma cells, vesicle shedding was dependent on the presence and concentration of fetal calf