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HMG2 and PLAG1 protein expression in Pleomorphic Adenoma Tumorigenesis and its recurrence and in the progression to Carcinoma ex-Pleomorphic Adenoma

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The Pleomorphic Adenoma (PA) is the most common neoplasm of salivary glands. Recurrences of APs are common and increase the probability of the malignant transformation giving rise to the Carcinoma Ex Pleomorphic Adenoma (CXPA), which, although rare, is an aggressive tumor with frequent metastasis and death related to the disease. As previously reported in the literature, the Pleomorphic Adenoma Gene 1 (PLAG1) and High Mobility Group AT-hook 2 (HMGA2) genes are associated with the onset and progression of PAs and CXPAs. HMGA2 plays a role in the architectural transcription factor, modulating the three-dimensional conformation of the DNA and consequently modulating the expression of several genes. The PLAG1 gene is involved in cell proliferation through the control of various target genes. In normal tissues, its activity is high during embryonic and fetal development, but in adult life, however, its participation is low or absent. The protein expression of PLAG1 and HMGA2 in 38 cases of PA, 36 cases of Recurrent PA (RPA) and 41 cases of CXAP was analyzed. The histological subtype and degree of tumor progression were considered. A significant association of PLAG1 with PAs was found (89.5%), while the HMGA2 gene protein was presented with a relevant association with the malignant counterpart of the disease (48.78%). A higher prevalence of HMGA2 protein expression in high grade and aggressive tumors considering the histological subtype and degree of tumor progression was observed. PLAG1 protein expression was lower when PA underwent malignant transformation, possibly due to other pathway activation and different clone cells. In addition, PLAG1 protein expression was present mainly in low-grade carcinomas and in cases with the early phase of invasion probably due to its property of regulation of oncogene-induced cell senescence. In CXPA, PLAG1 expression was mostly associated with myoepithelial differentiation. This way, the loss of PLAG1 protein expression can be considered a hallmark of CXPA carcinogenesis, mainly when there is only epithelial differentiation. Our study showed that these genes are promising targets for more effective therapies and consequently lower morbidity due to these neoplasms.

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