

Extended Abstract

Adenoid Cystic Carcinoma of Salivary Gland: An Immunohistochemical Study [†]

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Adenoid cystic carcinoma (ACC) is a basaloid tumour consisting of epithelial and myoepithelial cells in variable morphologic configurations, including tubular, cribriform and solid patterns. ACC accounts for 1% of head and neck cancers and 10% of salivary gland neoplasms. It is characterized by invasive growth, perineural infiltration, early local recurrence and late onset distant metastasis. Radical surgical excision, with or without postoperative radiotherapy, is the treatment of choice. 5-year survival rate is approximately 35% and local recurrences occur despite combined treatment [1]. Thus, novel therapies are urgently needed to supplement the treatment currently available. The study of the molecular mechanisms involved in the neoplasm development is necessary to identify new prognostic markers.

This study investigated the immunohistochemical expression of a panel of molecular markers (C-Kit, Ki-67, p53, and E-Cadherin) to evaluate the correlation with clinicopathological data and prognostic factors, to lay the foundation for further studying the pathogenesis and treatment of the ACC.

The study included surgical resection specimens obtained from 17 ACC. Data were retrieved and cataloged from clinical records and from the archive of the Institute of Pathology, Marche Polytechnic University. 4- μ m serial sections were incubated with the polyclonal rabbit anti-human CD117 antibody (Agilent-Dako), diluted 1:50; with the monoclonal mouse anti-human Ki-67 antigen antibody (Agilent-Dako), diluted 1:50; with the FLEX monoclonal mouse anti-human p53 protein antibody (Dako Omnis); and with the FLEX monoclonal mouse anti-human E-Cadherin antibody (Dako Omnis), in a humidified chamber at room temperature for 1 h. To evaluate the extension of markers expression, the mean percentage of positive cells was determined from the analysis of 1000 cells at $\times 40$ magnification. Staining intensity of CD117 and p53, were scored as “-” (negative staining); “+” (weak staining); “++” (moderate staining); “+++” (intense staining).

No significant associations between the evaluated markers and clinicopathological data were demonstrated. Closely related but not significant correlations were found analyzing the p53 immunohistochemical expression. A lower expression was observed in cribriform compared to solid growth pattern (10.4% vs 38.7%) and in I-II respect to III-IV stage-disease (14.9% vs 45.2%).

The results not reveal a prognostic role for these molecular markers. However, p53 expression could be correlated to worst prognosis. The lack of relationships could be attributed the low records available, due to the rarity of this malignancy. So, further studies on larger series should be conducted. Literature data are extremely heterogeneous and although important advances have been made in exploring the biological bases of ACC, many critical factors remain elusive. Therefore, explore the ACC prognostic indicators is necessary for perfecting the multidisciplinary treatment, improving the curative effect. Moreover, should be performed molecular biology investigations to study the biological function of markers and lay the biological basis for personalized targeted therapies [2].

Conflicts of Interest: The authors declare no conflict of interest.

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