ORIGINAL ARTICLE

IMMUNOHISTOCHEMICAL FEATURES OF THE EXPRESSION OF HUMAN PAPILLOMA VIRUS TYPE 16 IN PLEOMORPHIC ADENOMAS OF SALIVARY GLAND

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ABSTRACT

The aim is to reveal the immunohistochemical features of human papilloma virus type 16 expression in various histological variants of pleomorphic adenomas of the salivary gland.
Materials and methods: The material of the study was surgical and biopsy material from 30 patients with pleomorphic adenomas of the salivary glands, among which in 15 cases mesenchymal was detected, in 10 – mixed, in 5 cases – epithelial histological variant, respectively. Immunohistochemical study was performed, using mouse monoclonal antibody to human papilloma virus type 16. Visualization was performed, using an EnVision™ FLEX detection system. Histological sections of grade III cervical intraepithelial neoplasia (CIN III) were used as a positive control; for a negative control, the procedure was performed without primary antibodies. The immunohistochemical reaction was assessed by a semi-quantitative method by counting the percentage of positively stained cells in the field of view of a microscope × 400. Microspecimens were studied, photoarchived on an Olympus BX-41 microscope.
Results: Expression of human papilloma virus type 16 of varying severity was determined in 26 cases of pleomorphic adenomas of the salivary glands, which was 86.7%. The epithelial component of the pleomorphic adenoma of the salivary gland was characterized by a more pronounced expression of the monoclonal antibody to human papilloma virus type 16 compared to the mesenchymal component of the tumor. The severity of the immunohistochemical reaction with a monoclonal antibody to human papilloma virus type 16 depended on the histological variant of the pleomorphic adenoma of the salivary gland. Epithelial, mixed and mesenchymal variants of pleomorphic adenoma of the salivary gland were characterized, respectively, by the most pronounced, pronounced and moderately pronounced expression of a monoclonal antibody to human papilloma virus type 16.
Conclusions: A comprehensive immunohistochemical study with a monoclonal antibody to human papilloma virus type 16 revealed the presence of a causal relationship between the infection of a patient with human papilloma virus type 16 and development of pleomorphic adenoma of the salivary gland in him.

KEY WORDS: human papilloma virus type 16, immunohistochemistry, pleomorphic adenoma of the salivary gland

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INTRODUCTION

Salivary gland tumors are rare forms of head and neck tumors and benign cases constitute the greatest frequency since only 20 % are malignant. An overall European standardized rate of 4.2-4.9 per 100,000 person-years was reported with a female preponderance (1:1.43) and with an annual 1 % rise in female incidence [1].

Pleomorphic adenoma is the most common salivary gland neoplasm worldwide, accounting for 70-80 % of abnormal growths. It mainly occurs in the superficial lobe of the parotid gland but can also affect the submandibular and minor salivary glands [2].

Etiological factors, causing pleomorphic adenomas, should be well known to minimize their incidence [3]. The importance of human papilloma viruses in the development of pleomorphic adenomas of the salivary glands is a controversial issue. Some scientists note in their studies possible effect of human papilloma virus in pleomorphic adenoma development. On the other hand, some studies do not imply human papilloma virus as a causative agent of salivary gland pleomorphic adenoma [1].

Human papilloma viruses belong to the Papillomaviridae family. Papillomaviruses are one of the most heterogeneous groups of viruses that infect humans and animals. To date, more than 250 papilloma virus types have been identified, and each of these genotypes are associated with infection at particular anatomical sites [4]. Human papilloma viruses, due to differences in DNA sequence, are divided into alpha-, beta-, gamma-, mu- and nu-groups. Human papilloma viruses, taking into account the risk of developing malignant tumors, are classified into viruses of high carcinogenic risk (16, 18, 31, 33-35, 39, 45, 51, 52, 56, 58, 59, 66, 68, 70 types) and low carcinogenic risk (6, 11, 42, 43, 44 types) [5].

Oncogenic potential of papilloma virus type 16 is mainly due to the E6 and E7 oncoproteins, as they are key regulators of the cell cycle [6].