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ORIGINAL ARTICLE

Significance of Epstein-Barr Virus and Human Papillomavirus type 16 in the development of pleomorphic adenomas of the salivary glands

Igor S. Brodetskyi[1], Nataliia M. Savielieva[2], Mykhailo S. Myroshnychenko[2], Shaker J. Tarawneh[2], Liudmyla O. Brodetska[1], Yevheniia A. Hromko[2], Sergey B. Brodetskyi[3], Andrii A. Boiko[1], Yaroslava A. Kulbashna[1], Larysa V. Natrus[1], Yuliia G. Klys[1], Olha I. Koval[1]

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ABSTRACT

Aim: The aim of the study was to determine the role of Epstein-Barr virus and human papillomavirus (HPV) type 16 in the development of pleomorphic adenoma of the salivary gland by identifying the viruses in tumor tissue, oral mucosal epithelium, oral fluid, and blood.

Materials and Methods: The study included 30 patients with a morphologically confirmed diagnosis of pleomorphic adenoma of the parotid salivary glands, who were assigned to group 1. The control group (group 2) consisted of 10 patients who had no tumor or non-tumor salivary gland pathology either during life or at autopsy examination. To identify Epstein-Barr virus and HPV type 16, an immunohistochemical examination of tumor tissue fragments in group 1 and salivary gland tissue fragments in group 2 was performed, as well as polymerase chain reaction (PCR) analysis of blood, buccal scraping, and oral fluid. Statistical analysis of the obtained data was performed using the PAST software (version 4.15, Natural History Museum, University of Oslo, Norway). Mean values of indicators were compared using the Mann-Whitney U test.

Results: The immunohistochemical reaction with mouse monoclonal antibody (MMA) to HPV type 16 was positive in a significantly greater ($p<0.05$) number of cases in group 1 (26 cases, 86.7%) compared with group 2 (3 cases, 42.9%). The number of cases with a positive immunohistochemical reaction with MMA to anti-Epstein-Barr virus was significantly higher ($p<0.05$) in group 1 (8 cases, 26.7%) compared with group 2 (1 case, 14.3%). PCR study of buccal scraping, blood and oral fluid samples from patients in group 1 revealed a lower number of cases with a positive reaction compared with the immunohistochemical examination of tumor tissue. In group 2, this analysis did not detect any positive cases at all. In group 1 PCR enabled the detection of Epstein-Barr virus DNA in buccal scraping in 2 cases, accounting for 6.7%; in oral fluid samples in 4 patients (13.3%); in blood samples in 4 patients (13.3%). The study of buccal scraping, oral fluid and blood by PCR in patients from group 1 enabled the detection of HPV type 16 DNA, respectively, in 1 case (3.3%), 2 cases (6.7%), 2 cases (6.7%).

Conclusions: HPV type 16 appears to play a greater role in the genesis of pleomorphic adenomas of the parotid salivary glands compared with Epstein-Barr virus. Immunohistochemical examination of tumor tissue is an effective method for demonstrating the role of HPV type 16 and Epstein-Barr virus in the development of pleomorphic adenomas of the salivary glands. PCR analysis of oral fluid, blood, and buccal scraping is not a sufficiently sensitive or reliable method for identifying the investigated viruses in patients with pleomorphic adenomas.

KEYWORDS: Epstein-Barr virus, human papillomavirus type 16, pleomorphic adenoma, salivary gland, immunohistochemistry, polymerase chain reaction

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INTRODUCTION

Salivary gland tumors are uncommon lesions that account for approximately 3% to 6% of all head and neck tumors. The annual incidence of salivary gland tumors reaches 4-13.5 cases per 100 000 people in the world [1, 2]. They are characterized by clinical and biological complexity, histopathologic diversity [2, 3]. In the fifth edition of the World Health Organization classification of head and neck tumors, salivary gland tumors are represented by more than 20 malignant variants and more than 15 benign variants [4]. These tumors can develop in major or minor salivary glands; however, they are more frequently diagnosed in the parotid gland [4]. The most common malignant tumors of salivary glands are mucoepidermoid carcinoma and adenoid cystic carcinoma. Pleomorphic adenomas are the most common benign tumors, accounting for 85% of all salivary gland neoplasms and 60% of benign tumors [5, 6].

Pleomorphic adenomas are found mainly in the superficial lobe of parotid gland (90% cases). The deeper lobe of parotid gland is involved in pathological process in less than 10% of cases [7]. This tumor mainly develops in women (female-to-male ratio of 1.6 to 1) at a mature age (30-50 years), characterizes by slow growth, scant clinical symptoms and can relapse with recurrence rates of up to 6.8% [7, 8]. The transformation of pleomorphic adenoma into a malignant tumor, according to various researchers, occurs in 3.3-6.2%

of cases [9]. Morphologically pleomorphic adenoma is characterized by structural diversity and heterogeneity, which is due to the different ratio of the epithelial (parenchymal) and mesenchymal (stromal) components, forming this tumor; the complexity and multicomponent structure of the parenchyma itself and the stroma of the tumor [8].

Understanding the etiopathogenesis of pleomorphic adenomas is important for improving existing treatment approaches, developing new therapeutic methods, and predicting recurrence and malignant transformation of these tumors. Studies investigating the etiopathogenesis of pleomorphic adenomas remain rather limited, which is due to the relatively low incidence of these tumors within the overall structure of oncological diseases. According to various researchers, the development of pleomorphic adenomas may be associated with harmful habits (alcohol consumption and tobacco smoking), unbalanced nutrition, exposure to ionizing radiation and chemical agents, hazardous working conditions, genetic factors, autoimmune diseases, and immunodeficiency states [1, 10].

Recently, an increasing number of studies have appeared in the literature demonstrating the role of viruses, particularly the HPV type 16 and Epstein-Barr virus, in the genesis of tumor development [11]. Thus, the role of HPV type 16 in the development of cervical, oropharyngeal, and anal cancers, among others, has been proven. Epstein-Barr virus is associated with the development of lymphoproliferative disorders and nasopharyngeal carcinoma [12]. The role of Epstein-Barr virus and HPV type 16 in the development of pleomorphic adenomas of the salivary glands remains controversial and unresolved. This highlights the relevance of conducting a comprehensive study aimed at identifying HPV type 16 and Epstein-Barr virus in patients with pleomorphic adenomas, which may help to confirm the virus-associated genesis of this benign salivary gland tumor.

AIM

The aim of the study was to determine the role of Epstein-Barr virus and HPV type 16 in the development of pleomorphic adenomas of the salivary glands by identifying the viruses in tumor tissue, oral mucosal epithelium, oral fluid, and blood.

MATERIALS AND METHODS

The study included 30 patients with a morphologically confirmed diagnosis of pleomorphic adenoma of the parotid salivary gland (mesenchymal variant in 15 cases, mixed variant in 10 cases, and epithelial variant in 5 cases), who were assigned to group 1. The control group (group 2) consisted of 10 patients who had no tumor or non-tumor salivary gland pathology either during life or at autopsy examination. The causes of death in group 2 patients were acute heart failure and dislocation of the brain stem.

To identify Epstein-Barr virus and HPV type 16, an immunohistochemical examination of tumor tissue fragments in group 1 and salivary gland tissue fragments in group 2 was performed, as well as PCR analysis of blood, buccal scraping, and oral fluid.

Immunohistochemical examination was performed on adhesive Super Frost Plus slides (Menzel, Germany) using MMA to HPV type 16 (clone CAMVIR-1, Diagnostic BioSystems, USA) and anti-Epstein-Barr virus (LMP, clone CS.1-4, Dako, Denmark). The Master Polymer Plus Detection system (Peroxidase, DAB chromogen) (Master Diagnostica, Spain) was used, while citrate buffer (pH 6.0) and EDTA buffer (pH 8.0) were applied for high-temperature antigen epitope retrieval. The histological specimens were examined using a ZEISS Primostar 3 microscope (Carl Zeiss, Germany) equipped with an integrated color digital camera.

In group 1, blood and oral fluid samples, as well as buccal scraping, were collected from patients after surgical treatment for pleomorphic adenoma of the salivary glands, whereas in group 2 these samples were obtained during hospitalization. Peripheral blood samples were collected into sterile closed-system tubes with a volume of 2.5 mL containing ethylenediaminetetraacetic acid. Oral fluid samples were collected into a special sterile container. Buccal scraping was obtained using a sterile disposable probe from the inner surface of both cheeks and placed into a sterile tube containing a special transport medium. DNA extraction from the obtained biological samples was performed using the commercial Sansure Biotech kit (S50016E, China) based on magnetic particles, according to the manufacturer's instructions. A commercial Sansure Biotech test system (S3015E) was used for the detection of Epstein-Barr virus DNA, while Sansure Biotech test system (S3019E) was used for the detection of HPV type 16 DNA. Real-time PCR was carried out using the Applied Biosystems™ 7500 Real-Time PCR System (Life Technologies Corporation, USA).

Statistical analysis of the obtained data was performed using the PAST software (version 4.15, Natural History Museum, University of Oslo, Norway). Mean values of indicators were compared using the Mann-Whitney U test. Differences were considered statistically significant at $p<0.05$.

RESULTS

The immunohistochemical reaction with MMA to HPV type 16 was positive in a significantly greater ($p<0.05$) number of cases in group 1 (26 cases, 86.7%) compared with group 2 (3 cases, 42.9%) (fig. 1). In group 1, it was noteworthy that the parenchymal component of pleomorphic adenoma demonstrated more pronounced expression of the studied MMA compared with the stromal component. In patients with the epithelial variant of the tumor, a positive reaction was detected in all cases (5 cases, 100%); in the mixed variant, in 9 cases (90%); and in the mesenchymal variant, in 12 cases (80%) (table 1).

The number of cases with a positive immunohistochemical reaction with MMA to anti-Epstein-Barr virus was significantly higher ($p<0.05$) in group 1 (8 cases, 26.7%) compared with group 2 (1 case, 14.3%) (fig. 1). In group 1 patients, light microscopy revealed more pronounced expression in the parenchymal component of the tumor compared with the stromal component. In patients with the epithelial variant of pleomorphic adenoma, a positive reaction was

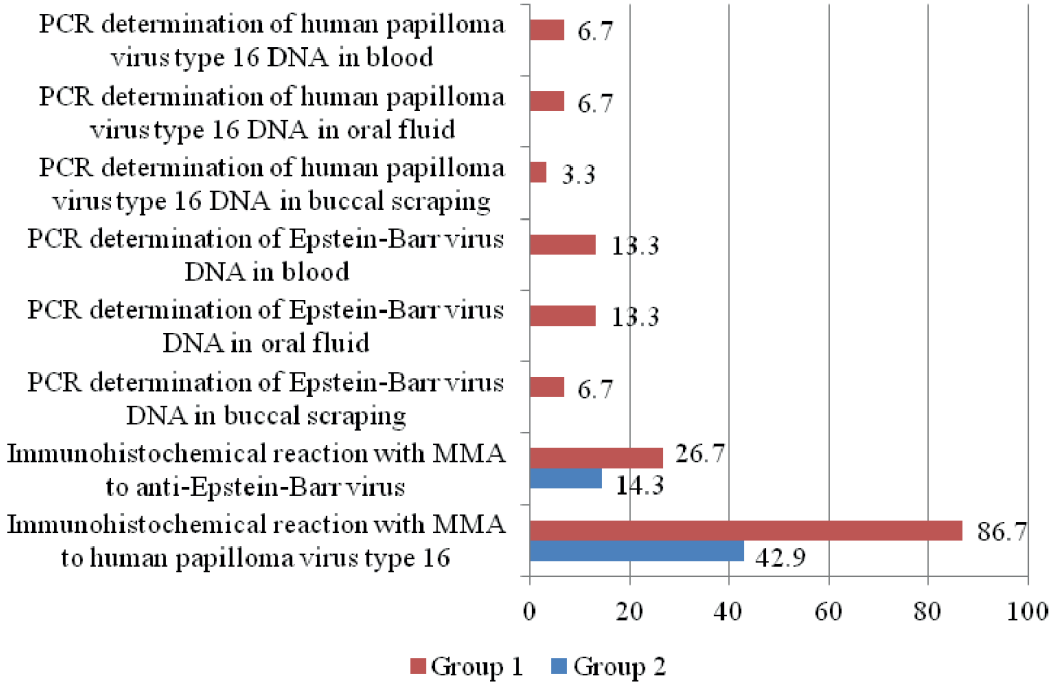


Fig. 1. Total number of cases with positive immunohistochemical reaction and PCR in groups 1-2.

Source: Own materials

Table 1. Results of immunohistochemical studies and PCR in group 1

Study name	Histological variant of pleomorphic adenoma					
	Mesenchymal (n=15)		Mixed (n=10)		Epithelial (n=5)	
	Number of cases with a positive reaction					
	Abs.	Rel., %	Abs.	Rel., %	Abs.	Rel., %
Immunohistochemical reaction with MMA to HPV type 16	12	80	9	90	5	100
Immunohistochemical reaction with MMA to anti-Epstein-Barr virus	1	6.7	3	30	4	80
PCR determination of Epstein-Barr virus DNA in buccal scraping	-	-	1	10	1	20
PCR determination of Epstein-Barr virus DNA in oral fluid	-	-	2	20	2	40
PCR determination of Epstein-Barr virus DNA in blood	-	-	2	20	2	40
PCR determination of HPV type 16 DNA in buccal scraping	-	-	1	10	-	-
PCR determination of HPV type 16 DNA in oral fluid	-	-	2	20	-	-
PCR determination of HPV type 16 DNA in blood	-	-	2	20	-	-

Source: Compiled by the authors of this study

observed in 4 cases (80%); in the mixed variant, in 3 cases (30%); and in the mesenchymal variant, in 1 case (6.7%).

PCR study of buccal scraping, blood and oral fluid samples from patients in group 1 revealed a lower number of cases with a positive reaction compared with the immunohistochemical examination of tumor tissue. In group 2, this analysis did not detect any positive cases at all. In group 1 PCR enabled the detection of Epstein-Barr virus DNA in buccal scraping in 2 cases (1 patient with the mixed variant of the tumor and 1 patient with the epithelial variant), accounting for 6.7%; in oral fluid samples in 4 patients (2 cases with the mixed variant and 2 cases with the epithelial variant); and in blood samples in 4 patients (2 patients with the mixed variant and 2 patients with the epithelial variant), accounting for 13.3%.

The study of buccal scraping, oral fluid and blood by PCR in patients from group 1 enabled the detection of HPV type 16 DNA, respectively, in 1 case (3.3%) with a mixed variant; in 2 cases (6.7%) with a mixed variant; in 2 cases (6.7%) with a mixed variant.

DISCUSSION

The evidence supporting the role of viruses in oncogenesis is based on the detection of viral particles, viral DNA or RNA, as well as viral proteins using both classical and modern research methods. The detection and analysis of viruses remain complex and challenging issues [13]. In the present study, in order to determine the role of Epstein-Barr virus and HPV type 16 in the development of pleomorphic adenomas of the salivary glands, an immunohistochemical examination of tumor tissue was performed, along with PCR study of buccal scraping, oral fluid, and blood samples.

Immunohistochemical examination, as is well known, is based on the detection of viral antigens in biopsy specimens using specific antibodies. In the present immunohistochemical study, MMA to HPV type 16 and anti-Epstein-Barr virus were used. Analysis of the immunohistochemical findings revealed a greater number of cases with a positive reaction with MMA to HPV type 16 in patients with pleomorphic adenomas (26 cases, 86.7%) compared with the control group (3 cases, 42.9%). The number of cases with a positive immunohistochemical reaction with MMA to anti-Epstein-Barr virus was also higher in group 1 (8 cases, 26.7%) compared with group 2 (1 case, 14.3%). Detailed characteristics of the expression of the studied MMA in patients with pleomorphic adenomas have been previously described and published [12]. The results of the immunohistochemical study suggest a greater role of HPV type 16 in the development of pleomorphic adenomas compared with Epstein-Barr virus.

The involvement of HPV type 16 in tumor development occurs through classical oncogenic mechanisms. The viral oncoproteins E6 and E7 induce the inactivation and degradation of the tumor suppressor proteins p53 and pRb, leading to loss of cell cycle control, inhibition of apoptosis, and uncontrolled proliferation of epithelial cells [14, 15]. E6 activates the telomerase enzyme (hTERT), promoting cellular immortalization, that is, the loss of limitations on the number of cell divisions, thereby enabling cells to proliferate almost indefinitely [14, 15]. E7, in turn, stimulates the expression of S-phase factors, thereby enhancing the replicative activity of cells [16]. Some studies have also reported the involvement of HPV type 16 in the epigenetic reprogramming of tumor cells. This epigenetic remodeling includes hypermethylation of tumor suppressor gene promoters, modification of histones and microRNAs, which results in the functional «silencing» of genes responsible for the regulation of the cell cycle and apoptosis [17, 18].

The potential oncogenic mechanisms of Epstein-Barr virus are mainly associated with latent infection and the expression of viral proteins such as LMP1 and EBNA2. LMP1 functions as a «pseudo-receptor» that activates intracellular signaling pathways, thereby stimulating cell proliferation, inhibiting apoptosis, and promoting the production of pro-inflammatory cytokines [19]. EBNA2 functions as a transcriptional regulator that activates the expression of both viral and cellular genes involved in the proliferation and transformation of infected cells [20].

Epstein-Barr virus is also capable of inducing the above-mentioned epigenetic changes (DNA methylation and histone modifications), which affect the expression of cellular genes [21], as well as promoting immune evasion, that is, the ability of tumor cells or viruses to avoid recognition and destruction by the immune system [22, 23]. Numerous studies have failed to detect a stable concentration of the investigated virus in different biological media, or have identified it in tissues only in low quantities, which suggest that it may act as a modifying factor of the tumor microenvironment rather than as a direct trigger of transformation [24].

Modern and accurate methods for the diagnosis of viral infections include PCR, which allows the identification of the virus within a short period after infection [25]. In the present study, PCR was used to identify the DNA of HPV type 16 and Epstein-Barr virus in buccal epithelium, oral fluid, and blood samples obtained from patients who underwent surgery for pleomorphic adenomas of the salivary glands. Using this method, Epstein-Barr virus DNA was detected in the buccal epithelium of 2 patients (6.7%), in oral fluid samples of 4 patients (13.3%), and in blood samples of 4 patients (13.3%). PCR also enabled the detection of HPV type 16 DNA in the buccal epithelium of 1 patient (3.3%), in oral fluid samples of 2 patients (6.7%), and in blood samples of 2 patients (6.7%).

The comparative analysis performed by the authors revealed discrepancies between the results of PCR study of blood, oral fluid, and buccal scraping and the immunohistochemical examination of tumor tissue. These discrepancies may indicate, first, the low effectiveness of PCR or of the investigated biological media for viral identification; second, the absence of viral replication or viral DNA in the studied media despite the presence of viral antigens in tumor tissue. The observed discrepancies may also be explained by the fact that the viral load in the investigated biological media was below the sensitivity threshold of the applied test systems. When HPV type 16 and Epstein-Barr virus enter the oral cavity or bloodstream from the salivary gland, they may alter their morphofunctional properties, which may prevent their complete detection by PCR and could account for the identified discrepancies.

Some studies have reported low sensitivity and specificity (16-27%) for the detection of human papillomavirus in oral fluid and oral mucosal epithelium using PCR [26]. According to some studies, the difficulty of detecting human papillomavirus in the oral cavity is associated with the presence of a large number of bacteria (approximately 50-100 billion) [27].

CONCLUSIONS

HPV type 16 appears to play a greater role in the genesis of pleomorphic adenomas of the parotid salivary glands compared with Epstein-Barr virus. Immunohistochemical examination of tumor tissue is an effective method for

demonstrating the role of HPV type 16 and Epstein-Barr virus in the development of pleomorphic adenomas of the salivary glands. PCR analysis of oral fluid, blood, and buccal scraping is not a sufficiently sensitive or reliable method for identifying the investigated viruses in patients with pleomorphic adenomas.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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