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Epidemiological study of HPV in oral mucosa through PCR

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Abstract

The Human Papillomavirus (HPV) belongs to the *Papillomaviridae* family and has a capsid and a single DNA strand. Its infection occurs mainly through sexual intercourse, having an important tropism for skin and mucosal cells.

Aim: To evaluate the HPV presence in normal oral mucosa of asymptomatic subjects and; in parallel, to correlate social behavioral habits with the virus.

Materials and Methods: Contemporary cohort cross-sectional study. The HPV was found by PCR, using general primers MY09/11 in 125 oral mucosa samples submitted to DNA extraction and PCR to search for the beta-globin gene in order to assess the quality of the extracted DNA. In parallel, we carried out a study of behavioral issues associated with the patients.

Results: All the samples had a positive diagnosis of the beta-hemoglobin gene. HPV was diagnosed in 23.2% of the samples analyzed.

Conclusion: The virus was present in 29 of the 125 patients, without them having any clinical-pathological manifestation associated with the HPV. As to the social behavior of the patients, we concluded that oral sex is statistically correlated to the virus, and besides the HPV has been statistically more present in female patients.

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INTRODUCTION

The human papillomavirus (HPV) belongs to the Papillomaviridae family. They are small, epitheliotropic and have about 55 mm in diameter. Their genome is made up of 7,200 to 8,000 base pairs with molecular weights of 5.2 x 106 daltons. The HPV is made up of a capsid with 72 capsomeres of icosahedral structures, with a lipoprotein envelope and one single circular double DNA molecule[1]. Its genome is divided into E (Early - coding the proteins which are initially produced) and L (Late - codes proteins which will be produced after those from the E region). These two regions represent 45% and 40% of the viral genome, respectively, and have the open reading frames (ORFS) E1, E2, E4, E5, E6, E7, L1 and L2. It is known that the E1 gene is involved in viral DNA replication and maintenance; E2 regulates viral transcription and E4 codes a protein which destroys cytoplasmic keratin, producing the koilocytosis halo image. It is believed that E5, E6 and E7 are involved in cell transformation and degradation, while L1 and L2 code the viral capsid proteins. Between the E and L regions, there is the non-coding region called LCR (long control region) or URR (upstream regulatory region), representing 15% of the viral genome involved in the control of viral gene expression[2].

HPV infection is basically through sexual intercourse. As such, both men and women are involved in the epidemiological chain of the infection and are capable, at the same time, of being asymptomatic carriers, transmitters and victims of HPV infections. In these regards, the risk factors are clearly associated with the individual's sexual behavior. The most important are: starting sexual intercourse at an early age, having a large number of sexual partners during one's life and having sexual contact with high-risk individuals (i.e. men having frequent contact with female prostitutes; in the case of women, having intercourse with men who have multiple sexual partners)[3].

HPV infects epithelial and mucosal cells, which can cause a number of hyperplastic lesions. Therefore, this virus can be mucosotropic, which infect the oral, respiratory and genital mucosae; and cutaneotropic, found in immune-competent individuals and in those with verruciform epidermoplasias[4]. Since the HPV infects only the epithelial layer, not going beyond the basal membrane, the primary immune exposure must happen by means of mechanisms present in this layer. The infected cell membrane expresses only the E5 protein, and this small quantity of surface viral antigens may damp the immune response. E5 binds and inactivates the protein that is needed to process the antigens. Since the epithelial cell is not a good antigen-presenting cell, the HPV remains inside it without being recognized by the immune system. Because the HPV does not cause host cell lysis or death, the virus remains isolated from contact with immune system cells, which would trigger the recognition process[5].

The viral infection may cause localized clinical, subclinical or latent lesions. In general, the HPV follows the classic viral productive cycle: adsorption, penetration, transcription, translation, DNA replication and maturation. However, in some cases, such process does not happen in a complete way, since the virus may integrate the genome of the host cell and cause carcinogenesis. In benign lesions, the virus is in its circular form, called episomal - not integrated to the host cell genome, and in a large number of copies. In malignant lesions, it is integrated to the host cell genome. Notwithstanding, it is possible to find episomal forms in the malignant cells and, once integrated, the virus cannot be reverted to its episomal form[6].

Viral types of high and low malignance risks differentiate according to the transformation capacity of E6 and E7 oncoproteins, coded by genes E6 and E7. E6 and E7 oncoproteins bind to p53 and pRB proteins, respectively, regulators of the cell cycle, considered cancer suppression. This phenomenon unblocks the cell cycle and causes genetic instability, which causes additional genetic changes, causing cancer by preventing apoptosis, leading to cell immortalization. This process is found only in high risk virus, and it is not seen in their low risk counterparts[7].

It is estimated that in Brazil there are from 500 thousand to 1 million new cases per year of HPV infection, while 80 thousand cases of AIDS, 200 thousand to 500 thousand cases of herpes, 100 thousand cases of syphilis and 800 thousand cases of gonorrhea are registered[8]. The presence of anogenital HPV 6/11 and 16/18 in the oral mucosa could mean orogenital transmission, which would make this virus an important cofactor in the development of oral cancer, as it is considered in the uterine cervix[9]. The HPV prevalence in the oral mucosa, macroscopically normal, varies substantially in the literature: between 0 to 81.1%, with a mean of 10%. The infections are not always seen macroscopically. Thus, the HPV infection may be classified into: (a) latent, which may only be diagnosed by means of molecular biology assays; (b) subclinical, which does not bear clinical symptoms, but there are subtle changes which can be detected by diagnostic methods such as peniscopy, colpocytology, colposcopy and/or biopsy; (c) clinical, in which there are evident lesions upon clinical exam. From the molecular viewpoint, it is not known how an HPV infection remains latent, and anther develops macroscopic lesion of intense viral replication[10]. Progresses in the field of molecular biology and genetics have contributed substantially to study these viruses. Of all HPV DNA detection techniques, the polymerase chain reaction (PCR) is the most sensitive[11].

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