ORAL HUMAN PAPILLOMAVIRUS: ABOUT A CASE OF SQUAMOUS PAPILLOMA

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Abstract: HPV is the papillomavirus which selectively infects humans; it exhibits a tropism for epithelial cells in the skin and mucosal areas of the body, but can survive free in the environment for several months, a fact which contributes significantly to its virulence. HPV causes many lesions in the oral cavity which are usually benign. The source of infection can be the skin, genitals or anal area of an infected person. HPV has been proved to be the causative agent of cervical cancers without doubt, but its role as an etiologic agent of oral cancers needs to be evaluated. We report in this article a case of squamous papilloma of uvula and what is known about oral HPV infection.

Keywords: Human papillomavirus, papilloma, transmission, oncogenic.

I. INTRODUCTION

Human papilloma virus (HPV) has a wide disease spectrum affecting the cutaneous and mucosal areas of the body, ranging from benign common warts to invasive carcinoma. HPV infections have been reported in a number of body sites, including the ano-genital tract, urethra, skin, larynx, tracheobronchial mucosa, nasal cavity, paranasal sinus, and oral cavity [1]. Oral HPV infection may be associated with different diseases of oral cavities which are usually benign-like oral papillomas, oral condylomas, and focal epithelial hyperplasia [1].

The objective of this article is to report a case of oral papilloma and to review the epidemiology, clinical aspect, diagnosis and the oncogenic potentiality of oral HPV infection.

II. CASE DESCRIPTION:

It is about a 17-year-old patient; immunocompetent, with no particular history of sexual contact; who has been feeling oropharyngeal foreign body for 5 months.

The clinical examination revealed a rounded, painless, clear-contoured, pale pink lesion, the surface of which is bristling with papillary projections, pediculated at the uvula, measuring 1cm (Fig. 1).

The patient underwent a biopsy-exeresis under local anesthesia by strangulation technique. The histopathological study came back in favour of papilloma.

IV. DISCUSSION

Human papillomavirus (HPV) infection accounts for approximately 5.2% of the worldwide human cancer burden including the cancer of the anus, genital tract, and oropharynx [2].

There are over 150 types of HPV, which have been categorized into “high-risk” and “low-risk” types based on their potential to induce malignancy in cervical cancer [2].

Epidemiology and natural history of oral HPV infection have not been well established. However, recent studies suggest oral HPV prevalence is substantially lower than genital HPV infection. It is ranging from 0.9 to 7.5% among young adults aged 16-49 years [2]. In the National Health and Nutrition Examination Survey (NHANES) study, oral HPV prevalence was notably higher in men than women (10.1% vs. 3.6%) [3].
The mode of transmission of oral HPV is unclear. Since HPVs can establish latent subclinical infection, they may have been acquired early in life, that is, originated at birth from cervical infection in the mother (perinatal) but most transmission is horizontal. HPVs can be sexually transmitted (orogenital contact) and also through nonsexual fomite transfer (moist towel sharing for example). There is no evidence for auto-inoculation or other non-sexual transmission, although we cannot yet exclude this possibility [4]. That was illustrated by our clinical case without a sexual history.

The most consistent risk factors associated with prevalent oral HPV infection are: male gender, sexual behaviours, tobacco exposure and HIV infection [3].

The human papillomavirus is responsible for different types of proliferative lesions of the oral mucosa according to subtypes of HPV; such as: squamous papilloma (HPV 6 and 11), verruca vulgaris (HPV 2 and 4), condyloma acuminatum (HPV 6 and 11), and focal epithelial hyperplasia (HPV 13 and 32) [1].

Diagnosis is based on clinical manifestation confirmed by histopathology. The HPV causes epithelial proliferation characterized by epithelial thickening, prominent keratohyalin granules, acanthosis, and sometimes hyperkeratosis. Koilocytes indicate the presence of productive HPV infection in exfoliated cells and biopsy specimens [1].

PCR (polymerase chain reaction), is the most commonly used tool in the detection of HPV DNA. Although entering the practice for gynaecological lesions, PCR is an examination that remains very limited for oral lesion; it allows giving the virus subtype [5].

The management of oral HPV is essentially physical: surgical excision, cryotherapy, electrocoagulation, laser CO2; and chemical: keratolytics and cytostatics. In some case abstention with regular follow up is recommended [5].

The evolution is almost benign. The contribution of HPV to head and neck carcinogenesis is unclear. However, HPV 16 DNA is detected in oral rinses of 50% of cases with HPV-16-positive oropharynx cancer patients [2].

I. CONCLUSIONS

Oral papillomas are the rare form of oral HPVs. If the clinical diagnosis is generally easy, confirmation requires an anatomopathological analysis. Oncogenic HPVs are associated with oral malignancies, but its prevalence varies widely in different studies. Although study results are mixed, it seems possible that smoking and alcohol use may interact with HPV infection to increase a person’s risk of oral cancer. So, oral HPV infections need to be studied and investigated deeply so that it can guide us for future cancer prevention programs, including oral HPV vaccination for oral HPV infections.

Figures:

Figure 1: clinical image of a squamous papilloma pediculated at the uvula
REFERENCES


