# Oral manifestations of human papillomavirus: review of the literature

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## Abstract

**Human papillomavirus (HPV) is one of the most common sexually transmitted infections, with significant implications for oral and oropharyngeal health. While low-risk HPV types (e.g., HPV 6, 11) are associated with benign lesions, high-risk types (e.g., HPV 16, 18) are linked to the development of malignancies, such as cervical and oropharyngeal cancers.**
**This study is a literature review based on articles selected from PubMed, Dialnet, and Dentistry & Oral Sciences Source databases. Only recent in vivo and in vitro studies published in English or Spanish were included.**
**Studies suggest that high-risk HPV types, particularly HPV 16, are strongly associated with oropharyngeal carcinomas, while low-risk types are prevalent in benign lesions. Vaccination emerges as the primary preventive strategy.**
**HPV vaccination is crucial in preventing HPV-related diseases, while early diagnosis and timely treatment, including surgery and laser therapy, are essential for managing oral lesions. Ongoing research is necessary to improve the understanding and treatment of HPV infections.**

**Keywords: Oral papillomavirus, Human papillomavirus, Oropharyngeal cancer**

## Introduction

Human Papillomavirus (HPV) is a small, non-enveloped, double-stranded DNA virus belonging to the Papillomaviridae family (1–6). It is one of the most prevalent sexually

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transmitted infections (STIs) worldwide, infecting epithelial cells in both the skin and mucosal membranes (7–12). Over 200 HPV genotypes have been identified and classified into five genera based on genomic similarities. HPV types 16, 18, 6, and 11 are most commonly associated with various health conditions, ranging from benign lesions to malignancies, particularly cervical and oropharyngeal cancers (13–18).

HPV is a major etiological factor in several cancers, notably cervical cancer, and also contributes to cancers of the anus, vulva, vagina, penis, and oropharynx (19–27). While most HPV infections are self-limiting and asymptomatic, persistent infection with high-risk HPV strains can lead to precancerous lesions, which may eventually progress to invasive cancer if untreated (28–36).

### *HPV Structure and Genomic Organization*

HPV has a simple yet highly effective structure for establishing infection. Its genome is circular and about 8,000 base pairs long, encapsulated in a non-enveloped icosahedral capsid of two proteins, L1 and L2 (37–45). The viral genome encodes 9-10 open reading frames (ORFs), which include both structural proteins (such as L1 and L2) and non-structural proteins (such as E1, E2, E6, and E7) (46–54).

- L1 and L2 are capsid proteins central to virus formation and the immune response (55–60).
- E1 is essential for viral genome replication (61–67).
- E2 regulates viral replication and gene expression (68–74).
- E6 and E7 are key oncogenes that inactivate tumor suppressor proteins (like p53 and Rb), contributing to uncontrolled cell proliferation and cancer development (75–82).

### *Genotypes and Tissue Tropism*

HPV infection can manifest in various forms depending on the genotype and tissue infected (Fig.1) (83–92).

1. Cutaneous HPV types: Low-risk types (e.g., HPV 1, 2, and 4) typically cause benign skin warts, such as common warts and plantar warts (93–99). These types generally do not lead to malignancy but can be a source of discomfort and stigma (100–106).
2. Mucosal HPV types: Mucosal HPV types include both low-risk types (e.g., HPV 6 and 11) and high-risk types (e.g., HPV 16 and 18) (107–114). Low-risk types cause genital warts, which can lead to significant distress but are generally benign (115–118). High-risk types, particularly HPV 16, are implicated in the development of cancers such as cervical, anal, vulvar, and penile cancers. HPV 16 is the most oncogenic strain, linked to the majority of HPV-related cancers (119–123).
3. Oral HPV types: HPV, especially HPV 16, is also associated with oropharyngeal cancers. The incidence of these cancers is rising, particularly among younger, sexually active individuals. HPV-related oropharyngeal cancers differ from those caused by tobacco and alcohol use, which are more common in older populations (124–128).
4. Respiratory HPV types: Types like HPV 6 and 11 can cause recurrent respiratory papillomatosis (RRP), in which warts form in the airways. This condition leads to breathing difficulties and is often acquired during childbirth (116,129–131, 291).

### *Transmission of HPV*

HPV is primarily transmitted through direct skin-to-skin contact, especially during vaginal, anal, or oral sex. The virus can be contracted even without visible symptoms or warts. Factors such as early sexual activity, multiple partners, and unprotected sex increase the risk of HPV transmission. Although condom use can reduce transmission, it is not completely protective, as it doesn't cover all areas of potential contact (132–136).

In addition to sexual transmission, HPV can be transmitted non-sexually through skin-to-skin contact, as seen in cases of cutaneous warts. Additionally, HPV can be passed from mother to child during childbirth, particularly in cases of respiratory papillomatosis (137–143).

### *HPV Infection and Lifecycle*

HPV infects basal epithelial cells through microabrasions or other disruptions in the epithelial surface. Initially, the virus remains latent in the basal layer, replicating minimally. As the infected cells differentiate and migrate toward the surface, the viral genome replicates and is packaged into new virions (144–150).

Persistent infection with high-risk HPV types can cause cellular changes, leading to precancerous lesions. If

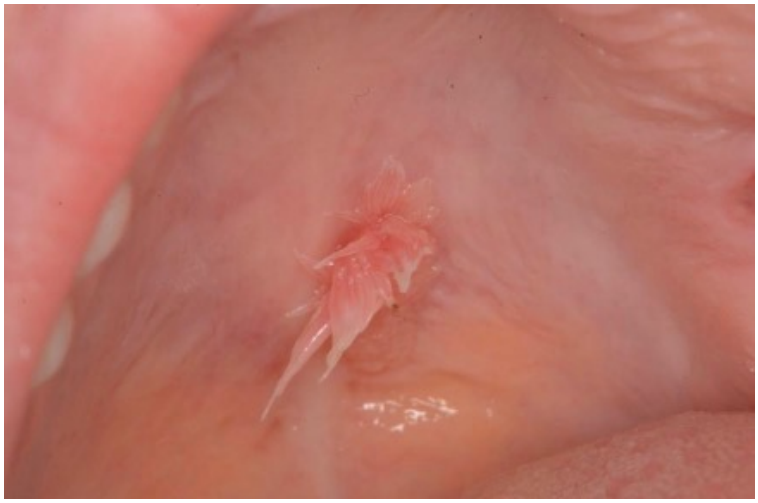

Figure 1. Typical oral papilloma lesion

untreated, these lesions may progress to invasive cancer (151–157). The transition from benign to malignant lesions can take years, with regular screening crucial in early detection (158–164).

### *Clinical Manifestations of HPV Infection*

HPV can lead to a wide range of clinical manifestations, from benign lesions to cancers, depending on the type and affected tissue (165–171).

- Benign Lesions: Low-risk HPV types, such as HPV 6 and 11, commonly cause warts on the skin and genital warts. While often self-limiting, they can recur and cause discomfort ( Fig.2)
- Malignant Lesions: High-risk HPV types, mainly HPV 16 and 18, are responsible for cancers in the cervix, anus, vulva, vagina, penis, and oropharynx. Persistent infection with these high-risk types can lead to precancerous changes, which may evolve into cancer over time.
- Recurrent Respiratory Papillomatosis (RRP), Caused by HPV 6 and 11, causes warts to form in the respiratory tract. This condition can potentially cause airway obstruction and require frequent surgeries (172–177).

### *HPV and Cancer Development*

High-risk HPV types, mainly HPV 16 and 18, are major contributors to cancer development, most notably cervical cancer. These types express oncoproteins (E6 and E7) that interfere with tumor suppressor proteins like p53 and Rb, leading to uncontrolled cell division and genetic instability. HPV-induced cancers typically take years to develop, and screening can detect precancerous lesions before they progress to invasive cancer. Early detection through regular Pap smears, HPV testing, and vaccination programs are essential in reducing the burden of HPV-related cancers (178–181).

## Materials and Methods

This work is a literature review based on a search for scientific evidence related to the research question.

### *Source of Study Materials*

The review utilized the PubMed/MEDLINE, Dentistry & Oral Sciences Source, and Dialnet databases to search for biomedical studies. Citations from included articles were also considered.

### *Search Strategy*

The search used relevant keywords from the PubMed/MEDLINE Medical Subject Headings (MeSH). Boolean operators were applied to create the final search chain. Key terms included “Papillomavirus,” “Oral cavity,” “Oral cancer,” “Oral papillomavirus treatment,” “Oral papillomavirus diagnostic,” “Oral papillomavirus benign lesion,” and others related to HPV and oral health.

### *Filters Applied*

- Date: Studies published in the last five years were prioritized. Additional articles were consulted for further comparison.
- Language: Only full-text articles in English or Spanish were considered.

### *Inclusion and Exclusion Criteria*

Studies were included based on their ability to answer the research question or relevance to the review’s objectives.
Inclusion criteria included:

- Design: Observational studies, clinical trials, meta-analyses, or literature reviews.
- Population: Individuals with oral lesions caused by papillomavirus.
- Date: Studies from the last ten years.
- Language: English.

Articles not meeting these criteria were excluded.

## Results

Human papillomavirus (HPV) and oral lesions, which can range from benign proliferative growths to cancers, are related, according to the research. Verruca vulgaris, condyloma acuminatum, and papillomas are benign lesions consistently associated with low-risk HPV strains, especially HPV 6 and 11. Although these lesions are usually not dangerous, they could need to be treated if they repeat or cause discomfort. In contrast, high-risk HPV strains such as HPV 16 and 18 are not found in benign lesions and are significantly linked to oropharyngeal cancer, especially in younger, nonsmoking people.

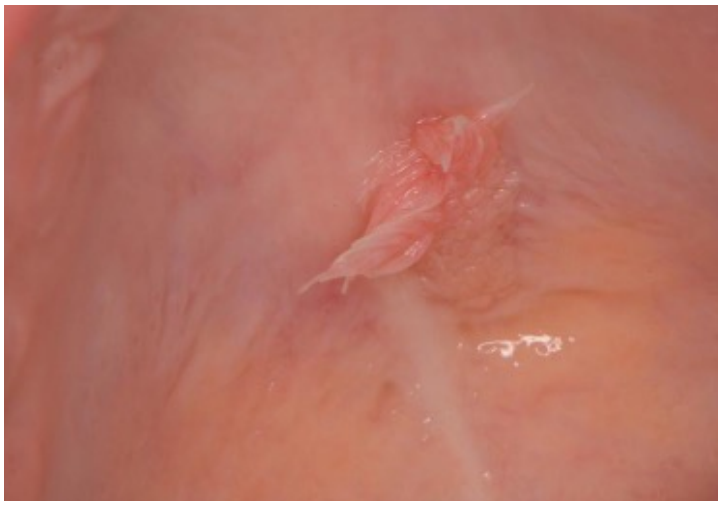

Figure 2. Benign papilloma virus lesion

Nevertheless, no meaningful association between high-risk HPV and laryngeal or oral malignancies has been discovered.

With seroconversion rates close to 100%, preventive HPV vaccination—including bivalent, quadrivalent, and nonavalent vaccines—has shown great effectiveness in lowering oral HPV infections. Adolescent HPV-related malignancies can be significantly decreased by vaccinating both sexes.

Benign HPV lesions can be managed with minimally invasive techniques, including laser therapy, photodynamic therapy, and surgical removal. Treatments, including chemoradiotherapy and transoral robotic surgery, provide better results and a better prognosis for HPV-positive malignancies than for HPV-negative ones. The results highlight the significance of early detection, prevention, and customized treatments to treat the range of oral disorders linked to HPV.

## Discussion

Human papillomavirus (HPV) infection, particularly high-risk strains, is a primary etiological agent for oral squamous cell carcinoma and oropharyngeal carcinoma (182–186). Oral HPV infections can be subclinical or associated with benign proliferative lesions (e.g., verruca vulgaris, condyloma acuminatum, papillomas, and multifocal epithelial hyperplasia), predominantly caused by low-risk HPV strains. Many studies have explored the relationship between HPV and benign lesions (187–193). Most studies show a positive association between low-risk HPV types and oral lesions, with HPV types 6 and 11 being the most commonly detected. High-risk HPV types (e.g., HPV 16 and 18) were not found in benign lesions (194–201).

High-risk HPV is frequently linked to head and neck carcinomas, especially oropharyngeal cancer, which is significantly associated with HPV type 16 (202–209). However, no association has been found between the oral cavity and laryngeal cancers (210–220). Studies also indicate that β1-HPV-5 is linked to oropharyngeal, oral cavity, and laryngeal carcinomas, while γ11 and γ12-HPV strains are associated with oral and laryngeal cancers (221–227). Smoking and alcohol consumption are additional risk factors, which highlighted HPV-16's association with oropharyngeal cancer in both smokers and non-smokers (228–233). This suggests that HPV may act synergistically with other carcinogens in the development of these cancers (234–242).

HPV vaccines are the most effective prevention against HPV infections and subsequent disease development (243–252). The available vaccines include quadrivalent, nonavalent, and bivalent vaccines targeting various HPV types (253–259). Studies show that vaccination, especially in adolescents around 12 years old, prevents oral HPV infections. Vaccinating both males and females is more effective in reducing HPV-related diseases than vaccinating females alone. Vaccines induce potent immune responses, with seroconversion rates approaching 100% in both men and women. Vaccination has been shown to dramatically reduce the incidence of HPV-associated cancers, emphasizing its crucial role in public health (260–262).

Surgical excision remains the preferred treatment for benign HPV-related lesions, with options including electrocautery, laser surgery, cryosurgery, and intralesional interferon injections (263–267). Laser surgery offers several benefits, including minimal postoperative pain, high precision in tissue destruction, and rapid healing. Other treatments, such as trichloroacetic acid and topical imiquimod, have also shown positive results in resolving these lesions. Photodynamic therapy (PDT) with aminolevulinic acid (ALA-PDT) has proven effective for condylomas and papillomas, with lesions gradually disappearing after a few sessions. These approaches offer effective, less invasive alternatives to traditional surgical methods (268–271).

HPV-related head and neck cancers tend to have a better prognosis than non-HPV-related cancers. Treatment often involves surgery and radiotherapy, with minimally invasive techniques like transoral laser microsurgery (TLM) and transoral robotic surgery (TORS) providing excellent outcomes for early-stage oropharyngeal cancers. Chemoradiotherapy with high-dose cisplatin is commonly used for advanced laryngeal cancers (272–275). Targeted therapies, such as immunotherapy for HPV-driven tumors, are also being explored, offering hope for more personalized and effective treatment options (276–279).

Given the prevalence of HPV and its association with both benign and malignant lesions, prevention, early detection, and effective management are crucial (280–284). Comprehensive intraoral and extraoral examinations are essential for early diagnosis, and further research is needed to understand HPV's clinical implications better and improve therapeutic strategies (285,286). Early intervention is critical for both reducing the burden of HPV-related cancers and improving patient outcomes. Additionally, as the virus continues to evolve and new strains emerge, ongoing surveillance and adaptation of treatment strategies will be essential in managing the full scope of HPV-related diseases (287–290).

## Conclusions

Human papillomavirus (HPV) is a common sexually transmitted infection that can cause both benign oral lesions and cancers, particularly in the oropharynx. While many infections resolve naturally, persistent infection with high-risk strains like HPV 16 and 18 can lead to malignancies, highlighting the need for prevention and early detection.

- HPV and Oral Lesions: HPV can cause both benign lesions and malignancies, including oral and oropharyngeal cancers.
- Risk Factors: Key risk factors include oral sex, multiple sexual partners, smoking, and alcohol consumption.
- Benign vs Malignant Lesions: Low-risk HPV types (e.g., HPV 6 and 11) cause benign lesions, while high-risk types (e.g., HPV 16 and 18) are linked to cancer.
- Prevention and Treatment: Vaccination reduces HPV-related diseases. Treatments for benign lesions include cryotherapy and laser therapy, while HPV-positive cancers have a better prognosis than HPV-negative cancers.

## Abbreviations

ALA-PDT - Aminolevulinic acid photodynamic therapy
E1 - Early gene 1
E2 - Early gene 2

E6 - Early gene 6
E7 - Early gene 7
HPV - Human Papillomavirus
L1 - Major capsid protein L1
L2 - Minor capsid protein L2
MeSH - Medical Subject Headings
ORFs - Open reading frames
RRP - Recurrent respiratory papillomatosis
STIs - Sexually transmitted infections
TLM - Transoral laser microsurgery
TORS - Transoral robotic surgery

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Not applicable.

## Conflicts of Interest

The authors declare no conflict of interest.

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