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Original Article

# Human papillomavirus-related oral lesions in people with HIV: A 20-year multinational cohort analysis

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**KEYWORDS**
Oropharyngeal cancer;
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Heck disease

**Abstract** *Background/purpose:* People with human immunodeficiency virus (PWH) are at increased risk of acquiring human papillomavirus (HPV) infection and subsequently developing HPV-related lesions. We aimed to determine the prevalence and strength of association between HPV-related oral lesions and HIV infection, using a large, real-world dataset.
*Materials and methods:* This retrospective cohort used 20 years of de-identified electronic health records from TriNetX (109 organizations, 24 countries). Adults ≥18 years with HIV (ICD-10 B20, LOINC antibodies 7917-8/7918-6, RNA 25835-0) were compared age and sex matched people without HIV. We quantified prevalence and associations for HPV-related benign lesions (papilloma, verruca, epithelial hyperplasia, condyloma) and malignant oropharyngeal cancer (tonsil/base of tongue; ICD-O). Analyses excluded pre-study lesions, assessed oropharyngeal squamous cell carcinoma (OPSCC) after HIV diagnosis, and examined smoking effects within PWH.
*Results:* In 253,847 PWH, condyloma (8.73 %) was most common, followed by verruca vulgaris (4.39 %), oral papilloma (2.63 %), epithelial hyperplasia (0.10 %), and HPV-OPSCC (0.26 %). All lesions were significantly more frequent than in people without HIV controls. OPSCC prevalence was 0.19 % in non-smokers, higher with smoking (0.26 %; odds ratio = 1.77). Elevated odds for OPSCC persisted in non-smokers, underscoring increased HPV-OPSCC disease risk in

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PWH regardless of tobacco exposure.
*Conclusion:* PWH face increased susceptibility to HPV-related oral lesions, particularly benign variants and OPSCC, with smoking further amplifying this risk. Despite the relatively low prevalence of OPSCC, there remains a critical need for targeted HIV care—including routine oral examinations, HPV vaccination, smoking cessation support, and advanced diagnostic approaches—to enable precise, evidence-based prevention strategies.


## Introduction

Human papillomavirus (HPV) is a double-stranded DNA virus encompassing more than 200 genotypes, each with a marked tropism for squamous epithelial cells. HPV can induce lesions on any cutaneous or mucosal surface, including extremities, genital tract, and oral cavity. Transmission occurs via direct skin-to-skin contact, most commonly through sexual activity involving the genitalia, oral cavity, or anus. HPV types are classified as low-risk (e.g., 2, 3, 6, 7, 10, 11, 27, 56), typically associated with benign lesions with little to no risk of malignancy, or high-risk (e.g., 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 68), which are linked to persistent infection and malignant transformation with HPV 16 being the most implicated subtype.[1–3] Benign lesions include condyloma acuminatum (venereal wart) [HPV 6, 11], verruca vulgaris (common wart) [HPV 2, 27, 56], papilloma [HPV 6, 11], epithelial hyperplasia (Heck disease) [HPV 13, 32], recurrent respiratory papillomatosis [HPV 11, 6], Butcher's wart [HPV 7], and flat wart [HPV 3, 10].[3] Malignancies associated with high-risk HPV include cancers of the cervix, anus, vagina, oropharynx, penis, and vulva. According to the National Cancer Institute, high-risk HPV subtypes are responsible for 100 % of all cervical cancers, 90 % of anal cancers, 70 % of oropharyngeal cancers, 75 % of vaginal cancers, 69 % of vulvar cancers, and 63 % of penile cancers. High-risk HPV genotypes express the viral oncoproteins E6 and E7, which inactivate the tumor-suppressor proteins p53 and Rb, respectively, thereby promoting oncogenesis.[4–6]

People with human immunodeficiency virus (PWH) are at increased risk of acquiring HPV infection and subsequently developing HPV-associated lesions—both benign (e.g., condyloma, papilloma, verruca vulgaris, epithelial hyperplasia) and malignant (e.g., cervical, anal, and oropharyngeal cancers).[7–14] This heightened susceptibility is primarily attributable to HIV-induced immunosuppression. In immunocompetent hosts, HPV infection is often cleared by the immune system;[15] however, in PWH, impaired immunity promotes viral persistence.[16,17] The combination of chronic inflammation and HIV-associated immune dysregulation further augments oncogenic potential.[18] Previous studies have shown that oral HPV is detected more frequently in saliva or oral rinse specimens from PWH than from HIV seronegative individuals.[19,20] The present study aimed to determine the prevalence and strength of association between HPV-related oral lesions and HIV infection, using a large, real-world dataset.

## Materials and methods

Data was obtained from the TriNetX Research Network (TRN), a global, federated health research platform containing de-identified electronic health records from 109 healthcare organizations across 24 countries. All data were de-identified and compliant with HIPAA regulations prior to analysis were deemed exempt from institutional review board oversight by A.T. Still University, as it qualified as non-human subject research. This retrospective cohort study included adults (≥18 years) with evidence of HIV infection, defined as: ICD-10-CM diagnosis code B20, HIV antibody detected in serum (LOINC: 7917-8, 7918-6), and HIV RNA detected in serum/plasma by nucleic acid amplification with probe detection (LOINC: 25835-0). Eligible records were from the preceding 20 years through the query date (August 8, 2025). Patients were categorized to assess the prevalence and magnitude of association between HIV and HPV-associated benign and malignant lesions and examine the relationship between HIV and HPV-associated oropharyngeal squamous cell carcinoma (OPSCC) by smoking status.

Benign HPV-associated lesions were identified using ICD codes for: oral papilloma (**D10.30, K13.7**), verruca vulgaris (**B07, B07.8, B07.0**), focal epithelial hyperplasia (**K13.29**), condyloma (**A63.0, B97.7, A51.31**). Malignant HPV-associated lesions - OPSCC were identified by ICD-O codes for cancers of the tonsils and base of tongue (**C10.9, C10.0, C09.9, C01**). To estimate risk, we performed 1:1 propensity score matching by age and sex between PWH and people without HIV (PWoH) control cohort (no documented HIV diagnosis and no serologic or molecular evidence of HIV). Patients with benign or malignant lesions diagnosed before the study period were excluded. A *P*-value of $<0.05$ is considered significant. Table 1summarizes the demographics characteristics of cohort before and after propensity matching and after exclusion of patients with HPV-related oral lesions before the study period.

To evaluate whether HIV is an independent risk factor for HPV-OPSCC in PWH, we compared OPSCC incidence in HIV-positive non-smokers with that in a control cohort of people without HIV, non-smoking individuals with no serologic or molecular evidence of HIV infection. A 1:1 propensity score match was performed by age and sex, and individuals with a history of OPSCC prior to the study period were excluded. Given the established role of smoking in head and neck cancer, we compared the risk of OPSCC in PWH who smoked versus those who did not. Baseline

**Table 1** Demographics characteristics of cohort before and after propensity matching, and exclusion of patients with HPV related lesions diagnosed before the study period.

| Variable | | Cohort 1 (PWH) | Cohort 2 (PWoH) | *P*-value |
|---|---|---|---|---|
| **Before propensity matching** | | | | |
| Age at index (Mean ± SD) | | 44.1 ± 13.7 | 47.5 ± 25.3 | |
| Gender n (%) | Female | 60,552 (26.14 %) | 4,289,132 (53.37 %) | *<0.0001* |
| | Male | 170,946 (73.79 %) | 3,743,116 (46.58 %) | *<0.0001* |
| **After propensity matching** | | | | |
| Age at index (Mean ± SD) | | 44.1 ± 13.7 | 44.1 ± 13.7 | *1.000* |
| Gender n (%) | Female | 60,552 (26.14 %) | 60,552 (26.14 %) | *1.000* |
| | Male | 170,946 (73.79 %) | 170,946 (73.79 %) | *1.000* |
| **After propensity matching and exclusion of patients with HPV related lesions diagnosed before the study period** | | | | |
| Condyloma n (%) | After matching & exclusion | 227,846 | 230,093 | *<0.0001* |
| | Outcome | 15,929 (6.99 %) | 1,195 (0.52 %) | |
| Verruca vulgaris n (%) | After matching & exclusion | 229,790 | 228,300 | *<0.0001* |
| | Outcome | 7,078 (3.08 %) | 2,638 (1.16 %) | |
| Oral papilloma n (%) | After matching & exclusion | 230,693 | 230,152 | *<0.0001* |
| | Outcome | 5,021 (2.18 %) | 2,177 (0.95 %) | |
| Epithelial hyperplasia n (%) | After matching & exclusion | 231,618 | 231,610 | *<0.0001* |
| | Outcome | 175 (0.08 %) | 91 (0.04 %) | |
| Oropharyngeal cancer n (%) | After matching & exclusion | 231,475 | 231,312 | *0.0016* |
| | Outcome | 412 (0.18 %) | 326 (0.14 %) | |

PWH — people with HIV, PWoH — people without HIV, SD — standard deviation, HPV — human papillomavirus.

demographics (age, sex) were collected, and 1:1 propensity score matching was repeated by age and sex. The primary outcome was an OPSCC diagnosis occurring after the HIV diagnosis. Patients with OPSCC prior to the study period were excluded.

## Results

### Prevalence of human papillomavirus-related oral lesions in people with human immunodeficiency virus

Table 2 summarizes the prevalence and magnitude of association of HPV-related oral lesions in PWH. Among 253,847 individuals diagnosed with HIV, condyloma was the most prevalent HPV-associated oral lesion, affecting 8.73 % (n = 22,168; 76.67 % male, 23.28 % female). This was followed by verruca vulgaris: 4.39 % (n = 11,155; 83.42 % male, 16.49 % female), oral papilloma: 2.63 % (n = 6,676; 73.08 % male, 26.77 % female), epithelial hyperplasia: 0.10 % (n = 251; 74.9 % male, 25.1 % female), and HPV-OPSCC: 0.26 % (n = 648; 85.03 % male, 14.97 % female). In the subgroup of 159,574 PWH non-smokers, the prevalence of HPV-OPSCC was 0.19 %.

### Magnitude of association between human immunodeficiency virus and human papillomavirus-related oral lesions

HPV-related oral lesions were significantly more common in PWH compared to PWoH controls (Fig. 1). PWH had markedly higher odds of condyloma: odds ratio (OR) = 14.39, $P < 0.0001$, verruca vulgaris: OR = 2.72, $P < 0.0001$, oral papilloma: OR = 2.33, $P < 0.0001$, epithelial hyperplasia: OR = 1.92, $P < 0.0001$, and HPV-OPSCC: OR = 1.26, $P = 0.0016$. Among non-smokers, PWH still had an elevated prevalence of HPV-OPSCC compared with PWoH (OR = 1.38, $P = 0.005$).

### Risk of human papillomavirus-oropharyngeal squamous cell carcinoma in people with human immunodeficiency virus by tobacco use

Table 3 summarizes the measures of association between HIV and HPV-OPSCC based on smoking status. Within the PWH cohort, the prevalence of HPV-OPSCC was 0.15 % among non-smokers and 0.26 % among those with a history of smoking. Smoking status significantly increased risk, with smokers exhibiting higher odds of HPV-OPSCC compared to non-smokers (OR = 1.77, $P < 0.0001$).

## Discussion

In this large, real-world cohort, PWH had a markedly higher prevalence of HPV-related oral lesions than people without HIV, with the greatest relative increase observed for condyloma. Odds were also significantly elevated for benign lesions—verruca vulgaris, oral papilloma, and epithelial hyperplasia. For OPSCC, PWH showed modestly higher odds compared with HIV seronegative individuals, and tobacco use further amplified risk within PWH. Despite these relative increases, the absolute prevalence of OPSCC remained low across all subgroups.

Our findings align with prior literature documenting excess HPV-related disease in HIV. Minkoff et al.[11] reported a 9.3-fold higher risk of condyloma among HIV-infected

**Table 2** Prevalence and magnitude of association of HPV-related oral lesions in PWH.

| HPV-related oral lesions | PWH | Patients with HPV-related oral lesions | % of males | % of females | Most prevalent age-group decade with HPV-related oral lesions | Prevalence of HPV-related oral lesions | Magnitude of association OR (95 % CI), *P*-value |
|---|---|---|---|---|---|---|---|
| Benign | | | | | | | |
| Condyloma | 253,847 | 22,168 | 76.67 % | 23.28 % | 4th (25.57 %) | 8.73 % | 14.39 (13.57—15.278), *< 0.0001* |
| Verruca vulgaris | 253,847 | 11,155 | 83.42 % | 16.49 % | 5th (30.75 %) | 4.39 % | 2.72 (2.59—2.84), *< 0.0001* |
| Papilloma | 253,847 | 6,676 | 73.08 % | 26.77 % | 5th (26.51 %) | 2.63 % | 2.33 (2.22—2.45), *< 0.0001* |
| Epithelial hyperplasia | 253,847 | 251 | 74.9 % | 25.1 % | 5th (35.86 %) | 0.1 % | 1.92 (1.49—2.48), *< 0.0001* |
| Malignant | | | | | | | |
| HPV-OPSCC | 253,847 | 648 | 85.03 % | 14.97 % | 5th (47.07 %) | 0.26 % | 1.26 (1.09—1.46), *0.0016* |
| HPV-OPSCC (PWH non-smokers) | 159,574 | 309 | 89 % | 11 % | 5th (44.66 %) | 0.19 % | 1.38 (1.1—1.72), *0.005* |

HPV — human papillomavirus, OPSCC — oropharyngeal squamous cell carcinoma, PWH — people with HIV, OR — odds ratio, CI — confidence interval.

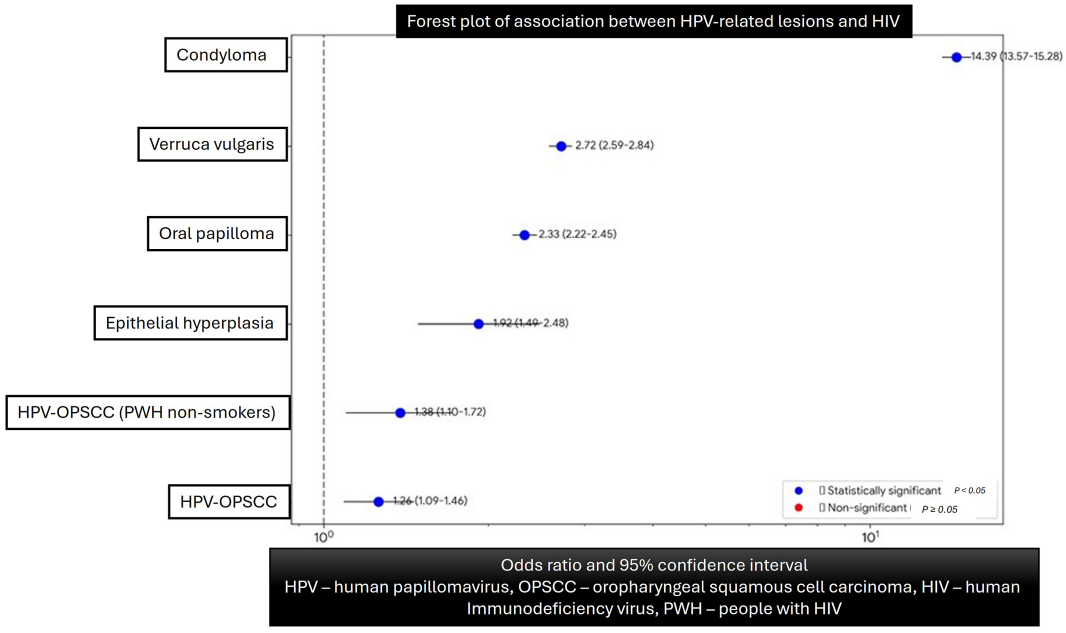


**Figure 1** Forest plot showing the association between HPV-related oral lesions and HIV.

**Table 3** Measures of association between HIV and HPV-OPSCC based on smoking status.

| | Patients in cohort | Patients with outcome (HPV-OPSCC) | Risk of outcome (%) | Odds ratio (95 % CI) | *P*-value |
|---|---|---|---|---|---|
| HIV (smokers) | 75,191 | 194 | 0.26 % | 1.77 (1.39—2.23) | *< 0.0001* |
| HIV (non-smokers) | 75,237 | 110 | 0.15 % | | |

HIV — human immunodeficiency virus, HPV — human papillomavirus, OPSCC — oropharyngeal squamous cell carcinoma.

women; Greenblatt et al.[21] observed a 6.78-fold increase in those with CD4+ counts <200 cells/mm$^3$; and Low et al.[13] reported a six-fold increase with CD4+ >200 cells/mm$^3$ and a 20-fold increase with CD4+ <200 cells/mm$^3$. In our analysis, PWH were 14.39 times more likely to develop condyloma than people without HIV. Dolev et al.[12] found a higher cumulative incidence of anogenital verrucae among HIV-infected women; correspondingly, we observed 2.72-fold higher odds of verruca vulgaris in PWH. Although Anaya-Saavedra et al.[22] and Camacho-Aguilar et al.[23] reported epithelial hyperplasia prevalence of 27.3 % and 62 % in PWH, respectively, we did not observe such high prevalence; however, the odds of epithelial hyperplasia were still increased by 92 % relative to people without HIV controls. Consistent with Chaturvedi et al.,[7] Beachler et al.,[24] and Mazul et al.,[25] we found that HPV-OPSCC is more frequent in PWH; notably, Beachler et al.[24] reported standardized incidence ratios approximately three times higher than in the general population.

HIV-associated CD4+ T-cell depletion and qualitative immune dysfunction likely impair the cell-mediated responses required for HPV clearance, promoting persistent infection. Chronic inflammation and immune activation may further create a pro-oncogenic microenvironment.[18] The HIV *tat* protein can activate NF-κB signaling and upregulate pro-inflammatory cytokines (IL-12, IL-6, IL-8, TNF-α); elevated levels of these cytokines have been detected in the saliva of PWH with malignant or premalignant oral lesions.[26,27] NF-κB activity has been linked to increased transcription of HPV E6 and E7 oncoproteins, facilitating p53 and pRB inactivation and supporting neoplastic progression.[26,28,29] HIV *tat* may also enhance HPV replication and the proliferation of infected keratinocytes.[18] Tobacco exposure plausibly potentiates these processes by causing mucosal injury, disrupting local immunity, and increasing genomic instability.

Strengths of this study include the use of a large, multi-institution dataset, which enhances statistical power and generalizability; application of propensity score matching by age and sex, allowing balanced comparisons between PWH and PWoH controls as well as between smokers and non-smokers within PWH; and results that reflect routine clinical practice across diverse healthcare settings. The limitations include reliance on ICD diagnostic codes that may lack site specificity—for example, condyloma and verruca vulgaris codes were not restricted to the oral cavity—and the use of anatomic codes for tonsil and base of tongue, which enrich for HPV-related tumors but do not confirm HPV status (e.g., p16, HPV DNA/RNA), potentially leading to misclassification. Propensity matching was limited to age and sex; other potentially important covariates such as sexual behaviors, alcohol use, HPV vaccination status, CD4 count, and viral load were not controlled for and may have introduced residual confounding. Prior studies have demonstrated that low CD4+ counts are significantly associated with HPV-related lesions, and that highly active antiretroviral therapy use does not mitigate the risk of developing such lesions.[7,12,13,21,24,30,31]

PWH experience a broad elevation in risk for HPV-related oral conditions, with the most pronounced increases in benign lesions and a modest but meaningful excess in OPSCC that is further amplified by tobacco use. While absolute OPSCC prevalence remains low, these data support pragmatic, risk-informed strategies in HIV care: integrate focused oral examinations, ensure HPV vaccination, and prioritize tobacco cessation. Improving diagnostic specificity and accounting for key confounders in future work will sharpen risk estimates and guide precise, implementable screening and prevention pathways.

## Declaration of competing interest

The authors declare no conflict of interest. This research received no external funding.

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