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# Prevalence of Human Papillomavirus in Oropharyngeal Carcinoma: A Tertiary Care Teaching Hospital-Based Cross-Sectional Study

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## ABSTRACT

**Background & objectives:** Oropharyngeal cancer is caused by multiple factors, including human papillomavirus (HPV). This cross-sectional study evaluated the prevalence and genotype distribution of HPV in histopathological confirmed oropharyngeal squamous cell carcinoma cases at Government Medical College Hospital, Kota, to establish baseline regional data for future screening and treatment protocols.

**Methods:** This was a hospital based descriptive cross-sectional study conducted at a tertiary care cancer center in Government Medical College Hospital over 2 years. One hundred patients with histopathologically confirmed oropharyngeal carcinoma meeting the inclusion criteria were included in the study. Oropharyngeal samples were collected from these patients by gently rubbing a swab on the target area and these were then sent to Multidisciplinary Research Unit (MRU) in viral transport media (VTM) for HPV testing and genotyping.

**Results:** Four out of 100 samples were HPV positive, yielding a prevalence of 4.0%. The mean age of participants was 54.48±12.60 years. HPV16 and other high-risk HPV genotypes 31,33,35,39,45,51,52,56,58,59,66,67,68 were detected. No cases with HPV 18 were identified.

**Conclusions:** HPV DNA prevalence was 4.0% in this study. HPV 16 and other high-risk genotypes were detected. Statistical assessment of clinicopathological correlates was not feasible due to few positive cases. Multicentric studies covering diverse geographical and socioeconomic groups are needed to determine the true burden and profile of HPV-associated oropharyngeal carcinoma in the Indian subcontinent

***Keywords:*** Human Papillomavirus, Oropharyngeal Neoplasms, Prevalence, India, Cross-Sectional Studies

## INTRODUCTION

Cancer is a complex disease influenced by multiple genetic and environmental factors. HPV is a significant contributor to various cancers. In India, HPV contributes

substantially to the burden of cervical and oropharyngeal cancers. Population-based cancer registry data show that the district of Papumpare reported the highest age-adjusted incidence rate (AAR) of cervical cancer at 27.7 per 100,000 women. For oropharyngeal cancer, the highest AAR among males was recorded in the East Khasi Hills district at 11.4 per 100,000.

HPV is a double-stranded DNA virus. It was first identified as oncogenic in cervical cancer in 1983 and later classified as a human carcinogen in 1996 [1]. It's the most common sexually transmitted infection in the US, capable of infecting the mouth and throat. Oropharyngeal cancers (in areas like the soft palate, tongue base, tonsils and pharyngeal wall) can take years to develop after HPV infection. In the US, HPV is linked to 60-70% of oropharyngeal cancers, but isn't associated with other head and neck cancers like those in the larynx or salivary glands [3].

There are over 200 HPV types, with about 40 transmitted through sexual contact to the genital areas, mouth, and throat. High-risk types include 16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 73, and 82. Notably, HPV 16 and 18 account for over 90% of HPV-related oropharyngeal cancers and increase risk up to 14-fold. Oral HPV infection is common, affecting 10% of men and 3.6% of women, and usually clears within 1-2 years. However, persistent infections can lead to oropharyngeal cancer, which typically occurs in younger individuals with a history of multiple sexual partners. Transmission occurs primarily through oral sex and possibly other routes.

HPV accounted for 31% of infection-related cancer cases in 2018, with 13% of all cancer cases linked to human infections [2]. Globally, around 3% of OPSCC are HPV-positive. In India, HPV prevalence in OPSCC varies widely (0-74%) [3].

Chronic infection with high-risk HPV types 16 and 18 is a key cause of cervical, anogenital, and head and neck malignancies. Prophylactic HPV vaccination prevents an estimated 80% of cervical cancers and other HPV-associated malignancies. The WHO global strategy targets cervical cancer elimination at an incidence rate below 4 per 100,000 women annually. India bears a disproportionate burden, contributing 7% of worldwide cancer cases and nearly one-fourth of all HPV-related cancers globally [5]. Within India, HPV 16 and 18 are responsible for the majority of cervical cancer cases. [6,7]. (The country is implementing opportunistic screening for cervical and oral cancers as a cancer control strategy [8]. Cancer registries will help monitor the impact of these interventions.

Understanding HPV-related cancer epidemiology is crucial for developing effective prevention strategies in India. This study examines the epidemiology of HPV-related oropharyngeal cancers using data from hospital-based cancer registries (HBCRs) under India's National Cancer Registry Programme [9,10].

## MATERIALS & METHODS

This study was conducted at a tertiary cancer care center in the Government Medical College Hospital over a 2-year period from May 2022 to May 2024. It was approved by the Institutional Ethics Committee of Government Medical College, Kota. Approval no.: F.1(A)/IEC/GMC, KOTA/2022/32, dated 09.05.2022. One hundred patients with OPSCC meeting the inclusion criteria were enrolled. Detailed history was taken, including personal history of betel quid chewing, tobacco, and alcohol consumption. Clinical examination, hematology, biochemistry, serology, thyroid hormone profile, imaging, and histopathological diagnosis were performed. ECOG performance status was recorded. Tumor staging was done according to the AJCC 8th edition, 2017, separately for HPV-positive and HPV-negative cases. After obtaining informed consent, an oropharyngeal sample was collected from each patient by gently rubbing a swab stick on the target area. The swab was transported in viral transport medium at 2-8°C to the MRU. At the MRU,

nucleic acid extraction was performed, followed by HPV testing and genotyping using real-time PCR with the QIAseq Screen HPV in vitro assay on a Rotor-Gene Q instrument. This assay qualitatively detects HPV DNA of high-risk HPV genotypes 16 and 18, and a pool of 13 non-16/18 HPV types: 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, 67, and 68.

The QIA screen HPV PCR Test separately detects HPV 16, HPV 18, and the pool of 13 other HR-HPV genotypes, with the human β-globin gene serving as a sample control to assess DNA quality and potential inhibitory substances. The assay was clinically validated according to international guidelines for HPV detection [11]. Treatment was given accordingly. All patients were followed up every monthly.

We acknowledge that oropharyngeal swab sampling may underestimate true HPV prevalence compared to tumor tissue testing. p16 immunohistochemistry was not performed due to resource constraints, which is a limitation of this study. Future studies should correlate RT-PCR findings with tumor tissue-based HPV DNA testing and p16 IHC for definitive HPV status attribution in OPSCC.

## Statistical Analysis

Data were analyzed using Microsoft Excel and SPSS v.26 for descriptive statistics

## RESULT

Out of 100 histopathologically confirmed cases of oropharyngeal squamous cell carcinoma screened, four samples were positive for HPV DNA, yielding an overall prevalence of 4%. No cases of HPV 18 were detected. The mean age of participants was 54.48 ± 12.60 years.

**Table 1. Genotype distribution in HPV-positive oropharyngeal carcinoma (n=4)**

| Genotype of HPV | No. of patients positive | Percentage |
|---|---|---|
| HPV 16 | 2 | 50.0% |
| High Risk | 2 | 50.0% |
| HPV 18 | 0 | 0% |

*Other high-risk types include HPV 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, 67, 68.

Table 1 depicts the genotype distribution among the four HPV-positive cases. HPV 16 was detected in two patients (50.0%), and other high-risk HPV genotypes were detected in the remaining two patients (50.0%). No HPV 18 was identified in this study.

**Table 2. Clinicopathological profile of HPV-positive patients (n=4)**

| Age | Sex | Site | Stage | Histopathology | Genotype of HPV |
|---|---|---|---|---|---|
| 45 | M | Tonsil | III | SCC | High Risk |
| 60 | F | Vallecula | III | SCC | High Risk |
| 36 | M | BOT | III | SCC | HPV 16 |
| 32 | F | Tonsil | III | SCC | HPV 16 |

Table 2 presents the clinicopathological profile of the four HPV-positive patients. The age ranged from 32 to 60 years, with a male-to-female ratio of 1:1. The most common primary subsite was the tonsil (50.0%, 2/4), followed by the base of tongue and vallecula (25.0% each, ¼). All four patients presented with Stage III disease at diagnosis and had squamous cell carcinoma on histopathology. High-risk HPV genotypes were detected in all cases, with HPV 16 identified in two patients (50.0%) and other high-risk genotypes in two patients (50.0%).

### Other Observations

**Table 3: Shows the Gender-Wise Distribution of HPV in oropharyngeal carcinomas (n= 100)**

| Gender | Number of Patients (n) | HPV Positive (n) | Incidence of HPV positivity (%) |
|---|---|---|---|
| **Male** | 91 | 2 | 2.19 |
| **Female** | 09 | 2 | 22.22 |
| **Total** | 100 | 4 | 4 |

Table 3 shows the gender-wise distribution of HPV in the study population. The male-to-female ratio among all screened patients was approximately 10:1, with males comprising 91.0% (n=91) and females 9.0% (n=9). HPV DNA was detected in 2 of 91 males (2.19%) and in 2 of 9 females (22.22%), yielding an overall prevalence of 4.0%.

**Table 4: Age-wise distribution of patients screened (n=100)**

| Age Group | Number of Patients | Percentage |
|---|---|---|
| **18-29** | **2** | **2** |
| **30-39** | **13** | **13** |
| **40-49** | **17** | **17** |
| **50-59** | **29** | **29** |
| **60-69** | **29** | **29** |
| **70-79** | **10** | **10** |
| **Total** | **100** | **100** |

Table 4 shows the age-wise distribution of screened patients. The mean age of the study population was 54.48 ± 12.60 years. The majority of cases were in the 50-59- and 60-69-year age groups, each comprising 29.0%.

The primary site of disease was in base of tongue and tonsil comprising of 84%.

**Table 5: Site-wise distribution of oropharyngeal carcinoma (n=100)**

| Site of Primary tumor | N |
|---|---|
| **Soft Palate** | 12 |
| **Tonsil** | 46 |
| **Base of Tongue** | 38 |
| **Vallecula** | 4 |

Table 5 shows the site-wise distribution of primary tumors in the study. The most common primary site was the tonsil (46.0%, n=46), followed by the base of tongue (38.0%, n=38). Collectively, base of tongue and tonsil accounted for 84.0% of all cases. Soft palate comprised 12.0% (n=12) and vallecula 4.0% (n=4) of cases.

## Histologically

**Table 6: Histopathological type of oropharyngeal carcinoma (n=100)**

| Type of carcinoma | Number of patients |
|---|---|
| Squamous Cell Carcinoma (SCC) | 96 |
| Adeno Carcinoma (ADC) | 4 |

Table 6 shows the histopathological distribution of tumors in the study population. Squamous cell carcinoma was the predominant histological type, accounting for 96.0% (n=96) of cases. Adenocarcinoma was identified in 4.0% (n=4) of cases. All four HPV-positive cases were detected in patients with squamous cell carcinoma.

Tumor grade I-II was observed in 87.0% of patients. In the overall cohort, 93.0% of patients presented with Stage III or IV disease. A history of betel quid chewing, tobacco use, or bidi smoking was reported in 93.0% of patients

## DISCUSSION

In India, HPV infection contributes to oropharyngeal cancer in 22% of cases [4]. Recent studies show varying HPV prevalence: 0% in south India, 44.4% in north India, and 26.7% in west India, with HPV 16 and 18 being predominant [1,12-16]. Variations are likely due to differences in detection methods, population, lifestyle, geographic location, and sample size. Tobacco use 59% and alcohol consumption 4.7% are major contributors to oropharyngeal cancer in India [17,18]. Existing policies to control these risk factors need reinforcement [19]. Stage I and II oropharyngeal cancers have better 5-year survival rates than Stage III and IV [20].

Concurrent chemoradiation is recommended for patients with T3-4 disease, ≥2 positive nodes, or a single node >3 cm, showed improved overall survival and loco regional control[21-28]. For T1-2 N1 disease (single lymph node ≤3 cm), treatment options vary:

- T1N1: Radiation therapy (RT) alone is recommended due to limited data on concurrent systemic therapy.
- T2N1: RT alone or RT with concurrent systemic therapy is considered appropriate [24-26,29-31]. Concurrent chemo radiation improves overall survival, but induction systemic therapy shows no survival benefit [27,32-34].

A study by Chera BS (2015) [35] showed that 60 Gy in 30 fractions with concurrent chemoradiation (weekly cisplatin 30 mg/M$^2$) achieved a pathological complete response and reduced toxicities compared to standard chemoradiation. HPV-related head and neck cancers have high survival rates, making radiotherapy dose de-escalation feasible. However, personalized combinations of radiation and drugs based on genetic and biological features are needed to improve outcomes. The goal for HPV-positive head and neck cancer is to maintain or improve survival rates while reducing toxicities and improving quality of life [36].

## CONCLUSION

The role of HPV is well established in OPSCC. Early detection ascertains an extremely good prognosis in HPV-positive oropharyngeal carcinomas. However, the role of HPV in OPSCC is widely debated since there is a lot of discrepancy in its presence around the globe. The discrepancy can be attributed to the varied methodologies employed in diagnosis. The role of HPV in OPSCC may be better ascertained through advanced molecular diagnostic techniques applied to large, multi-ethnic studies across multiple centers worldwide. The therapeutic radiation dose is relatively lower compared to HPV-negative tumors, thereby reducing acute and late radiation-related toxicities and thus prolonging survival. Implementation of effective prevention and control strategies like HPV vaccination and scaling up of screening could reduce the burden of HPV-related cancers. Our study was limited to a tertiary cancer care center located in a small geographic area in the southeastern part of Rajasthan State, India, with a limited sample size. Studies with wider geographic representation including other parts of India are required before generalizing findings of our study. In this tertiary care descriptive cross-sectional study, the observed HPV DNA prevalence was 4% of oropharyngeal carcinoma cases. HPV16 and other high-risk genotypes were identified. Due to the descriptive nature of this study and limited sample size, no statistical association between HPV and OPSCC can be inferred. Multicentric studies employing standard molecular techniques, tumor tissue-based HPV testing, and p16 immunohistochemistry are required to delineate the role of HPV in OPSCC in the Indian subcontinent.

### *Declaration by Authors*

**Ethical Approval:** This study was approved by the Institutional Ethics Committee of Government Medical College and associated group of Hospitals, Kota,

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**Conflict of Interest:** The authors declare no conflict of interest.

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