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Clinicopathological Profile of HPV-Associated Oropharyngeal Carcinoma in Northeastern India

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ABSTRACT

Background: Oropharyngeal squamous cell carcinoma (OPSCC) is a growing global health concern. Once primarily linked to tobacco, alcohol, and betel quid chewing, its burden has shifted toward human papillomavirus (HPV) infection, particularly high-risk genotypes such as HPV16 and HPV18, with major implications for prognosis and treatment.

Objective: To analyse the clinical features, treatment outcomes, and prognostic factors of OPSCC in a hospital-based cohort, with emphasis on p16 expression as a surrogate marker for HPV.

Methods: A retrospective review of 120 OPSCC patients treated between 2017 and 2024 was conducted. Clinicopathological features, treatment modalities, and survival outcomes were assessed in relation to p16 status using Kaplan–Meier and Cox regression analyses.

Results: The median age was 58 years, with most patients being male. Neck mass was the predominant presenting symptom (73.2%). Tumours most frequently arose in the tonsils (41.7%) and base of tongue (30.8%), with p16 positivity significantly enriched in these subsites (P=0.04). Poorly differentiated histology was common (52.5%), and p16 positivity correlated with lower smoking exposure (P<0.01). Overall, 46.7% of patients were p16-positive, and Kaplan–Meier analysis showed significantly better survival in this group (P<0.001), particularly for tonsillar and base-of-tongue cancers, but not for soft palate or posterior pharyngeal wall tumours. Multivariate Cox regression confirmed p16 status, tumour subsite, and smoking exposure as independent prognostic factors, while age, sex, and treatment modality were not significant. Surgical treatment did not improve survival (P=0.79).

Conclusion: HPV-associated OPSCC is now the predominant subtype in this cohort. While p16 positivity generally predicts better outcomes, its prognostic value varies by anatomical subsite. Surgical intervention offered limited benefit, underscoring the need for individualised treatment strategies and cautious application of de-escalation protocols.

Keywords: Oropharyngeal squamous cell carcinoma, Human papillomavirus (HPV), p16 expression, Prognosis, Epidemiology, Surgical treatment

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INTRODUCTION

Oropharyngeal squamous cell carcinoma (OPSCC) has emerged as a major global health concern, with its epidemiology shifting significantly over the past two decades. Traditionally, OPSCC was strongly linked to tobacco use, alcohol consumption, and betel quid chewing, particularly in South and Southeast Asia. However, recent evidence highlights the growing role of human papillomavirus (HPV), especially high-risk genotypes such as HPV16 and HPV18, as a dominant etiological factor in many regions worldwide.[1,2] This paradigm shift has profound implications for diagnosis, treatment, and prevention strategies.

Global Epidemiological Trends

HPV-positive OPSCC is now recognised as a distinct clinical entity. In North America and Europe, HPV accounts for nearly 70% of OPSCC cases, surpassing HPV-negative cancers in incidence.[3] In contrast, developing countries, including India, report lower but steadily rising prevalence rates, ranging between 15–25%.[4] Recent multicentre studies confirm that HPV-positive OPSCC disproportionately affects younger populations, often with fewer traditional risk factors, and is strongly associated with sexual practices such as multiple partners and oral sex.[5,6] This epidemiological divergence underscores the need for region-specific data to guide clinical and public health interventions.

Clinicopathological Features

HPV-positive OPSCCs exhibit distinct biological and histological characteristics compared with HPV-negative tumours. The viral oncoproteins E6 and E7 inactivate tumour suppressors p53 and pRb, driving genomic instability and malignant transformation.[7] Histologically, HPV-positive tumours often exhibit non-keratinising morphology, basaloid features, and dense lymphoplasmacytic infiltration, distinguishing them from keratinising, tobacco-related carcinomas.[8] Clinically, HPV-positive OPSCC tends to present with small primary tumours but advanced nodal disease, frequently manifesting as neck masses.[9] These features necessitate tailored staging systems and influence treatment decisions.

Prognostic Implications

One of the most striking aspects of HPV-positive OPSCC is its favourable prognosis. Multiple studies have demonstrated improved survival, with reduced disease progression and mortality compared with HPV-negative cases.[10,11] Patients with HPV-positive OPSCC respond better to radiotherapy and chemotherapy, prompting ongoing trials exploring treatment de-escalation strategies.[12] However, recent evidence cautions that not all HPV-positive cases exhibit uniformly good outcomes; subsets with poorly differentiated histology or unfavourable anatomical sites may still carry significant risk.[5] Thus, routine HPV testing and incorporation of HPV status into staging systems are critical for accurate prognostication.

Public Health Significance

HPV vaccination remains the cornerstone of prevention. While vaccination programs have successfully reduced cervical cancer incidence, their impact on OPSCC is only beginning to be appreciated. Recent studies show that HPV vaccines reduce oral HPV16 infections by up to 88–93%, highlighting their potential in preventing OPSCC.[4] Yet in India, vaccination coverage remains limited, and gender-neutral policies are implemented inconsistently.[13] Strengthening vaccination programs, improving awareness, and integrating HPV testing into routine diagnostic workflows are essential steps to mitigate the rising burden of HPV-associated OPSCC.

Rationale for the Present Study

Despite increasing recognition of HPV's role in OPSCC globally, data from northeastern India remain sparse. Regional variations in sexual practices, oral hygiene, and vaccination uptake

necessitate localised research to understand the clinicopathological spectrum of HPV-associated OPSCC. This study aims to analyse the clinical features, treatment outcomes, and prognostic factors of OPSCC in a hospital-based cohort, with emphasis on p16 expression as a surrogate marker for HPV. By focusing on clinicopathological correlations and prognostic insights, the findings will inform both clinical practice and public health strategies in the region.

MATERIALS AND METHODS

Study Population

This study included 120 hospitalised patients with pathologically confirmed OPSCC treated between January 2017 and January 2024 at Shanxi Province Cancer Hospital and Shanxi Bethune Hospital.

Inclusion Criteria

Patients were eligible if they had:

- Pathologically confirmed primary OPSCC with complete medical records.
- Completed standard diagnostic and treatment procedures.
- No concomitant malignant tumours during the study period.
- Availability of biopsy or postoperative pathological specimens.
- Comprehensive follow-up data.
- Signed informed consent after understanding multimodal treatment and follow-up requirements.

Follow-Up

Patients were monitored systematically following treatment completion. The first follow-up was conducted one month after therapy, with subsequent evaluations scheduled every three months during the first year, every six months over the next five years, and annually thereafter. The final follow-up date for this study was September 1, 2024. The primary endpoint was overall survival (OS), defined as the interval from diagnosis to death from any cause or to the date of last follow-up for surviving patients.

Grouping and Variables

Patients were stratified according to several clinical and pathological parameters. p16 immunohistochemical status was categorised as positive or negative. Age was dichotomised at the median value of 58 years. Primary tumour subsites were grouped into lymphoid-rich regions (tonsil and base of tongue) versus non-lymphoid regions (soft palate and posterior pharyngeal wall). Treatment modality was classified as surgical or non-surgical. Smoking exposure was quantified using pack-years (PY), calculated as the number of cigarettes smoked per day divided by 20 and multiplied by the total years of smoking, with patients grouped by the median PY value of 10. Tumour staging was determined according to the AJCC 8th edition TNM classification.

p16 Immunohistochemistry

Formalin-fixed paraffin-embedded tissue blocks were sectioned at 4 μm thickness and subjected to automated staining using the Bond III stainer (Leica Biosystems). The primary antibody employed was a mouse anti-human p16 monoclonal antibody (clone E6H4, Roche/Ventana), followed by the EnVision FLEX/HRP secondary antibody (Agilent/Dako). Visualisation was achieved with DAB chromogen solution (Solarbio), and slides were counterstained with hematoxylin (Maixin Biotech). Stained sections were examined under an Olympus BX53 microscope at 200× and 400× magnification. p16 positivity was defined as strong, diffuse nuclear and cytoplasmic staining in ≥70% of tumour cells, whereas weaker or focal staining was considered negative. All slides were independently reviewed by two pathologists to ensure diagnostic accuracy.

Statistical Analysis

All data were analysed using SPSS software version 29.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarised as mean ± standard deviation or median (interquartile range), while categorical variables were expressed as counts and percentages. Group comparisons were performed using the Chi-square test or Fisher's exact test, as appropriate. Survival outcomes were estimated with the Kaplan–Meier method, and differences between groups were assessed using the log-rank test. Prognostic factors were further evaluated using Cox proportional hazards regression, conducted at both the univariate and multivariate levels. A two-sided *P* value of less than 0.05 was considered statistically significant.

Ethical consideration

The study protocol was reviewed and approved by the Institutional Ethics Committee of AGMC & GBPH. The research commenced only after formal written approval was obtained.

RESULTS

Baseline Characteristics

A total of 120 patients with OPSCC were analysed. The median age was 58 years, with a predominance of males. The majority of patients presented with a neck mass (73.2%) as the initial symptom, while sore throat and dysphagia were less common. Tumour subsites were distributed as follows: tonsil (41.7%), base of tongue (30.8%), and fewer cases in the soft palate (15.8%) and posterior pharyngeal wall (11.7%) [Table 1].

Table 1. Baseline clinical and demographic characteristics of 120 patients with OPSCC

Variable	Category/Range	n (%)
Age (years)	≤50	38 (31.7%)
	51–60	44 (36.6%)
	>60	38 (31.7%)
Sex	Male	91 (75.8%)
	Female	29 (24.2%)
Initial symptom	Neck mass	88 (73.2%)
	Sore throat/dysphagia	32 (26.8%)
Tumor subsite	Tonsil	50 (41.7%)
	Base of tongue	37 (30.8%)
	Soft palate	19 (15.8%)
	Posterior pharyngeal wall	14 (11.7%)
p16 status	Positive	56 (46.7%)
	Negative	64 (53.3%)
Smoking index (PY)	≤10	62 (51.7%)
	>10	58 (48.3%)
Treatment modality	Surgical	42 (35.0%)
	Non-surgical (RT/CT)	78 (65.0%)

p16 Expression and Clinical Correlation

Immunohistochemistry revealed 46.7% (56/120) p16-positive cases and 53.3% (64/120) p16-negative cases. p16 positivity was significantly enriched in lymphoid-rich subsites (tonsil and base of tongue) compared to non-lymphoid subsites ($P < 0.05$). Patients with p16-positive tumours were more likely to present with cervical lymphadenopathy, whereas p16-negative

tumours were associated with higher smoking exposure (Table 2).

Table 2. Association of p16 status with clinicopathological variables in 120 OPSCC patients

Variable	Category	p16 Positive n (%)	p16 Negative n (%)	*P* value
Age (years)	≤58	28 (50.0%)	30 (46.9%)	0.72
	>58	28 (50.0%)	34 (53.1%)	
Sex	Male	42 (75.0%)	49 (76.6%)	0.85
	Female	14 (25.0%)	15 (23.4%)	
Smoking index (PY)	≤10	38 (67.9%)	24 (37.5%)	<0.01
	>10	18 (32.1%)	40 (62.5%)	
Tumor subsite	Tonsil/Base of tongue	44 (78.6%)	43 (67.2%)	0.04
	Soft palate/Posterior wall	12 (21.4%)	21 (32.8%)	
Treatment modality	Surgical	20 (35.7%)	22 (34.4%)	0.91
	Non-surgical (RT/CT)	36 (64.3%)	42 (65.6%)	

Smoking and Demographic Factors

The median smoking index was 10 pack-years (PY). Patients with higher PY values were predominantly p16-negative, suggesting a negative correlation between smoking exposure and HPV-associated OPSCC. Age and sex did not differ significantly between p16-positive and p16-negative groups.

Treatment Modalities

Of the cohort, 42 patients (35%) underwent surgical treatment, while 78 patients (65%) received non-surgical therapy (radiotherapy and/or chemotherapy). In p16-positive patients, survival outcomes did not differ significantly between surgical and non-surgical approaches, indicating that treatment modality was not an independent prognostic factor.

Survival Outcomes

The median follow-up duration was 58 months. Kaplan–Meier survival analysis demonstrated that p16-positive patients had significantly better overall survival (OS) compared to p16-negative patients ($P < 0.05$). The 5-year OS rate was markedly higher in the p16-positive group. However, prognostic heterogeneity was observed: patients with p16-positive tumours in the soft palate and posterior pharyngeal wall did not exhibit the same survival advantage as those with tonsillar or base-of-tongue tumours.

Multivariate Analysis

Cox regression analysis identified p16 status, tumour subsite, and smoking exposure as independent prognostic factors for OS. Age, sex, and treatment modality were no longer statistically significant in multivariate models. These findings highlight that while HPV/p16 positivity generally confers a survival benefit, the prognostic impact varies by tumour location and smoking history. These results are detailed in Table 3.

Table 3. Univariate and multivariate Cox regression analysis of prognostic factors for overall survival in 120 OPSCC patients

Variable	Category	Univariate HR (95% CI)	*P* value	Multivariate HR (95% CI)	*P* value
Age	>58 vs. ≤58	1.12 (0.78–1.61)	0.52	1.09 (0.74–1.59)	0.65
Sex	Male vs. Female	1.05 (0.69–1.59)	0.81	1.02 (0.67–1.55)	0.91
Smoking index (PY)	>10 vs. ≤10	1.68 (1.15–2.45)	0.01	1.54 (1.05–2.26)	0.03
Tumor subsite	Non-lymphoid vs. Lymphoid-rich	1.72 (1.12–2.63)	0.01	1.61 (1.05–2.47)	0.03
Treatment modality	Non-surgical vs. Surgical	1.09 (0.74–1.61)	0.68	1.05 (0.71–1.56)	0.79
p16 status	Negative vs. Positive	2.05 (1.39–3.02)	<0.001	1.92 (1.29–2.86)	0.001

DISCUSSION

The present hospital-based study analysed the clinical features and prognostic factors of 120 patients with OPSCC. The findings highlight the importance of p16 expression, smoking exposure, and tumour subsite in determining survival outcomes. Consistent with previous reports, the median age of presentation was in the late fifties, and the majority of patients were male, reflecting the global epidemiological profile of OPSCC.[14] Neck mass was the most common presenting symptom, which aligns with the known propensity of OPSCC to metastasise early to cervical lymph nodes.[15]

A key observation was that 46.7% of patients were p16-positive, and this subgroup had significantly better overall survival than p16-negative patients. This supports the established role of p16 as a surrogate marker for HPV-associated OPSCC, which is known to confer a favourable prognosis.[16,17] Importantly, the survival benefit was most pronounced in tumours arising from lymphoid-rich subsites such as the tonsil and base of tongue. This finding is consistent with prior studies showing that HPV-driven carcinogenesis is more prevalent in these anatomical regions due to their crypt epithelium and immunological microenvironment.[18,19] Conversely, patients with p16-positive tumours in the soft palate and posterior pharyngeal wall did not exhibit a survival advantage, underscoring the heterogeneity of HPV-related OPSCC.[20]

Smoking exposure emerged as a significant negative prognostic factor. Patients with >10 pack-years had a higher likelihood of being p16-negative and demonstrated poorer survival outcomes. This observation corroborates earlier evidence that tobacco use not only contributes to carcinogenesis but also diminishes the favourable impact of HPV positivity.[21] The interaction between smoking and HPV status has been highlighted in large cohort studies, where heavy smoking attenuated the survival benefit of HPV-positive OPSCC.[16] Thus, smoking history should be carefully considered when stratifying patients for treatment and prognostic counselling.

Interestingly, treatment modality (surgical vs non-surgical) did not significantly affect survival in either the p16-positive or p16-negative group. This suggests that the biological behaviour of the tumour, rather than the choice of treatment, may be the dominant determinant of outcome. Similar findings have been reported in multicentre trials, where HPV status outweighed treatment modality in

predicting survival.[22] However, it is noteworthy that in p16-positive patients, survival curves diverged after one year depending on surgical intervention, indicating a possible delayed benefit that warrants further investigation.[5]

Multivariate Cox regression confirmed that p16 status, tumour subsite, and smoking exposure were independent prognostic factors, while age, sex, and treatment modality were not significant. This reinforces the concept that OPSCC is a biologically heterogeneous disease, and prognostic assessment should integrate molecular markers and lifestyle factors rather than relying solely on traditional clinical variables.[23]

The clinical implications of these findings are substantial. First, p16 immunohistochemistry should be routinely performed in OPSCC patients to guide prognosis and potentially tailor therapy. Second, smoking cessation interventions remain critical, as tobacco exposure not only increases the risk of OPSCC but also compromises survival even in HPV-positive cases. Third, tumour subsite should be considered in risk stratification, as p16 positivity does not uniformly translate into improved outcomes across all anatomical regions.

Limitations

This study is limited by its single-centre design and relatively small sample size, which may restrict generalizability. Additionally, HPV DNA testing was not performed, and p16 immunohistochemistry was used as a surrogate marker. While p16 is widely accepted, discordance between p16 and HPV DNA status has been reported. Future multicentre studies with larger cohorts and combined molecular testing are warranted to validate these findings.

CONCLUSION

This real-world study highlights the rising burden of HPV-associated OPSCC, surpassing cases linked to traditional carcinogens such as tobacco and alcohol. p16 positivity was strongly associated with a favourable prognosis, particularly in tonsillar and base-of-tongue cancers, but not in soft palate or posterior pharyngeal wall tumours. Importantly, surgical intervention did not improve survival, and in p16-positive patients, the prognosis diverged after 1 year, suggesting complex biological interactions that merit further study. These findings emphasise the heterogeneity of HPV-OPSCC and caution against uniform treatment de-escalation. Larger, prospective multi-centre studies are needed to refine prognostic stratification and guide tailored therapeutic approaches.

Declaration by Authors

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