

N Engl J Med. 2007 May 10;356(19):1944-56.

Comment in: N Engl J Med. 2007 May 10;356(19):1993-5.

Case-control study of human papillomavirus and oropharyngeal cancer.

D'Souza G, Kreimer AR, Viscidi R, Pawlita M, Fakhry C, Koch WM, Westra WH, Gillison ML.

Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, USA.

BACKGROUND: Substantial molecular evidence suggests a role for human papillomavirus (HPV) in the pathogenesis of oropharyngeal squamous-cell carcinoma, but epidemiologic data have been inconsistent.

METHODS: We performed a hospital-based, case-control study of 100 patients with newly diagnosed oropharyngeal cancer and 200 control patients without cancer to evaluate associations between HPV infection and oropharyngeal cancer. Multivariate logistic-regression models were used for case-control comparisons.

RESULTS: A high lifetime number of vaginal-sex partners (26 or more) was associated with oropharyngeal cancer (odds ratio, 3.1; 95% confidence interval [CI], 1.5 to 6.5), as was a high lifetime number of oral-sex partners (6 or more) (odds ratio, 3.4; 95% CI, 1.3 to 8.8). The degree of association increased with the number of vaginal-sex and oral-sex partners (P values for trend, 0.002 and 0.009, respectively). Oropharyngeal cancer was significantly associated with oral HPV type 16 (HPV-16) infection (odds ratio, 14.6; 95% CI, 6.3 to 36.6), oral infection with any of 37 types of HPV (odds ratio, 12.3; 95% CI, 5.4 to 26.4), and seropositivity for the HPV-16 L1 capsid protein (odds ratio, 32.2; 95% CI, 14.6 to 71.3). HPV-16 DNA was detected in 72% (95% CI, 62 to 81) of 100 paraffin-embedded tumor specimens, and 64% of patients with cancer were seropositive for the HPV-16 oncoprotein E6, E7, or both. HPV-16 L1 seropositivity was highly associated with oropharyngeal cancer among subjects with a history of heavy tobacco and alcohol use (odds ratio, 19.4; 95% CI, 3.3 to 113.9) and among those without such a history (odds ratio, 33.6; 95% CI, 13.3 to 84.8). The association was similarly increased among subjects with oral HPV-16 infection, regardless of their tobacco and alcohol use. By contrast, tobacco and alcohol use increased the association with oropharyngeal cancer primarily among subjects without exposure to HPV-16.

CONCLUSIONS: Oral HPV infection is strongly associated with oropharyngeal cancer among subjects with or without the established risk factors of tobacco and alcohol use.

Copyright 2007 Massachusetts Medical Society.

PMID: 17494927 [PubMed - indexed for MEDLINE]