

Study on the prevalence and incidence of dysplasia and HPV infection of the oropharyngeal, cervical and anal mucosa (oropharyngeal) of people with HIV

Carmen Hidalgo-Tenorio(1), Inmaculada Calle-Gómez (2), Raquel Moya (3), Mohamed Omar (4). Javier Lopez-Hidalgo (5), Javier Rodriguez-Granges (6), Leopoldo Muñoz (7), Carmen García-Martinez (8). (1)Unit of Infectious Diseases, Hospital Universitario Virgen de las Nieves (HUVN), IBS-Granada, UGR, Spain. (2)Internal Medicine Department, Hospital de Baza, Granada, (3) Internal Medicine Department, Complejo Hospitalario de Jaén; (4) Unit of Infectious Diseases, Complejo Hospitalario de Jaén; (5) Pathology Department, HUVN; (6)Microbiology Department, HUVN; (7) Unit of Infectious Diseases, Hospital Universitario San Cecilio; (8)Internal Medicine Department, HUVN, Spain.

Background: PLHIV under ART with undetectable viral load have persistent chronic inflammation. This inflammatory status favors the emergence of comorbid non-AIDS defining diseases, including non-AIDS neoplasms, and cardiovascular disease. The main objectives of this study of PLHIV participating in a program for the screening, diagnosis, treatment, and prophylaxis of anal and cervical dysplastic lesions were to determine the prevalence and incidence of HPV-related dysplasia and the clearance and acquisition rates of high-risk HPV (HR-HPV) in the oropharyngeal and anal mucosae of PLHIV, and in the genital mucosa of women living with HIV (WLWH), and to evaluate factors related to HR-HPV infection in OP mucosa at 12 months.

Results: 276 PLHIV were included (table 1 and 2). The HPV infection, other STIs and oropharyngeal dysplasia at baseline visit at table 3. At 12 months, the incidence of OP mucosa dysplasia was zero, with HR-HPV clearance and acquisition rates of 5.5% and 4.4%, respectively. The incidence of anal HSIL was 1,811.6 cases x 100,000 people-year, with HR-HPV clearance and acquisition rates of 16.2% and 25.6%, respectively; 6.2% had concomitant infection in oropharyngeal and anal mucosa. The incidence of CIN2/CIN3 or cervical cancer was zero, while the incidence of CIN1 was 3,488.3 per 100,000 women/year, with HR-HPV clearance and acquisition rates of 11.3% and 7.5%, respectively; 1.8% had concomitant HPV infection in oropharyngeal and genital mucosa. Factors related to HR-HPV infection at 12 months of follow up in oropharyngeal mucosa undetectable viral load retained (HR=0.184; 95% CI, 0.035-0.959).

Table1. Epidemiological variables	N=276	Table 2. HIV related variables	N=276	Table 3. HPV infection, other STIs, and oropharyngeal dysplasia at baseline	N= 276
Age, mean (years), (± SD)	45.3 (10.8)	HIV infection acquisition, n (%)			
Male	218 (79)			HPV positive PCR in oropharyngeal region, n (%)	31 (11.2)
Spanish nationality, n (%)	243 (88)	MSM	276 (78.3)		. ,
Median NP12m (IQR)	1(1-1)	Heterosexual	43 (15.6)	High-risk HPV, n (%)	22 (8)
Median NPT (IQR)	5 (3 – 13)	IDU	13 (4.7)	Number of high-risk HPV genotypes, median (IQR)	0 (0 – 0)
Years since first sexual relations (IQR)	29 (31–44.5)	Other	4 (1.4)	Low-risk HPV, n (%)	14 (5.1)
Use of condoms for oral sex, n (%)	4 (1.5)	Years HIV infection diagnosis (IQR)	23 (17 – 29)	Number of low-risk HPV genotypes, median (IQR)	0 (0 – 0)
Use of condoms for vaginal sex, n (%)	96 (35.8)	AIDS (A3, B3, C), n (%)	67 (24.3)	Mixed HPV (high- and low-risk), n (%)	4 (1.4)
Use of condoms for anal sex, n (%)	12 (37.5)	CD4 at diagnosis, cells/uL, mean (\pm SD)	398.8 (278.4)	HPV 6, n (%)	3 (1.1)
Retired	30 (10.9)	Cd4 nadir, cells/uL, mean (± SD)	335.5 (231.5)	HPV 11, n (%)	1 (0.4)
Illiterate	13 (4.7)	CD8 at diagnosis, cells/uL, mean (\pm SD)	1023.8 (477.2)	HPV 16, n (%)	6 (2.2)
Primary	59 (21.4)	VL at diagnosis, \log_{10} , mean (± SD)	5.5 (6.1)	HPV 18, n (%)	2 (0.7)
Secondary	80 (29)	Current CD4, cells/uL, mean (± SD)	725.2 (300.2)	HPV 31, 32, 33, , 35, 42, 43, , 52, 53, 54 n (%)	1 (0.4)
University	124 (44.9)	Current CD8. cells/uL. mean (± SD)	867.5 (417.1)		A (1 A)
Smoker, n (%)	100 (36.4)			HPV 44, n (%)	4 (1.4)
Alcohol, n (%)	38 (13.8)	Current CD4/CD8, ratio, mean (± SD)	0.96(0.5)	HPV 59, n (%)	3 (1.1)
IDU, n (%)	2 (0.7)	Current VL, log ₁₀ , mean (± SD)	2.5 (3.7)	HPV 39, 40, 56, 61, 66, 68, 69, 70, n (%)	2 (0.7)
Polypharmacy, n (%)	23 (8.3)	Current HIV-RNA VL undetectable n (%)	239 (85.2)	HPV-related oropharyngeal dysplasia, n (%)	0 (0)
HPV vaccination, n (%)	93 (33.7)	Naïve, n (%)	O (O)	Positive PCR for other STIs, n (%)	15 (5.4)
Chronic HBV infection, n (%)	5 (1.8)	Years with ART, median (IQR)	20.2 (13.1 – 23.4)	- N. gonorrhoeae	13 (86.7)
Active chronic HCV infection, n (%)	15 (5.4)	ART lines, median (IQR)	5.5 (3 – 7)	- C. trachomatis	2 (13.3)
Syphilis at baseline, n (%)	9 (3.3)	Current ART, n (%)	276 (100)		
Other STIs at baseline, n (%)	20 (7.2)				
Condylomas at baseline, n (%)	8 (2.9)	Virological failure, n (%)	2 (0.7)		
History of genital condulomas n (%)	82 (29 7)				

Conclusions: Among PLHIV, HSIL incidence and HR-HPV acquisition rate are higher in anal *versus* oropharyngeal and genital mucosae. Non-detectability protects against oropharyngeal HPV infection.