No evidence of differences in anal HPV 16/18 load between HIV-positive and HIV-negative MSM

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Background

Persistent HPV infections can cause genital warts, cervical-, vaginal-, vulvar-, anal-, penile-, and head-and-neck cancer. HPV-type 16 and HPV-type 18 are the most carcinogenic. Anal HPV-related disease burden is higher among HIV-positive compared to HIV-negative men who have sex with men (MSM) (anal cancer incidence 77.8 vs. 5.1 per 100,000 per year, respectively). A persistent HPV infection is a prerequisite for the development of HPV-related disease. A potential factor influencing effectiveness and persistence of HPV infection, is HPV viral load, a marker of productiveness of an HPV infection.

Objectives

In this study we assessed differences in anal HPV 16/18 viral load between HIV-positive and HIV-negative MSM in Amsterdam, the Netherlands.

Conclusions

The anal HPV viral load of HPV 16 and HPV 18 did not differ significantly between HIV-positive and HIV-negative MSM. Therefore, we can conclude that difference in anal HPV-related disease burden of HPV 16/18 infection is not caused by differences in baseline HPV viral load.

Results

Table 1: Characteristics of the study population (N=777), stratified by HIV-status

	HIV-negative (N=460)		HIV-positive (N=317)		
	No.	%	No.	%	P-value
Median age in years (IQR)	38	(33-42)	46	(39-53)	<0.001
Country of birth					0.021
The Netherlands	381	84%	242	77%	
Any other country	74	16%	72	23%	
Tobacco smoking					0.006
Never	179	42%	81	30%	
Ever/in the past	107	25%	77	28%	
Current	145	34%	115	42%	
Median lifetime no. of male sex partners (IQR)	105	(50-400)	300	(100-1000)	<0.001
Median no. of anal sex partners last 6 months (IQR)	2	(1-4)	4	(1-10)	<0.001
Condom use during anal sex in the preceding 6 months					<0.001
Never	94	25%	36	14%	
Sometimes	159	42%	156	60%	
Always	126	33%	68	26%	
HPV 16/18 positivity					0.010
Only HPV 16	52	11%	49	15%	
Only HPV 18	20	4%	24	8%	
Both HPV 16/18	2	0%	15	5%	
HIV-related variables					
Use of cART at enrolment	235	87%			
Median CD4+ cell count at enrolment (cells/µ)(IQR)	530	(410-700)			
Median nadir CD4+ cell count (cells/µ) (IQR)	227	(160-320)			
Median HIV viral load at enrolment(copies/µ)(IQR)	20	(0-50)			

Table 2. HPV 16/18 viral load characteristics of the study population (N=777), overall and stratified by HIV-status (H2M study, Amsterdam 2010-2011)

	HIV-negative			HIV-positive		
	N	GMT	(95% CI)	N	GMT	(95% CI)
HPV 16	54	26.2	(10.2 - 67.1)	65	16.2	(6.4 - 41.1)
HPV 18	22	5.9	(0.5 - 66.6)	40	11.4	(3.2 - 40.0)

abbreviations: GMT - geometric mean titre, HPV - human papillomavirus, HIV - human immunodeficiency virus, CI - confidence interval

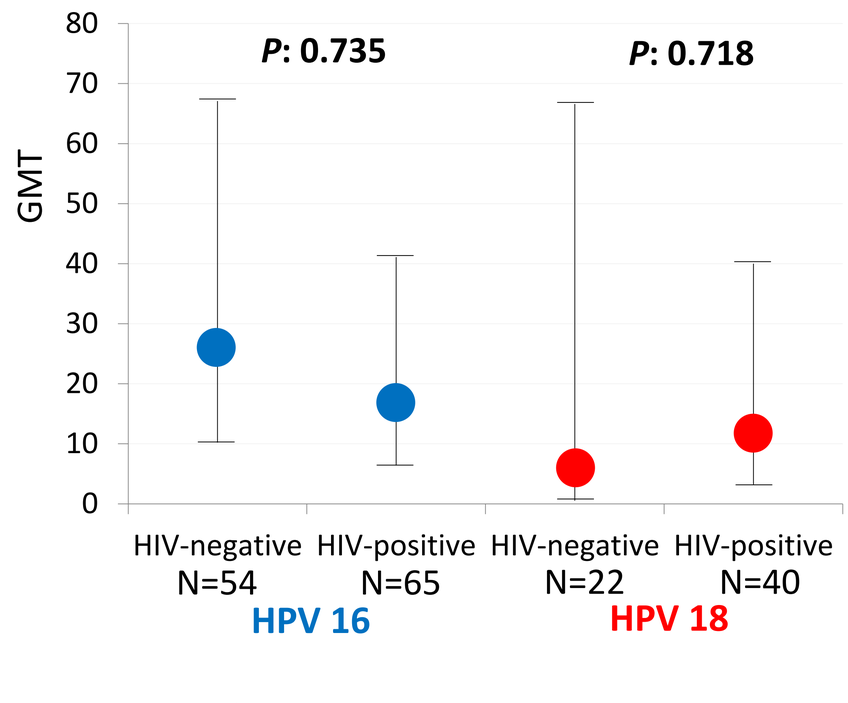

Figure 1: HPV 16/18 viral load geometric mean titer (GMT) of the no. of DNA copies per human cell, by HIV-status

Methods

MSM aged ≥18 years were recruited in Amsterdam, the Netherlands, in 2010-2011 (H2M study). Anal-self swabs were collected and typed using the SPF_{10}-PCR-DEIA-$LIPA_{25}$-system version 1.0. Anal HPV 16 and HPV 18 load was determined using a HPV 16 and HPV 18 type specific quantitative (q)PCR. The geometric mean titer (GMT) of HPV 16/18 load (DNA copies per human cell) was assessed and HPV 16/18 load of HIV-positive and HIV-negative men were compared with the Wilcoxon Ranksum test.

Discussion

Despite a relative large sample size we did not find a difference in HPV viral load between HIV-positive and HIV-negative MSM. The HPV viral loads are measured at baseline, therefore we were unable to determine the kinetics of infection at that point in time. A longitudinal comparison of HIV-positive and HIV-negative MSM, including persistence and clearance might provide more insight.