Journal of Medical Biomedical and Applied Sciences

J Med Biomed App Sci 8 (1), 324–330 (2020) ISSN (O) 2349-0748

Molecular Genotyping of Human Papillomavirus among HIV-infected and HIV- uninfected Women in Ouagadougou, Burkina Faso

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DOI: 10.15520/jmbas.v8i1.207

Accepted 9 January 2020; Received 29 December 2019; Publish Online 18 January 2020

Reviewed By: Dr. Daniel V.

ABSTRACT

This study particularly focused on the human papillomavirus (HPV) that causes cervical cancer. The objective was to study the profile and genotypic prevalence of HPV among HIV infected and HIV uninfected women.

Method: The study was conducted in Ouagadougou, from February 2009 to January 2013 and involved 421 women: 183 HIV positive women (HIV+) and 238 HIV-negative women (HIV-). PCR/hybridization and real-time PCR were performed for the detection of high and low-risk HPV genotypes.

Results: The two populations of women differed in socio-economic, behavioral and sexual characteristics. HPV prevalence was 24.8% and 63.9%, respectively, among HIV- and HIV+ women. Except for HPV16, HPV52, HPV58 and HPV6, which were higher in HIV- women compared to HIV+ women, it's the opposite effect that was found for the other genotypes. We found many more cases of co-infection with three or more genotypes in HIV- women compared to HIV+ women. Contraceptive use and low CD4 count were associated with HPV infection in HIV+ women ($p < 0.05$). Parameters such as age group, marital status, occupation, level of education, history of gynecological infection, and condom use differed according to HIV status ($p < 0.05$).

Conclusion: Through this study, we found that HPV are highly prevalent among HIV positive women in Burkina Faso. However, given the high prevalence of other HPV types than 16 and 18, in our study, another type of vaccine should be considered to cover them. In the meantime, the results of this study could be a springboard for the introduction of vaccines against HPV already existing in Burkina Faso.

Key words: HPV–HIV–Genotypes–Women–Burkina Faso

1 INTRODUCTION

Since the onset of Human Immunodeficiency Virus (HIV) infection, an increase in the frequency of cervical lesions (dysplasia and invasive cervical cancer) has been described [1]. From 1997, the Center of Disease Control and prevention of Atlanta recommended regular gynaecological monitoring of women infected with HIV [2] . It is now certified that high-risk HPV (HR-HPV) cause approximately 95% of precancerous and cancerous lesions of the cervix. Currently, there are 3 prophylactic vaccines on the market: Cervarix (GlaxoSmithKline, Brentford, UK) which is bivalent and target HPV16/18; Gardasil (Merck Inc, NY, USA), which is quadrivalent and target HPV6/11/16/18 and Gardasil-9 (Merck Inc., NY, USA), which is nonavalent and target HPV 6/11/16/18/31/33/45/52/58. This means that prevention against cervical cancer is a possibility especially for developing countries like Burkina Faso where prevalence of HPV is high. The relationship between the frequency, severity of cervical dysplasia and the degree of immune deficiency was quickly established. The transmission of HPV is mainly through sex. Indeed, HPV infection is the commonest STI in the world. Seventy-five percent of women are infected with HPV at least once during their sex life [3, 4]. At the ages of greatest sexual activity, the prevalence of infection by subclinical HPV can reach more than 40% of the female population [5]. However, it varies according to the different regions of the world with the highest in Africa [6]. In general, HPV16 is the most widespread with a prevalence of 26.3% in the world population [7].

Burkina Faso is no exception in terms of the prevalence of HPV. For the past ten years, fairly regular studies have been carried out in several regions of the country and they are all unanimous regarding the prevalence of HPV, which is between 24 to 41% for women in the general population and around 60% for women infected with HIV [8–16].

Most comparative studies in Africa have found very high prevalence of HPV in HIV positive women compared to HIV negative women [17–23].

The objective of this study was to compare the frequencies and genotypes of HPV in HIV positive and HIV negative women in Ouagadougou, Burkina Faso.

2 METHODS

2.1 Study design and population, sample collection

The samples were taken from May 2009 to January 2010 in three reference and much frequented health centres in the capital of the country, Ouagadougou: Saint Camille hospital of Ouagadougou (HOSCO), the teaching hospital centre of Bogodogo and the Pietro Annigoni Biomolecular Research Centre (CERBA). These three centres are located on the outskirts of the city and are accessible to all strata of the population. The real-time PCR analysis was performed.

The Saint Camille Hospital of Ouagadougou (HOSCO) and the Biomolecular Research Centre Pietro Annigoni (CERBA) are reference centres in Burkina Faso for the management of people living with HIV/AIDS (PLHIV). The samples were taken by three gynaecologists on site and the samples were stored at -80 ° C pending further handling.

We included in the study 183 women screened positive for the carriage of anti-HIV antibodies (HIV positive), at the asymptomatic stage of infection and 238 HIV negative women came in gynaecological consultation without distinction of age. HIV-positive women were selected from those monitored at HOSCO and CERBA and who must be screened annually for cervical cancer. The consent of these two groups of women was obtained by the doctor after detailed explanation of the type of examination to be performed, the possible results expected as well as the possible management from which they will benefit. Each woman answered a questionnaire in order to determine socio-economic, professional status and certain behavioural habits.

2.2 Ethical Aspects

The Ethics Committee of the Saint Camille Medical Center and the Pietro Annigoni Biomolecular Research Center has given its formal agreement for this study (n°2009-009/CR/135 of 22 April 2009) and each woman gave her informed consent before the samples were taken.

2.3 Sampling

Samples were collected with sterile cotton swabs from the endocervix. The collected samples were then allowed in sterile and dry extraction tube and stored at −80 ∘C prior to DNA extraction.

2.4 DNA Extraction

The DNA extraction was assayed from swabs using “INSTANT Virus DNA Kit” Analytkjena® (Italy) bio solutions following the protocol provided by the manufacturer. We used extraction columns in which we placed the DNA extract obtained after the lysis of membranes and hydrolysis of the proteins. The DNA extract was then washed and eluted.

2.5 Research and genotyping of HPV

For this step, we used two different techniques: a first one called PCR/Hybridization for the diagnosis of high risk HPV 16, 18, 45, 30’S, 50’S and low risk HPV 6 and 11. This technique does not allow us to specify the 30’S and 50’S genotypes. We completed our genotyping with a second technique using the Sacace biotechnologies® kit which allowed us to specify the following high-risk genotypes: 16, 18, 31, 39, 45, 59, 33, 35, 56, 51, 52, 58. The use of these two techniques allowed us at the same time to verify the concordance of the results between the two methods.

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2.5.1 PCR / Hybridization

The PCR/hybridization was performed using a kit "HPV Blot STAR" of Diatech® (Italy). This assay enables the detection of HPV and the differentiation of high-risk and low-risk HPV strains by in vitro amplification of the gene L1 with biotinylated primers and subsequent reverse dot blot hybridization with sequence-specific oligonucleotides probes. The kit can detect the following HPV genotypes: 16, 18, 30'S, 45, 50'S, 6, and 11. The PCR program was previously described [8].

2.5.2 Detection of HPV genotypes with the kit « HPV High Risk Typing Real-TM» (SACACE biotechnologies®, Italie)

The High Risk HPV Typing Real-TM kit is based on two major processes: isolation of DNA from specimens, and multiplex Real Time amplification of 4 tubes for each sample. Each tube contains primers directed against regions of three HPV types and the β-globin gene used as Internal Control. PCR conditions were as follows : 1 cycle of 95°C for 15 minutes ; 5 cycles of 95°C for 05s, 60°C for 20s and 72°C for 15s; and 40 cycles of 95°C for 05s, 60°Cfor 30s and 72°C for 15s.

2.6 Statistical analysis

Data were analysed using SPSS 20.0 and Epi Info 3.5.1 softwares. The Chi-square test was used for comparisons. Statistical difference was calculated with Epi Info 6 and was significant for $p \leq 0.05$.

3 RESULTS

3.1 Socio-economic, behavioral and sexual characteristics of women in this study based on HIV status

We enrolled a total of 421 women in our study: 238 HIV negative women constituting the control group and 183 other HIV positive women considered as cases. The average age of our study population was 33.91 ± 6.18 years (Minimum = 20; Maximum = 53) and 30.67 ± 8.03 years (Minimum = 15 years; Maximum = 63 years) respectively for HIV + and HIV- women. By comparing the socio-economic, behavioural and sexual characteristics of these two groups of women, we notice that there are statistically significant differences between them regarding all characteristics exept the number of visits to the gynaecologist by year and the use of contraceptives. Table 1. In HIV positive women, the average CD4 count was 401.67 ± 211.03 cells / uL (Minimum = 6 and Maximum = 1382).

3.2 Different genotypes of HPV found among HIV positive and HIV negative women

Overall, 41.81% of the women in this study were diagnosed with HPV, with 24.8% (59/238) of HPV in HIV uninfected women and 63.9% (117/183) in HIV infected women ($p < 0.001$). Genotyping of different HPV found in our sample shows quite different prevalence of HPV strains based on HIV status. The HPVs 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 and 6 are found in the two groups. We obtained respectively in HIV+ and HIV- women the following proportions Table 2 : HPV18 (18.8% versus 15.2%), HPV35 (13.3% versus 8.1%), HPV31 (10.7% versus 7.1%), HPV52 (8.8% versus 12.1%), HPV58 (8.1% versus 11.1%), HPV56 (7.8% versus 7.1%), HPV45 (6.2% versus 6.1%), HPV59 (5.8% versus 5.1%), HPV33 (5.8% versus 1, 9%), HPV51 (5.2% versus 5.1%), HPV16 (4.9% versus 7.1%), HPV39

Table 1. Socio-economic, behavioural and sexual characteristics of women in our study based on HIV status

		HIV- n(%)	HIV+ n(%)	P value
Average age (years)		30.67±8.03	33.91±6.18	< 0.001
Average age at first intercourse (years)		18.90±2.66	18.18±2.36	< 0.001
Marital status	Married	178 (74.8%)	107 (58.5%)	< 0.0001
	Divorced	5 (2.1%)	18 (9.8%)	
	Widow	2 (0.8%)	34 (18.6%)	
	Single	53 (22.2%)	24 (13.1%)	
Profession	Unemployed	90 (37.8%)	116 (63.4%)	< 0.0001
	Merchant	21 (8.8%)	9 (4.9%)	
	Salaried	24 (10.1%)	23 (12.6%)	
	Informal sector	69 (29.0%)	31 (16.9%)	
	Students	34 (14.3%)	4 (2.2%)	
Study level	No formal education	76 (31.9%)	53 (29.0%)	< 0.0001
	Primary school	47 (19.7%)	62 (33.9%)	
	Secondary school	87 (36.6%)	65 (35.5%)	
	University	28 (11.8%)	3 (1.6%)	
Number of visits to the gynaecologist by year	Never	174 (73.1%)	133 (72.6%)	0.994
	1<X≤2	37(15.6%)	29 (15.9%)	
	X> 2	27 (11.3%)	21 (11.5%)	
History of gynecological infection	Yes	97 (40.8%)	116 (63.4%)	< 0.0001
	No	141 (59.2%)	67 (36.6%)	
Contraceptive use	Yes	70 (29.4%)	41 (22.4%)	
	No	168 (70.6%)	142 (77.6%)	0.106
HPV frequency	HPV+	59 (24.8%)	117 (63.9%)	
	HPV-	179 (75.2%)	66 (36.1%)	< 0.0001

(2.6% versus 1.9%), HPV6 (2.0% versus 12.1%).

Table 2. Different genotypes of HPV found among HIV positive and HIV negative women

HPV Genotypes	HIV+	HIV-	P value
HPV16	15 (4.9%)	7 (7.1%)	0.4
HPV18	58 (18.8%)	15 (15.2%)	0.406
HPV31	33 (10.7%)	7 (7.1%)	0.289
HPV33	18 (5.8%)	2 (1.9%)	0.206
HPV35	41 (13.3%)	8 (8.1%)	0.164
HPV39	8 (2.6%)	2 (1.9%)	0.96
HPV45	19 (6.2%)	6 (6.1%)	0.968
HPV51	16 (5.2%)	5 (5.1%)	0.955
HPV52	27 (8.8%)	12 (12.1%)	0.324
HPV56	24 (7.8%)	7 (7.1%)	0.813
HPV58	25 (8.1%)	11 (11.1%)	0.361
HPV59	18 (5.8%)	5 (5.1%)	0.766
HPV6	6 (2.0%)	12 (12.1%)	< 0.0001
Total	308 (100%)	99 (100%)	

Multiple HPV infections were found for 53.4% (94/176) of all HPV infections. These types of infections concerned 62.4% of all infections in HIV negative women and 35.6% of all infections in HIV positive women. In 46.6% (82/176) remaining, only one type of HPV was present. The maximum number of genotypes found in HIV negative women was 5 and 7 in HIV positive women.Table 3

Table 3. Co-infections with one or more genotypes depending on HIV status

Co-infections	HIV+	HIV-	P value
1 genotype	38 (64.4%)	44 (37.6%)	0.0007
2 genotypes	9 (15.3%)	14 (12.0%)	0.541
3 genotypes	7 (11.9%)	23 (19.6%)	0.194
4 genotypes	3 (5.0%)	14 (12.0%)	0.144
5 genotypes and more	2 (3.4%)	22 (18.8%)	0.005
Total	59 (100%)	117 (100%)	

3.3 HPV prevalence in relation to HIV status and socio-economic, behavioural and sexual characteristics

In HIV negative women, no characteristic was correlated with HPV infection. In contrast, among HIV+ women, taking contraceptives and low CD4 counts were statistically associated with HPV infection. However, when we compare HIV-/HPV+ women to HIV+/HPV+ women, we found that age classes, marital status, profession, education, history of gynaecological infections and use of condoms were significantly associated with HPV infection and HIV status.Table 4

4 DISCUSSION

The average age of our two study populations is around 30 years, reflecting screening for HPV in predominantly young women, and in an age group where they are sexually active. HIV uninfected women had their first sexual intercourse on average later than HIV positive women. This information, added to the fact that they more often live alone than HIV uninfected women (over 74% are married) could help to explain why there are more of gynaecological infections history in HIV+ women than HIV- women ($p < 0.0001$). In terms of occupation as well as level of education, HIV+ women were less represented in the workforce than HIV- women. This is complementary to the high level of study (higher and secondary) which was much more common among HIV-women. Although we have not investigated the discrimination of positive HIV status, there is no longer any evidence that people living with HIV experience discrimination that is even more pronounced among women. And this fact, in addition to the disease could help explain why in our population of women infected with HIV we have fewer working women. We find through our results that women infected with HIV attend health centres because of their status but this did not influence their number of visits to the gynaecologist per year. Generally, a woman goes to the gynaecologist when she is referred by a health worker. And health workers only refer patients to specialists when indicated as per the working guidelines. Precisely through regular medical consultations enjoyed by women living with HIV, this promotes fast and fairly effective management of diseases that otherwise could have developed negatively. We also note that contraceptives usage rate is low for both HIV+ and HIV- women. This reflects the national reality of the use of contraceptive methods.

The prevalence of HPV was 24.8% and 63.9% respectively among HIV negative and HIV positive women ($p < 0.0001$). This confirms that HIV+ status has a negative influence on the prevalence of HPV, especially because these two viruses have in common the sexual route as the main route of transmission. Other studies have come to a similar conclusion [24, 25]. Knowing that HIV-induced immuno suppression can also play an important role in the persistence of HPV infection and the onset/persistence of precancerous and cancerous lesions, it seems important to us that the population of HIV positive women has very early burden of precancerous lesions. We also recommend the implementation of systematic vaccination of all young girls infected with HIV (from the age of 9) who have contracted their infection vertically (young girls born to HIV positive mothers). After an initial infection and a latency of around three weeks, active viral replication of HPV follows and continues for a variable duration. When HPV DNA can no longer be detected by available technology, it is ”clearance”, but the virus can be latent and reactivate when conditions are favourable. Among HIV-negative populations with normal immunity, approximately 10-20% of infections become persistent. It is suspected that in PLHIV, this prevalence is higher. Host factors are therefore important in determining viral suppression via cellular immunity [26, 27].

A total of 13 different HPV genotypes were found in this study. This is mainly due to the limit of our genotyping kits that did not allow us to look for more. In women infected with HIV, the genotypes found in decreasing order were: HPV18 (18.8%), HPV35 (13.3%), HPV31 (10.7%), HPV52 (8.8%), HPV58 (8.1%), HPV56 (7.8%),

Table 4. HPV prevalence in relation to HIV status andsocio-economic, behavioural and sexual characteristics

		HIV negative N= 238			HIV positive N=183			Total (HIV+/HIV-) N=421		P***
		HPV- N= 179	HPV+ N= 59	p*	HPV- N= 66	HPV+ N= 117	p**	HPV- N= 245	HPV+ N= 176	
Age group (years)	X< 29	92 (51.4%)	31 (52.5%)	0.364	19 (28.8%)	26 (22.2%)	0.468	111 (45.3%)	57 (32.4%)	< 0.0001
	30≤X<39	65 (36.3%)	17 (28.8%)		34 (51.5%)	71 (60.7%)		99 (40.4%)	88 (50.0%)	
	X ≥ 40	22 (12.3%)	11 (18.6%)		13 (19.7%)	20 (17.1%)		35 (14.3%)	31 (17.6%)	
Marital statut	Married	139 (78.1%)	39 (66.1%)	-	42 (63.6%)	65 (55.6%)	0.441	181 (73.8%)	104 (59.1%)	< 0.0001
	Divorced	2 (1.1%)	3 (5.1%)		5 (7.6%)	13 (11.1%)		7 (2.9%)	16 (9.1%)	
	Widow	2 (1.1%)	-		9 (13.6%)	25 (21.4%)		11 (4.5%)	25 (14.2%)	
	Single	36 (19.7%)	17 (28.8%)		10 (15.2%)	14 (12.0%)		46 (18.8%)	31 (17.6%)	
Profession	Unemployed	72 (40.2%)	18(30.5%)	0.384	45 (68.2%)	71 (60.7%)	-	117 (47.8%)	89 (50.6%)	< 0.0001
	Merchant	16 (8.9%)	5 (8.5%)		3 (4.5%)	6 (5.1%)		19 (7.8%)	11 (6.3%)	
	Salaried	16 (8.9%)	8 (13.6%)		4 (6.1%)	19 (16.2%)		20 (8.2%)	27 (15.3%)	
	Informal sector	53 (29.7%)	16 (27.1%)		14 (21.2%)	17 (14.5%)		67 (27.3%)	33 (18.7%)	
	Students	22 (12.3%)	12 (20.3%)		-	4 (3.4%)		22 (8.9%)	16 (9.1%)	
	No formal education	61 (34.1%)	15 (25.4%)		17 (25.7%)	36 (30.8%)		78 (31.8%)	51 (29.0%)	
Study level	Primary school	36 (20.1%)	11 (18.6%)	0.519	24 (36.4%)	38 (32.5%)	-	60 (24.5%)	49 (27.8%)	0.005
	Secondary school	63 (35.2%)	24 (40.7%)		25 (37.9%)	40 (34.2%)		88 (35.9%)	64 (36.4%)	
	University	19 (10.6%)	9 (15.3%)		-	3 (2.5%)		19 (7.8%)	12 (6.8%)	
Number of visits to the gynecologist last year	Never	129 (72.1%)	45 (76.2%)	0.667	51 (77.3%)	82 (70.1%)	0.345	180 (73.5%)	127 (72.1%)	0.503
	1<X≤2	30 (16.8%)	7 (11.9%)		7 (10.6%)	22 (18.8%)		37 (15.1%)	29 (16.5%)	
	X> 2	20 (11.1%)	7 (11.9%)		8 (12.1%)	13 (11.1%)		28 (11.4%)	20 (11.4%)	
History of sexually transmitted infection	Yes	75 (41.9%)	22 (37.3%)	0.532	41 (62.1%)	75 (64.1%)	0.789	116 (47.3%)	97 (55.1%)	0.0007
	No	104 (58.1%)	37 (62.7%)		25 (37.9%)	42 (35.9%)		129 (52.7%)	79 (44.9%)	
Use of contraceptive	Yes	53 (29.6%)	17 (28.8%)	0.907	22 (33.3%)	19 (16.2%)	0.008	75 (30.6%)	36 (20.5%)	0.051
	No	126 (70.4%)	42 (71.2%)		44 (66.7%)	98 (83.8%)		170 (69.4%)	140 (79.5%)	
Use of condom during sexual intercourse	No	127 (70.9%)	32 (54.2%)	0.075	11 (16.7%)	25 (21.4%)	-	138 (56.3%)	57 (32.4%)	< 0.0001
	Rarely	5 (2.8%)	4 (6.8%)		-	2 (1.7%)		5 (2.0%)	6 (3.4%)	
	Sometimes	21 (11.7%)	11 (18.6%)		11 (16.7%)	19 (16.2%)		32 (13.1%)	30 (17.1%)	
	Every time	15 (8.4%)	4 (6.8%)		28 (42.4%)	39 (33.3%)		43 (17.6%))	43 (24.4%)	
	No answer	11 (6.2%)	8 (13.6%)		16 (24.2%)	32 (27.4%)		27 (11.0%)	40 (22.7%)	
Number of pregnancies	0	36 (20.1%)	13 (22.0%)	0.591	3 (4.5%)	8 (6.8%)	0.9	39 (15.9%)	21 (11.9%)	0.203
	1	41 (22.9%)	15 (25.4%)		8 (12.1%)	12 (10.3%)		49 (20.0%)	27 (15.3%)	
	2	29 (16.2%)	10 (16.9%)		10 (15.2%)	20 (17.1%)		39 (15.9%)	30 (17.1%)	
	3	33 (18.4%)	6 (10.2%)		9 (13.6%)	11 (9.4%)		42 (17.4%)	17 (9.7%)	
	4	22 (12.3%)	5 (8.5%)		5 (7.6%)	11 (9.4%)		27 (11.0%)	16 (9.1%)	
	5 et plus	18 (10.1%)	9 (15.3%)		6 (9.1%)	13 (11.1%)		24 (9.8%)	22 (12.5%)	
	Non répondant	-	1 (1.7%)		25 (37.9%)	42 (35.9%)		25 (10.2%)	43 (24.4%)	
CD4 count	X≤ 350 cells/uL	-	-	-	21 (31.8%)	58 (49.6%)	0.019	-	-	-
	X> 350 cells/uL	-	-		45 (68.2%)	59 (50.4%)		-	-	

HPV45 (6.2%), HPV59 (5.8%), HPV33 (5.8%), HPV51 (5.2%), HPV16 (4.9%), HPV39 (2.6%) and HPV6 (2.0%). In HIV infected women, the genotypes found in decreasing order were: HPV18 (15.2%), HPV52 (12.1%), HPV6 (12.1%), HPV58 (11.1 %), HPV35 (8.1%), HPV56 (7.1%), HPV31 (7.1%), HPV16 (7.1%), HPV45 (6.1%), HPV51 (5.1%), HPV59 (5.1%), HPV33 (1.9%), HPV39 (1.9%). HPV18, 35, 52 and 58 are among those who are leading in prevalence in women infected with HIV or not. These are high-risk HPV and therefore may cause damage that may change later to cervical cancer. In women not infected with HIV, HPV6 is still one of the top 5 genotypes found in terms of prevalence. HPV6 is relatively uncommon in women infected with HIV, as confirmed by a study carried out in Burkina in 2012 [28]. The authors of this study had found 1.5% of HPV11, which is not the case in our study. We assume that with a larger population, we would have had roughly the same frequencies. The most prevalent high-risk genotypes in our study differ from those found in other countries, like Kenya where the top 4 in women infected with HIV was HPV16, 53, 66 and 58 and in women not infected, we had HPV58, 45, 52 and 53 [29]. In Kenya, a meta-analysis found that HPV52 and 35 were the most common genotypes found in women with HIV infection [30]. In Ghana, Obiri-Yeboah et al., found HPV35, 52, 58 and 18 as the most prevalent genotypes in HIV infected women and HPV35, 33, 18 and 56 as the most prevalent genotypes in HIV uninfected women [25]. However, we do keep in mind that we have been looking for HPV genotypes in women who did not necessarily have cervical lesions. It would be interesting to repeat the same study but in women with cancerous lesions. This will allow us to better specify the genotypes responsible for cancer in Burkina.

By comparing the socio-economic, behavioural and sexual parameters of our study population with HIV status and HPV infection, we find that only contraceptive use and low CD4 count appear to be associated with HPV infection in women infected with HIV. However when we compare these same parameters between HIV+/HPV+ women and HIV-/HPV+ women, we have a $p < 0.05$ for some of the parameters such as age group, marital status, profession, level of education, history of gynaecological infections and condom use. Regarding age groups, HPV was more found among women not infected with HIV under 29 and for those aged 30-39 infected with HIV. These two age groups roughly correspond to the active period of sexual life, therefore an increased risk of contracting HPV infection in women. Our results are similar to those of Obiri-Yeboah et al., 2017 [25]. For marital status, married women were more affected by HPV in the two study populations. This means that the status of a married woman does not protect against acquiring HPV infection in our study. Perhaps also the number of sexual partners could explain it. Especially that men play a key role in the virus transmission chain.

When we look at the profession, we notice that housewives/unemployed women were more affected by HPV in women infected with HIV compared to uninfected women. This could be partly due to the fact that this study has find many more housewives in women infected with HIV compared to uninfected women. And this may be the result of the discrimination that people have against PLHIV. This last explanation is consistent with the results we obtained in relation to the level of study: women with a secondary and higher school level were more infected with HPV in HIV- women (56.0%) compared to HIV+ women (36, 7%). Clearly, in HIV+ women, the history of gynaecological infection was associated with HPV infection (62.1%) while in HIV- women it was quite the opposite (37.3%).

5 CONCLUSION

Women infected with HIV are much more infected with HPV than women who are not infected with HIV. As a special population, these women are at increased risk of cervical cancer. The overall risk of co-morbidities will increase with the aging of the HIV positive population favoured by the effectiveness of treatment. Prevention of cervical cancer, therefore, include the implementation of an effective vaccine against HPV in HIV positive children, extending the screening program for cervical cancer, early diagnosis and treatment of precancerous and cancerous cervix lesions.

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