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HPV Genotypes and Determinants of Infection Among HIV Positive Women in Federal Medical Centre Keffi: A Cross-Sectional Study

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Abstract:

- ***Background:***

Human immunodeficiency virus (HIV) infection facilitates the acquisition, enhance persistence and also alters the spectrum of human papillomavirus (HPV) infection in HIV infected individuals. The knowledge of the predominant genotypes and associated factors of high-risk human papillomavirus (HrHPV) infection among HIV-positive women is necessary for implementing interventions such as targeted screening for this vulnerable group and developing effective polyvalent HPV vaccine.

- ***Objectives:***

We determined the predominant HrHPV genotypes and associated risk factors for infection among HIV positive women in Keffi, Nasarawa state, Nigeria.

- ***Methods:***

In a cross- sectional study, we analyzed the data of 220 HIV positive women who came for routine ART clinic visits. Consenting participants had their socio-demographic and other personal information taken, thereafter cervical sampling was done for each which was used for molecular analysis. Multivariable binary logistic regression models were performed to explore factors associated with HPV infections in the participants.

- ***Results:***

Of the 220 participants enrolled, 119 tested positive for HPV DNA, giving a total HPV prevalence of 54.1%, while participants with the HrHPV genotypes were 79 out of the total 119 positive for HPV DNA, giving a prevalence of 35.9% for HrHPV genotypes. The five predominant high-risk genotypes are HPV35 (20.5%), HPV16 (14.2%), HPV45 (13.4%), HPV33 (9.4%) and HPV18 (4.7%) in decreasing order while the predominant low risk HPV genotypes were HPV6 (7.1%), HPV11 (4.7%), HPV72 (4.7%), HPV81 (3.9%) and HPV42, HPV43 both with 3.1%. Multiple infections were recorded in 22.5% of the participants having more than one genotype.

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➢ ***Conclusion:***

Our finding highlights different HrHPV genotype distribution in HIV-positive women and thus the need for targeted screening in this vulnerable group, development of polyvalent HPV vaccine with a wider coverage to include the prevalent identified genotypes in our study and put in place program for mass vaccination of young girls.

Keywords: *HPV, HIV, Genotypes, High Risk HPV, Vaccination.*

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I. INTRODUCTION

HPV is the most prevalent sexually transmitted virus, with estimates suggesting about 75% of sexually active individuals will contract a genital HPV infection at some point in their lives, with approximately 20 million women infected globally [1].

The global prevalence of genital HPV has been estimated at about 11–12% [2].

Human papilloma viruses are double-stranded, non-enveloped DNA viruses. These viruses infect mucosal and cutaneous epithelia [1].

More than 100 different genotypes of HPV have been discovered, several of which affect the genital area [2].

The prevalence of HPV differ in different regions of the world with the highest prevalence of 22.1% reported in Africa [2]. There are also regional variations within Africa depending on the population being considered [2].

In Sudan and Senegal, HPV prevalence has been documented at 2.2% and 18%, respectively, while South Africa, Burkina Faso, Zimbabwe, and Benin Republic reported a prevalence between 20% and 33%. In rural areas of Mozambique, Nairobi, Morocco, and Zambia, much higher prevalence rates of 40% and above have been observed. In Nigeria, the prevalence of HPV differs across the country, with rates ranging from 10.0% in Irun, Ondo State, to 21.6% in Ile-Ife, Osun State, and 26.3% in Ibadan, Oyo State [2].

Prevalence rates of 30.4 and 36.5% were also reported in Lagos [1] and [3] respectively. A pooled prevalence of 14.9% was reported in Saudi Arabia [4] while in Beijing China , a prevalence of 12.18% was reported among healthy females [5].

The prevalence of HPV infection among HIV infected women in Uganda was put at 87.8% [6].

HIV is associated with higher rates of HPV acquisition, decreased clearance of HPV, precancerous lesions, and increased risk of cervical cancer [7].

Persistent HPV infection may be influenced by a number of viral, host, and environmental factors, such as HPV type, multiple HPV infections, viral load, infection with human immune deficiency virus (HIV), older age, and smoking [8].

Both HPV and HIV are sexually transmitted and infection with either of them could facilitate infection with the other. And HIV is also known to alter the spectrum of HPV serotypes in HIV positive women [9].

II. METHODOLOGY

➢ *Study Area and Population*

The study was conducted at the antiretroviral clinic of Federal Medical Centre, Keffi in Nigeria, between August 2016 and May 2017. Prospective participants who met the criteria were briefed about the study and were enrolled, they were aged between 20 and 50 years of age, not pregnant and did not have a history of hysterectomy. During the enrolment visit, socio-demographic data were collected and cervical sampling for HPV testing was done. CD4+T cell counts and HIV viral load done within six months prior to the study were also retrieved from the patients' records. A written informed consent was obtained from each participant at enrollment.

- *Ethical Approval*

Ethical approval was granted by the Health Research Ethics Committee of the Federal Medical Centre, Keffi (KNH-ERC/01/3618).

➢ *Specimen Collection and Storage*

Rovers® Cervex-Brush® cell sampling device (Rovers Medical Devices B.V 5347 KV Oss, The Netherlands) and liquid based cytological processing and preservative reagent (Zeni-Prep®, Zenith diagnostics, USA) were used for specimen collection and transport. Specimen was obtained by inserting the cytobrush into the cervical canal and rotating it four times to collect all the cervical epithelial cells which adhered to the flat sides of the bristles. The brush was then transferred into the vial containing preservative fluid.

➢ *DNA Extraction*

HPV testing was done at the DNA Labs, Kaduna, Nigeria. Gel extraction prior to DNA purification for sequencing was done using QIAquick Gel Extraction Kit (QIAGEN Sample & Assay Technologies, Germany). Briefly, HPV DNA was extracted from exfoliated cervical cells using proteinase K-based digestion protocol. Cells were incubated with proteinase K solution (100 µg/ml) for three

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hours at 55°C. DNA was then further purified by spin column chromatography.

➢ *Detection and Typing of HPV*

The extracted DNA was amplified for HPV using a nested PCR approach with GP5+/GP6+([GP5 + 5¹-TTT GTT ACT GTG GTA GAT ACTAC-3¹], GP6+[5¹-GAAAAATAAACTGTAAATCATATTC-3¹]) and PGMY 09/11 primer sets (MY/GP(+)**[10]** that amplify a conserved 150 bp sequence of the L1 open reading frame. AccuPowerHotStart PCR Premix (Bioneer Corporation, South Korea) was used for the PCR while genotypic identification was achieved by direct sequencing using the Gp6+ oligoprimer. Assays were normalized to a reference gene and a calibrator was included in every run. Thermal cycler (BioRad) and Beckman Coultier CEQ 2000XL were used for PCR and sequencing respectively. Sequence alignments were performed against various standard HPV genotype sequences stored in the GenBank database by on-line BLAST analysis to arrive at specific genotyping.

➢ *Data Analysis*

Descriptive and inferential statistical analysis of the data was done using the statistical package for social sciences (SPSS) version 20 (SPSS Inc, Illinois, USA) for analysis after validation. Categorical variables were summarized as proportions and further analyzed using Chi square test to assess significance of association between the variables.

III. RESULT

Two hundred and twenty subjects participated in the study, of which majority 79 (35.9%) were in the age group 32-38 years followed by 56(25.5%) in the age groups 25-31years. More than half of the participants 133(60.5%) were married with 149 (67.7%) being Christians and 70 (31.8%) Muslims. Most of them 138 (65.4%) live in a monogamous family setting while 73(34.6%) live in polygamous families. Low level of education was recorded among the participants, where 67 (30.5%) had no formal education, 69 (31.4%) had only primary education, 60 (27.3%) had secondary education and only 24 (10.9%) had tertiary education. Majority of the participants 115 (52.3%) were unemployed while 33 (15%) were employed either in the public or private sector.

Low economic status was seen in the participants with 175(79.5%) having a monthly family income of less than 20,000 naira, Early sexual debut at 16-20 year was recorded in majority of them 125 (56.8%) while 40(18.2%) reported ages 21-25years and 37(16.8%) reported sexual debut before 16 years of age.

Multiple sexual partners of 2-4 as a risk factor was seen in (45.5%) of the participants while 18(8.2%) had between 5-7 sexual partners and 6(2.7%) had between 8-10 partners.

Majority of the participants 119(54.1%) had never used a condom during sexual intercourse while 81(36.8%) reported using condom only sometimes. HPV DNA was detected in 119(54.1%) of the participants and of this, 79 (35.9%) were high risk HPV while 34 (15.5%) were low risk HPV. The five predominant high-risk genotypes were HPV35 (20.5%), HPV16 (14.2%), HPV45 (13.4%), HPV33 (9.4%) and HPV18 (4.7%). The predominant low risk HPV serotypes were HPV6 (7.1%), HPV11 (4.7%), HPV72 (4.7%), HPV81 (3.9%) and HPV42, HPV43 both with (3.1%). Multiple infections were recorded in 22.5% 0f the participants.

HPV infection was highest (31.1%) in the age group 32-38years followed by ages 39-45years (26.1%) and ages 25-31years (23.5%) respectively.

Among the 175 participants with family income of less than 20,000 naira per month, 90(51.4%) were positive for HPV DNA and of the 28 who reported earning 20,000 - 39,000 naira per month, 14(50.0%) were positive for HPV DNA while 9(69.2%) of the 13 who reported earning 40,000-59,000 naira per month also had HPV infection. None of the 3 who earn between 60,000–79,000 naira per month and the one who earned at least 80,000 naira per month was positive for HPV infection. There was no significant statistical association between monthly family income and HPV infection in the study population.

Concerning number of life time sexual partners, of the 100 who had between 2-4 sexual partners, 54 (54%) tested positive for HPV DNA with 37 (68.5%) of this 54 having high risk HPV. while among the 91 who had only one sexual partner, 45(49.5%) were positive for HPV DNA of which 33(73.3%) had high risk HPV. The 18 participants who had between 5-7 sexual partners, 9(50%) were positive for HPV DNA with 6(66.7%) of them being high risk.

Of the six participants who had between 8-10 partners, 2(33.3%) were positive for HPV DNA with 1(50%) of these two being high risk. Also among the 5 participants who had ten or more sexual partners, 3(60%) were positive for HPV DNA with 2(66.7%) of them being high risk. There was no statistically significant association between life time sexual partners and HPV infection in this study.

Pertaining condom use, of the 119(54.1%) who reported they ‘never used a condom’, 64(53.8%) of them were positive for HPV DNA with 45(70.35) of this 64 being high risk, while of the 20 (9.1%) that reported they ‘use condom always’, 8(40%) were positive for HPV DNA with 5(62.5%) of them being high risk. Of the 81 (36.8%) participants who reported they ‘used condom only sometimes’, 64(79.0%) were positive for HPV DNA with 45(70.3%) of them being high risk. There was no significant statistical association between condom use and HPV infection.

Two hundred and fifteen 215 (97.7%) of the participants were on HAART and CD4+ counts estimated within six months of commencement of study were available for 198 (90%) of the participants. Ninety-one (46%) of the participants had CD4+ values greater than 500 cells/ mm3, 39(19.7%) had CD4+ values between 301-400 cell/mm3, 22(11.1%) had CD4+ values between 201-300 cells/mm3 and only 18(9.1%) had values below 200cells/mm3. There was no significant statistical association between CD4+ count and HPV infection.

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Of the 220 participants, only 50(22.7%) had viral load estimation within six months of sample collection. Thirty-seven (74%) had less than 5000 copies /ml, 3(6%) had between 5001-10,000copies/ml, 2(4%) had estimates between 10001-15000copies/ml, 2(4%) had between 15001-20000 copies/ml and 6(12%) had estimates of more than 20,000 copies/ml. There was no statistically significant association between HIV viral load and HPV infection.

Table 1; Frequency of HPV Genotypes Among Participants

HPV Ggenotype	**No of patients**	**Percentage**
HPV6	9	7.1
HPV11	6	4.7
HPV16**	18	14.2
HPV18** HPV31**	6 2	4.7 1.6
HPV33**	12	9.4
HPV35**	26	20.5
HPV39**	1	0.8
HPV40	1	0.8
HPV42	4	3.1
HPV43	4	3.1
HPV44	1	0.8
HPV45**	17	13.4
HPV51**	1	0.8
HPV52**	1	0.8
HPV56**	4	3.1
HPV66	3	2.4
HPV72	6	4.7
HPV81	5	3.9
Multiplicity of HPV infection		
Single infection	84	77.5
More than one infection	25	22.5
Total	119	100.0

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Table 2; Sexual Risk Factors of the Participants

Variable	Frequency	Percentage
Age at sex		
<16	37	16.8
16-20	125	56.8
21-25	40	18.2
26-30	8	3.6
>30	10	4.5
Total	**220**	**100**
Number of life time sexual partners		
1	91	41.4
2-4	100	45.5
5-7	18	8.2
8-10	6	2.7
>10	5	2.3
Total	**220**	**100**
Condom use		
Always	20	9.1
sometimes	81	36.8
Never	119	54.1
Total	**220**	**100**
Does your husband have other women outside?		
Yes	54	94.0
No	19	6.0
I don't know	147	66.8
Total	**220**	**100.0**
Have you had an STI in the past?		
Yes	107	48.6
No	113	51.4
Total	**220**	**100.0**
Did you notice wart growth on your partner's genital area?		
Yes	8	3.6
No	212	96.4
Total	**220**	**100.0**

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Table 3; Association Between Selected Sexual Risk Factors and HPV Infection

Variables	High risk	Low risk	χ^2 value	p-values
Age at first intercourse			1.440	0.881
<16	16(66.7)	8(33.3)		
16-20	42(70.0)	18(30.0)		
21-25 26-30	16(76.2) 1(50.0)	5(23.8) 1(50.0)		
>30	4(66.7)	2(33.3)		
Total	**79(69.9)**	**34(30.1)**	0.898	
No. of lifetime sexual partner				
1	33(73.3)	12(26.7)		
2-4	37(68.5)	17(31.5)		
5-7	6(66.7)	3(33.3)		
8-10	1(50.0)	1(50.0)	0.391	0.898
>10	2(66.7)	1(50.0)		
Total	**79(69.9)**	**34(30.1)**		
Use of condom				
Always	5(62.5)	3.1		
Sometimes	29(70.7)	0.8		
Never	45(70.3)	13.4		
Total	**79(69.9)**	**34(30.1)**		
Husband having other women outside				
Yes	17(56.7)	13(43.3)		
No	7(63.3)	4(36.4)	0.355	0.551
I don’t know	55(76.4)	17(23.6)		
Total	79(69.9)	34(30.1)		
Having ever had STI				
yes	37(67.3)	18(32.7)		
No	42(72.4)	16(27.6)		
Total	**79(69.9)**	**34(30.1)**		

0.05 level of significance

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IV. DISCUSSION

The objective of this study was to showcase the common genotypes of HPV among HIV positive women in this region and demonstrate the determinants for HPV acquisition in them and then consequently relate the genotypes and factors to what is obtained in other regions of the world.

We found a total HPV prevalence of 119(54.1%) out of which high risk HPV (HrHPV) was 35.9% while low risk HPV (LrHPV) was 15%. In this study, the prevalence of HrHPV among HIV positive women is higher than the 13.4% reported among HIV positive women in Lagos [11] but this is significantly lower than that 64% reported in Kenya [12] and the 42.6% reported in Bamako Mali [13]. The total HPV prevalence is also lower than the total HPV prevalence of 87.8% reported among HIV positive women in Uganda [6].

The total HPV prevalence of 54.1% and the HrHPV prevalence of 35.9% recorded in our study aligns with existing literature across the world, suggesting that HIV-infected individuals have a high burden of HPV infections [9].

In this study, we found the predominant HrHPV to be the HPV35 (20.5%), HPV16 (14.2%), HPV45 (13.4%), HPV33 (9.4%) and HPV18 (4.7%), while the predominant LrHPV genotypes are; HPV6 (7.1%), HPV11 (4.7%), HPV72 (4.7%), HPV81 (3.9%)c, HPV42 (3.1%) and HPV43 (3.1%). This is slightly similar to a study conducted in Abuja, Nigeria, where the predominant HrHPV genotype among HIV positive women was HPV35 followed by HPV56 [14]. This slight similarity in HrHPV genotype predominance may be attributed to geographical proximity and shared cultural identities. In a similar study among HIV positive women in Lagos Nigeria, the predominant HrHPV genotypes were HPV16, HPV52, HPV31, HPV58 and HPV51 in decreasing order [11], whereas in our study HPV16 is the second predominant after HPV35. Regional and geographical variations coupled with socio-cultural life styles might have contributed to these differences.

Both our study and the Abuja study identify HPV35 as the most prevalent HrHPV. This contrasts with existing studies that show HPV16 as the most common HrHPV type among the global population. This suggests that HPV35 may have surpassed HPV16 as the most prevalent HrHPV type especially in Keffi and Abuja sub-regions. In a systemic review and meta-analysis among HIV positive women in kenya, the most predominant HrHPV genotype was HPV16 followed by HPV35 [12], this Kenyan study is in agreement with the global population studies that HPV16 is the most prevalent HrHPV, while t the second most prevalent in their study is HPV35 which is first place in our study.

In Kampala, Uganda, the most prevalent HrHPV genotype in decreasing order were HPV52, HPV51, HPV18, and HPV16 [6]. This is completely different from the genotype distribution in our study. The diverse picture of HPV seen in different areas may highlight regional and geographical variability in HPV genotype distribution as reported by Han S *et al* in a China study [15]. Compared to other studies conducted in distant parts of the world, genotype 53, 58, 31 and 16 were found to be predominant in North Eastern Brazil **[16]** , while 56, 53, 16, 58, 52, 33 were found to be predominant in New York USA [17]. There is thus great variability in the genotype distribution of high-risk HPV among HIV positive women across the globe.

The nonavalent HPV vaccine 'Gardasil-9' covers for the low risk genotypes 6, 11 and high risk types 16, 18, 31, 33, 45, 52 and 58 [18]. Though it covered nearly all of the genotypes detected in our current study, genotype 35 which is the most predominant in this study is not covered and this is a cause for serious concern being a risk factor for cervical cancer in HIV positive sub-population in this region. It then means that large population study in our region, Nigeria and the entire subtropical region is needed to advocate for wider polyvalent HPV vaccine that will include the predominant high-risk genotype in our sub-region.

Our study found no significant statistical association between HPV acquisition and family monthly income, number of lifetime sexual partners and condom use. This lack of association suggest that HPV infection is widespread across various socio-economic and behavioral profiles within the population of Women Living with HIV infections. It may also highlight the potential effect of HIV immunosuppression on patient's susceptibility to HPV infection and its persistence which potentially overshadow the effect of socioeconomic and behavioral risk factors.

Additionally, our study found no statistical association between HIV viral load and HPV acquisition and also no statistical association between CD4+ cell count and HPV acquisition. This is at variance with findings in a study conducted by Okunade *et al* in lagos [11] and by Ezechi *et* [19], where HPV infection was higher in women with lower CD4+ counts.

This study is limited by the fact that it was conducted among women enrolled on HAART and may not give the true picture of HPV infection in women living with HIV who are not on HAART.

V. CONCLUSION.

Our study highlights a different HrHPV genotype distribution among HIV positive women in our locality which is not covered by the polyvalent HPV vaccine. A wider population study is desirable to form the basis for advocacy for a wider polyvalent HPV vaccine that covers the predominant genotypes in our local region as the studied population is at higher risk of HrHPV persistence and thus increased risk of progression to precancerous lesions.

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CONFLICT OF INTEREST

We declare no potential conflicts of interest with respect to this research, its authorship or publication.

AUTHORS' CONTRIBUTION

Maimako Martins Yakubu and Peter Jonah designed the study while Maimako Martins Yakubu and Maishanu Sumaiya carried out the laboratory bench work (PCR and Sequencing). Maimako martins Yakubu , Titilayo Okpala-Obanewo, Oluwasesan Abdul and Bilqisu Goni collated the data. Maimako Martins Yakubu, Titilayo Okpala-Obanewo, Oluwasesan Abdul and Bilqisu Alkali Goni drafted the article while Peter Jonah and Maimako Martins Yakubu revised it for appropriate intellectual content.

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