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# Molecular Identification of Human Papillomavirus (HPV) in a Population of Women Living with HIV in Senegal

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**Abstract** **Original Research Article**

***Background:*** HPV infection is more frequent and more persistent in HIV+ women; this increases the risk of invasive cervical cancer that would progress about 10 years earlier in HIV+ women than in HIV-women. This data argues for accurate molecular epidemiology of genotypes and early detection of high-risk HPV infections for effective cervical cancer prevention. ***Objective:*** To genotype HPV with molecular biology technique and to evaluate the socio-demographic and clinical factors favoring HPV infection among women living with HIV (WLHIV). ***Materials and Methods:*** The molecular identification of 28 HPV genotypes (19 HPV-HR and 9 HPV-LR) was performed on endocervical samples using the Seegene AnyplexTM II HPV 28 Detection using the Bio CFX96 of Biorad. Data analysis was performed by the R studio software (version 3.6.0). Proportion comparisons were made by Pearson's Chi-square tests or Fisher's exact test. The result of a test was significant if P<5%. ***Results:*** The prevalence of HPV was 78.95% with 72.18% HPV-HR of which the most dominant genotype was HPV 56 (46.62%). Prevalence of HPV-LR was 57.14% with a predominance of HPV 42 (31.8%). In our group, 62.48% of women had multiple infections (at least two genotypes). Types 16 and 18 had a prevalence of 20.30% and 8.27% respectively. The rate of HPV infection is high in WLHIV with varied genotype representation. HPV infection was associated with low CD4 count and high viral load (P<0.05). HPV infection was not related to marital status, age, HIV type, treatment, contraception, abortion, nor the number of births (P>0.05). ***Conclusion:*** HIV+ women experienced high prevalence of HPV of various genotypes with HPV56, HPV16, HPV82, HPV51 and HPV33 predominant among high-risk genotype and HPV42 and HPV43 in low-risk genotype. Immunosuppression and elevated HIV viral load promotes HPV infection. Correct management of these high-risk population with special care to those who are infected by a genotype not included in the vaccines would provide better prevention of cervical cancers and a better prognosis in women co-infected with HIV and HPV.

**Keywords:** HIV, HPV, Genotyping, Prevalence, Multiplex PCR, Anyplex II HPV28.



## INTRODUCTION

Papillomavirus (HPV) infections are among the most common human viral infections worldwide [1]. About 80% of sexually active people will be infected at some point in their lives but their transmission is not exclusively sexual [2, 3]. Given the similar patterns of transmission, several types of HPV can be simultaneously or successively inoculated to the same individual; Co-infections are therefore frequent [4]. HPV has a very high genetic diversity with a host specificity [5]. They are indeed associated with different types of cancer, including cancer of the cervix. Estimates point to approximately 569,847 new cases of cervical cancer diagnosed each year. Making it the 3rd leading cause of cancer among women worldwide [6]. In Senegal, approximately 1876 new cervical cancer cases are diagnosed each year (estimates for 2018). Cervical cancer is the leading cause of cancer among women in Senegal [7]. More than 200 genotypes are known including 120 sequenced [8]. The HPVs most commonly associated with cervical cancers are called high-risk HPV (HR-HPV) (HPV16, 18, 31), while the least common are low-risk HPV (LR-HPV) (HPV 6, 11) [9]. A study of more than one million women worldwide shows a global prevalence of HPV of about 12% of which women in sub-Saharan Africa have shown the highest prevalence (24%) in front of women

from Eastern Europe (21.4%) or Latin America (16.1%) [10]. In Africa, Guinea (48%) and Mozambique (41%) were reported to have the highest prevalence [3]. However, in women living with HIV (WLHIV) it has been found that the prevalence of HPV is high. HPV infection rate in WLHIV to HIV-negative women was 1.47 in France [11] and 2.88 in Senegal [12, 13]. Various studies show links between HIV infection, HPV infection and cervical cancer. WLHIV are more likely to be multi-infected and contract significantly more HPV-HR; due to immune depression [14]. HIV + men and women would have a higher prevalence of HPV-HR and HPV-LR types than men and women that are HIV- [15-19]. In addition, HPV infection is more persistent among WLHIV this increases the risk of cervical cell abnormalities and invasive cervical cancer [20]. Cervical cancer is progressing about 10 years earlier in WLHIV than in HIV- women [21]. This data argues for accurate molecular epidemiology of genotypes and early detection of high-risk HPV infections for effective cervical cancer prevention.

HPV genome is a circular double stranded DNA, with one coding strand and consists of approximately 800 base pairs [22]. The classification of HPV is based on their genomic sequence their tropism and their oncogenic power [23]. L1 is the most conserved gene and has been used to identify and classify papillomavirus (PV) genotype for the last 15 years [24].

Of the HPVs that have been cataloged, 40 types have a mucosal tropism to the aerodigestive tract and the lower part of the genital tract. Other types with genital tropism such as 6, 11, 42, 44 and other rare types are considered non-oncogenic and usually cause subclinical and clinical lesions in the form of condyloma acuminata [25]. HPV genotyping is an important test that can contribute to the prevention of invasive cervical cancer [26]. Techniques based essentially on the detection of the viral genome by molecular biology have been put in place since these viruses are difficult to cultivate routinely. These techniques target L1-E1 regions or the whole genomes of the viruses [27]. The most common molecular diagnostic methods are based on reverse hybridization of microarrays and multiplexed real-time quantitative PCR [28, 29]. The objective of our study was to identify genotype and evaluate the prevalence of HPV in a population of women living with HIV followed by the AIDS program of the armed forces at the Ouakam Military Hospital in Dakar.

## MATERIALS AND METHODS

### Type and Population of the Study

This transversal, descriptive and analytical study was performed at the Molecular Biology Laboratory of the AIDS Program of the Senegal Armed Forces in Ouakam Military Hospital (HMO) Dakar, Senegal from 5 June 2018 to 16 May 2019.

The target population was 133 women living with HIV followed at the Ouakam Military Hospital (HMO) whose consent was obtained by the treating physician after a detailed explanation of the study. As part of the prevention checkups women perform Pap smear and HPV screening to identify those at risk for developing cervical cancer. Risk evaluation is based on HPV-HR genotype in cervical samples. For each woman a survey sheet collecting information on identity, socio-demographic parameters such as age, marital status, abortion, age of first sexual intercourse, the number of sexual partners, contraception has been filled.

### Sample Collection

A total of 133 endo-cervical samples were collected with a cytobrush by swabbing endocervix cells. The cytobrush is then introduced into Abbott multi-collect specimen collection kit (Abbott GmbH & Co. KG Max Plank-Ring, Germany) and the sample is stored at -80° C prior to testing.

### DNA Extraction and Nanodrop DNA Assay

DNA extraction was done using the ZR Viral DNA KIT kit from ZYMO RESEARCH, USA www.Zymoresearch.com according to the manufacturer's protocol from 200µl of endocervical sample. The viral DNA extract was analyzed with Nano Drop LITE to evaluate its quality and quantity and subsequently stored at -20° C until PCR testing.

### Molecular analysis with Seegene Anyplex II HPV28

The molecular analysis was performed by multiplex PCR with the Seegene Anyplex II HPV28 Kit on the CFX96 real-time BIORAD RT-PCR machine according to the manufacturer's protocol. The Anyplex II HPV28 method can detect in vitro 28 HPV genotypes in cytological fluids or cervical samples. This technique allows the detection and individual identification of 19 high-risk HPV (16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 69, 73, 82) and 9 low risk HPV (6, 11, 40, 42, 43,44, 54, 61, 70), using DPO technologies. (Dual Priming Oligonucleotides) and TOCETM (Tagging Oligonucleotide Cleavage and Extension) [30, 31].

## STATISTICAL ANALYSIS

Patient information collected from the questionnaires was recorded in an Excel database. Data analysis was performed by the R studio software (version 3.6.0). Qualitative variables were described by absolute and relative frequency calculations. Proportion comparisons were made by Pearson's Chi-square tests or Fisher's exact test. The result of a test was significant if the P <5%.

# RESULTS

**Prevalence and characterization of HPV in the general population**

Of 133 patients, 78.95% (105/133) hosted HPV. HPV-HR and HPV-LR had respective prevalence of 72.18% and 57.14%. The multi-infection was present in 68.42% of the women i.e. hosting at least two genotypes of HPV.

**Prevalence of HPV by age**

The average median age in our study population was 42.5 years old with extremes of 23 to 68 years old. To study the influence of age on HPV carriage our cohort was classified into three groups. HPV prevalences were 77.42%, 75% and 88.24% respectively in the age groups <35 years, 35 to 49 years and ≥ 50 years (p=0.29) (Table-2). HPV 16 and 18 are present in all age groups with a prevalence of respectively 19.35% and 6.45% in women with an age <35 years, 17.65% and 6.45% among women aged 35 to 49, 26.47% and 2.14% among women ≥ 50 years of age.

**Prevalence of HPV According to Marital Status**

According to marital status HPVs had a higher prevalence among single women 83.93% than among married women75% (p = 0.40 Table-2). The infection rate of HPV was high in both groups.

**Prevalence of HPV according to abortion**

HPV carriage of 78.26% and 79.37% was detected in women without abortion and in women with abortion respectively. Abortion is not thought to be associated to HPV infection rates among women living with HIV (p = 0.86) (Table-2).

**Influence of CD4/mm$^3$ levels on HPV infection**

To see the impact of immunity on HPV infection we divided our cohort into two CD4 patient groups; <350/mm$^3$ (N=49) and CD4 ≥350 /mm$^3$ (N=84). The prevalence of HPV was significantly higher in patients with a CD4 count <350 then in patients with CD4≥350/mm$^3$ 93.88% vs.70.24% (p=0.0026; Table-2). Immunosuppression promotes HPV carriage in HIV+ women.

**Influence of HIV viral load on HPV carriage**

The influence of HIV viral load on HPV carriage was studied by dividing our cohort into two groups of patients with viral load greater than and less than 1,000 copies / ml. The rate of HPV infection is significantly higher in patients with higher viral load 84.85% vs. 77% (P <0.0001 Table-2). Higher viral load was associated with HPV carriage in HIV+ women. The type of virus (HIV-1 vs. HIV-2) had no influence on HPV infection (p=0.23 Table-2).

**Influence of HIV treatment on the prevalence of HPV**

Of the patients under antiretroviral therapy (ART) 76.24% carry HPV versus 87.50% for patient not taking ARV (P= 0.17, Table-2).

**Influence of contraception on HPV carriage**

79.81% of the patients who never used contraception and 73.07% of the patients on contraception harbored HPV (P = 0.50 Table-2) In HIV + women contraception does not influence the level of HPV infection.

**Molecular identification of HPV genotypes**

In our study population the HPV-HR and HPV-LR frequencies were 72.18% and 57.14% respectively (Table-1). Our method of study allows to detect 28 genotypes listed in Table-1 We have described HPV the most representative. The genotypes HPV56, HPV42 and HPV16 were the main virus identified with respective prevalence of 46.62%, 31.58% and 20.30%. Genotypes HPV24 (18.05%), HPV51 (15.79%), HPV43 (15.79%) and HPV53 (15.04) were also found. Due to their highly oncogenic HPV16 and 18 we were interested and had a prevalence of 20.30% and 8.27% respectively (Table-1).

**Table-1: HPV genotype and prevalence in the overall population**

| HPV Genotypes | N | Percentage (%) |
|---|---|---|
| **Number of patients** | 133 | |
| **HPV prevalence** | 105 | **78.95** |
| **Number of multi-infected** | 91 | **68.42** |
| **HPV-HR prevalence** | 96 | **72.18** |
| **HPV-LR prevalence** | 76 | 57.14 |
| **HPV6** | 15 | 11.28 |
| **HPV11** | 0 | 0 |
| **HPV40** | 4 | 3.01 |
| **HPV42** | 42 | **31.58** |
| **HPV43** | 21 | 15.79 |
| **HPV44** | 11 | 8.27 |
| **HPV54** | 15 | 11.28 |
| **HPV61** | 6 | 4.51 |
| **HPV70** | 13 | 9.77 |
| **HPV16** | 27 | **20.30** |
| **HPV18** | 11 | **8.27** |
| **HPV26** | 0 | 0.00 |
| **HPV31** | 11 | 8.27 |
| **HPV33** | 19 | 14.29 |
| **HPV35** | 8 | 6.02 |
| **HPV39** | 9 | 6.77 |
| **HPV45** | 5 | 3.76 |
| **HPV51** | 21 | 15.79 |
| **HPV52** | 12 | 9.02 |
| **HPV53** | 20 | 15.04 |
| **HPV56** | 62 | **46.62** |
| **HPV58** | 17 | 12.78 |
| **HPV59** | 2 | 1.50 |
| **HPV66** | 9 | 6.77 |
| **HPV68** | 15 | 11.28 |
| **HPV69** | 0 | 0.00 |
| **HPV73** | 13 | 9.77 |
| **HPV82** | 24 | 18.05 |

**Table-2: Relationship between HPV infection and variables**

| Socio-demographic and patient clinics parameters | | Positives | Percentage (%) | P-value |
|---|---|---|---|---|
| **Age (year)** | <35 (31) | 24 | 77.42 | 0.29 |
| | 35-49 (68) | 51 | 75.00 | |
| | ≤50 (34) | 30 | 88.24 | |
| **Marital status** | Maried (76) | 57 | 75.00 | 0.4 |
| | Single (56) | 47 | 83.93 | |
| | Missing data (1) | 1 | | |
| **Abortion** | No abortion (69) | 54 | 78.26 | 0.86 |
| | abortion (63) | 50 | 79.37 | |
| | Missing data (1) | 1 | | |
| **CD4** | < 350 (49) | 46 | 93.88 | 0.0026 |
| | ≥ 350 (84) | 59 | 70.24 | |
| **HIV Viral load** | < 1000 (100) | 77 | 77.00 | < 0.001 |
| | ≥ 1000 (33) | 28 | 84.85 | |
| **VIH type** | VIH1 (150) | 118 | 78.67 | 0.23 |
| | VIH2 (12) | 10 | 83.33 | |
| | VIH 1+2 (3) | 3 | | |
| **Treatment** | treatment (101) | 77 | 76.24 | 0.17 |
| | No ART treatment (32) | 28 | 87.50 | |
| **Contraception** | contraception (26) | 19 | 73.08 | 0.50 |
| | No contraception (104) | 83 | 79.81 | |
| | Missing data (3) | 3 | | |

## DISCUSSION

Women living with HIV are known to be at high risk of developing cervical cancer due to high levels of HPV and their persistence due to immunosuppression. Women living with HIV can carry several HPV genotypes with multi-infections [32].

In our study we found a high HPV prevalence (78.95%) in our study population of women living with HIV. This prevalence is higher than the 59.6% described in Burkina Faso [33] and in Brazil ranging from 48% to 68% [34, 35]; similar to France 73.3% [11] and Brazil 73.5% [36] in the USA, HPV prevalence ranges from 54% to 73% [37-38] and in Senegal prevalence has been previously measured at 78.2% [12].

High-risk HPV genotypes were detected in 72.18% of our female patients. Similar results have been reported in South Africa with HPV-HR of 75% [26] and above the 57.5% described in Brazil [36]. In addition, our results showed that 68.42% of our patients were multi-infected i.e. hosting at least two genotypes of HPV. This multi-infection rate is similar to the 62.3% described by Hanisch et al (2013) in Senegal [12]. Genotyping of HPV in our study revealed that HPV56 was dominant at 46.62% (62/133) among detected HPV. This predominance of HPV56 was also described in the Bahamas [39]. A high prevalence of this genotype has been described in Brazil [40-41-36] but also in India [32-42]. Genotypes HPV42, HPV82, HPV16, HPV51, HPV43, HPV53, HPV33, HPV58 follow with prevalence ranging from 31.58% to 12.78%. Similar results on the prevalence of these genotypes in HIV + women have been described in Senegal, in France and South Africa [12, 11, 13, 19].

HPV16 and HPV18 deserve special attention because they are involved in 70% of cervical cancers worldwide [3]. HPV16 and 18 were present respectively in 20.30% and 8.27% of our female patients. In our cohort, HPV16 rate is higher than the 15% prevalence previously measured in South Africa [43] To 4.7% of Burkina Faso [44] and 13.1% in Senegal [12]. This difference in rate can be explained by the high sensitivity and specificity of our genotyping method compared to other techniques, it detects 28 genotypes of HPV which offers a broad-spectrum performance [45-46-47]. For HPV18, our result is similar to the 8.5% prevalence previously described in South Africa [43].

Our results increase the knowledge of the molecular epidemiology of HPV circulating in Senegal. This data emphasis the importance of cervical exams in the fight against cervical cancer with special attention to women living with HIV.

Factors such as immunity and viral load of patients and their association with HPV carriage in women living with HIV were assessed. Our results showed an association between CD4 count and HPV prevalence among women living with HIV. Those with CD4<350/ml had a higher carriage rate than those with CD4>350/ml (p=0.0026).

This result has also been observed in Southern Brazil and South Africa [14-35-43]. This result can be explained by the hypothesis that HPV16 escapes immunological surveillance of the host while other

HPVs are often well controlled by the immune response [48-49]. In case of immunodepression the control of non-HPV16 by the immune system is impaired, favoring the increase of this genotypes [50] and also the reactivation of other latent HPV [2]. Our results showed a correlation between the HIV viral load and HPV infection in patients. Patients with high viral loads (>1,000 copies per ml) were more infected than those with low viral loads (<1,000 copies per ml) ($p<0.0001$). This could be explained by the correlation of the immunological status and the viral load [50].

The socio-demographic factors of the patients and their relationship with HPV carriage in women living with HIV were evaluated. Our results showed that there was no association between HPV infection and age, marital status, gestation, the type of HIV or the number of abortions, ART treatment and contraception of patients. This lack of association between HPV infection and several socio-demographic criteria is explained by the dominant effect of HIV. HIV increases the susceptibility of patients to HPV promotes the reactivation of latent HPV and the persistence of infection due to immunosuppression. In this situation all the other socio-demographic criteria become secondary.

## CONCLUSION

HIV+ women experienced high prevalence of HPV of various genotypes with HPV56, HPV16, HPV82, HPV51 and HPV33 predominant among High-risk HPV and HPV42 and HPV43 in low-risk HPV. Immunosuppression and elevated HIV viral load promote HPV infection. Correct management of these high-risk population and correct identification of HPV genotype, especially those not included in the vaccines would provide good prevention of cervical cancers and a better prognosis in women co-infected with HIV/HPV.

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