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Prevalence of High-risk Human Papillomaviruses and Associated Factors in Women Living with HIV and Followed at the Outpatient Treatment Center in Brazzaville, Congo

Maryse N. Akolbout-Manguilla[1,2,*], Edith-Sophie Kombo Bayonne[1], Norcely Makela[2], Valchy Miegakanda[3], Laure Ghoma Linguissi[4], Rocide Moukouba[2], Merlin Isidore Diafouka[2], Jean Félix Peko[1,5]

[1]Faculty of Health Sciences, Marien Ngouabi University, Congo
[2]Outpatient Treatment Center, Brazzaville, Congo
[3]National Public Health Laboratory, Brazzaville, Congo
[4]National Institute for Health Sciences Research, Brazzaville, Congo
[5]University Hospital Center, Brazzaville, Congo

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(b): *Maryse N. Akolbout-Manguilla, Edith-Sophie Kombo Bayonne, Norcely Makela, Valchy Miegakanda, Laure Ghoma Linguissi, Rocide Moukouba, Merlin Isidore Diafouka, Jean Félix Peko (2023). Prevalence of High-risk Human Papillomaviruses and Associated Factors in Women Living with HIV and Followed at the Outpatient Treatment Center in Brazzaville, Congo. Universal Journal of Public Health, 11(6), 961 - 969. DOI: 10.13189/ujph.2023.110621.*

Abstract Genital Human Papillomavirus infection and HIV infection are both sexually transmitted infections. High-risk genotypes (HR-HPV) predispose patients to cancer development. This study aims to determine the prevalence of HR-HPV in cervix specimens and to identify factors associated with the presence of HR-HPV in women living with HIV (WLHIV). This was a cross-sectional study conducted from April 2021 to March 2022 at the Outpatient Treatment Center, and at the National Public Health Laboratory in Brazzaville. WLHIV aged over 17 years were included. Socio-demographic data, history, clinical data, and biological data were collected. The identification of HR-HPV genotypes was performed from endocervix samples by the real-time PCR technique. Statistical analyses were carried out using SPSS Statistics software. A total of 276 WLHIV were enrolled. The mean age was 44.6 (SD 8.9) years. The prevalence of HR-HPV was 44.2%. In multivariate analysis, the level of primary education (p=0.034; CI 95%; ORa=2.62 [1.07–6.39]) was identified as a factor associated with HPV/HIV co-infection, and a CD4 count greater than or equal to 200 cells/mm^3 as a protective factor against HR-HPV infection (p=0.04; CI 95%; ORa=0.28 [0.07-0.87]). This study reveals a high prevalence of HR-HPV among WLHIV. It shows a significant association between the low level of education and genital infection by HR-HPV, as well as the protective role of the CD4 count greater than 200/mm^3, in this population group. This suggests the establishment of a systematic screening of HPV-HR in women affected by HIV and the strengthening of preventive measures for sexually transmitted infections. The means of communication used for prevention must be adapted to the target populations' level of education.

Keywords Prevalence, High-risk Human Papillomavirus, HIV, Sexually Transmitted Diseases,

Realtime PCR, Congo

1. Introduction

Human papillomaviruses (HPVs) are DNA viruses specific to humans. They belong to the *Papillomaviridae* family. They have a tropism for skin and mucous membranes' epithelial cells, and they are cosmopolitan viruses. More than 200 genotypes have already been identified [1]. Each genotype has its preferred sites [1]. In most infected people, HPV is eliminated via the immune system [2]. In some cases, the virus may persist and cause symptomatic or subclinical skin or mucosal lesions [3]. The lesions can be benign. The virus can also cause changes in epithelial cells that predispose patients to the development of cancers [4]. HPV genotypes that cause precancerous and cancerous epithelial lesions are known as "high-risk HPV" (HR-HPV) or "oncogenic HPV". They have tropism for the cervix, vagina, vulva, penis, anus, oropharynx, and larynx [5]. Fourteen types of oncogenic HPV have been identified: genotypes 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, and 68 [5-7]. The World Health Organization (WHO) reports that 99% of cervical cancers are linked to infection with high-risk HPV which is a sexually transmitted infection [8].

The immune response to HPV infection is a cell-mediated response. Thus, a decrease in cellular immunity can cause HPV to persist [5,9]. Conditions responsible for decreasing cellular immunity, such as HIV infection, may increase the risk of HPV infection. HIV-infected women have a higher prevalence of HPV-HR infection and a higher risk of progression to cancer [9,10]. Studies on the prevalence of HPV-HR show great diversity across global regions, countries, and HIV status, ranging from 1.9% to 64% [11,12].

In Congo, the prevalence of HR-HPV among persons living with HIV and the factors contributing to infection with these viruses are unknown. To provide preventive interventions for genital cancers in HIV-positive women, we conducted a study whose objective was to determine the prevalence of HR-HPV in cervical specimens and to identify factors associated with the presence of HR-HPV in women living with HIV (WLHIV).

2. Materials and Methods

2.1. Study Design

This was a cross-sectional study conducted over a 12-month period from April 2021 to March 2022.

The study was conducted at the Outpatient Treatment Center (OTC) and at the National Public Health Laboratory, Brazzaville. The Outpatient Treatment Center is a health facility specializing in the prevention and treatment of HIV infection. It is located within the University Hospital Center of Brazzaville. It provides daytime hospital services as part of the overall management of HIV infection. The National Public Health Laboratory is the reference laboratory for molecular analyses in Congo.

2.2. Study Population

The study population comprised HIV-positive (HIV+) women treated at the Outpatient Treatment Center in Brazzaville. The study's inclusion criteria were as follows: confirmed HIV+ status, age over 17 years, and having arrived at OTC for the supply of antiretroviral drugs, for a periodic follow-up visit, or for a health problem. Women who met these criteria and who signed an informed consent form were included. Non-inclusion criteria were as follows: menstruating women and those with a history of total hysterectomy.

2.3. Data Collection

The data were collected on a standardized survey with two parts: one part for socio-demographic data, history, and clinical data and one part for biological data. Before patients filled out the questionnaire, the objectives of the study, the terms contained in the document, and the sampling procedures were explained to the women surveyed. The survey was filled out at the OTC.

Socio-demographic data consisted of the women's age, level of education, and marital status. Historical data comprised the characteristics of sexual activity, age of first sexual intercourse, method of contraception, number of sexual partners, condom use, prostitution, and the history of HIV (duration of HIV, antiretroviral treatment protocol, and duration of antiretroviral treatment). Biological data were CD4 count, plasma HIV-1 viral load, and the result of HPV-HR testing in the cervix sample.

Interview data were collected by questioning the women. Clinical data were extracted from patients' medical records. The biological data were collected after blood and cervical samples were taken by health personnel trained for this purpose.

2.4. Sample Collection

The CD4 count and the plasma viral load of HIV-1 were carried out simultaneously by the OTC laboratory team and made available to the investigators.

For the detection of HPV, the endocervical sample was taken from the woman lying in the gynecological position on an examination table. The vaginal walls were spread using a sterile speculum. The sample was taken using a sterile single-use Cervexbrush from the Abbot Cervi-Collect specimen collection kit® by applying the cytobrush to the endocervical wall and making three complete, gentle turns in the same direction. The specimen was then

suspended in a collection vial containing 2.5 ml of transport buffer from the Cervi-Collect Abbott® specimen collection kit, which was composed of guanidine thiocyanate and a chaotropic salt. The samples collected were conditioned at -20˚C and then transported to the National Public Health Laboratory in an insulated cooler containing dry ice to comply with good laboratory practices (GLPs) for the transport of biological samples. Cervical cell samples were stored in Abbot Cervi-Collect specimen liquid medium until the day of analysis.

2.5. Molecular Analysis

Detection was based on the identification of HR-HPV genotypes. The real-time PCR technique using the m2000 System Abbot real-time technology from the ABBOT Realtime High-Risk HPV kit (Abbott Realtime High Risk HPV Assay, m2000sp, m2000rt, m24sp) was used [13]. This is a qualitative test for detecting HR-HPV DNA corresponding to one of the following genotypes: 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, and 68.

2.6. Statistical Analysis

Statistical analyses were carried out using SPSS Statistics software from IBM, version 25. The continuous variables were expressed as the mean with standard deviation (SD and the minimum and maximum values). Categorical variables were expressed as numbers and percentages. To identify the factors associated with the presence of HR-HPV, a bivariate analysis was performed as the first step. Pearson's Chi-square test (including Yates's correction for small samples) and Student's test were used to analyze association. The variables used to evaluate associated factors with the presence of HR-HPV DNA (dependent variable) were socio-demographic, behavioral, and biological (independent variables). The strength of the association was determined by calculating the odds ratio (OR), with the confidence interval (CI) defined at 95%. Variables with a *p-value* of <0.05 were considered statistically significant and were included in the multivariate logistic regression models. For the purposes of parsimony, variables whose p-value was less than 0.05 were inserted into the logistic regression model. To ensure that the likelihood has not been altered, the successive models were constituted using the ascending step-by-step method with the progressive introduction of blocks of variables. The strength of the association between each independent variable and the dependent variable was measured using the adjusted odds ratio (ORa).

2.7. Ethical Considerations

The present study received the approval of the Ethics Committee for Research in Health Sciences in Congo (N°228 /MRSIT/IRSSA/CERSA- September 20, 2019). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki for medical research involving human subjects. All women signed a written consent prior to enrolment.

3. Results

3.1. Characteristics of the Study Population

A total of 276 women living with HIV were enrolled. The mean age of the women was 44.6 (SD 8.9) [19-71] years. The distribution of enrolled WLHIV according to socio-demographic characteristics is shown in Table 1. The mean time to HIV testing was 11.4 (SD 4.8) [1-22] years. All women were on antiretroviral therapy. The mean duration of treatment was 10.7 (SD 4.6) [0,6-21] years. Table 2 presents the distribution of study population according to the history of HIV infection.

Table 1. Distribution of 276 women living with HIV by socio-demographic characteristics

Variables	Number (n)	Percentage (%)
Age range (years)		
<20	1	0.4
20-29	17	6.2
30-39	49	17.8
40-49	127	46
50-59	72	26.1
>60	10	3.6
Marital status		
Single	155	56.2
Divorcee	1	0.4
Bride	14	5.1
Cohabitation	94	34.1
Widow	12	4.3
Level of education		
None	5	1.8
Primary	40	14.5
Secondary	182	65.9
Academic	49	17.8

Table 2. Distribution of 276 women living with HIV by history of HIV infection

Variables	Number (n)	Percentage (%)
Duration of antiretroviral therapy (years)		
<11	138	50
≥11	138	50
Type of HIV		
HIV-1	276	100.0
Treatment line		
First line	259	93.8
Second and third line	17	6.2
Duration of HIV (years)		
≤10	123	44.6
>10	153	55.4

3.2. Prevalence of HR-HPV in Cervix Specimens

Of the 276 cervix specimens screened by PCR for the detection of HR-HPV DNA, 122 were positive. The prevalence of HR-HPV was 44.2% (CI 95% 38.3-50.3). The distribution of HR-HPV positive cases according to age is presented in Figure 1.

3.3. Factors Associated with the Presence of HR-HPV in Cervix Specimens

The mean age at first sexual intercourse was 16.25 (SD 2.21) [10-26] years. The average frequency of monthly sexual intercourse was 1.7 (SD 3.2) [0-31]. None of the women declared practicing prostitution or using tobacco. Of the 276 included women, 268 (97.1%) were tested for CD4 and 273 (98.9%) for viral load. The mean CD4 value was 623.7 (SD 286.7) [11-1623] cells/mm^3.

In bivariate analysis, the level of primary education (p=0.042; CI 95%; OR=2.42 [1.03–5.69]) and the detectable viral load (p=0.047; CI 95%; OR=1.96 [1.002–3.86]) were identified as factors associated with HPV/HIV co-infection, and a CD4 count greater than or equal to 200 cells/mm^3 was a protective factor. Table 3 presents the bivariate analysis of socio-demographic and behavioral factors. Table 4 presents the bivariate analysis of the immuno-virological factors.

Multivariate analysis using the logistic regression method confirmed the statistically significant link between the level of primary education and the presence of HPV-HR in cervical samples (p=0.034; CI 95%; ORa=2.62 [1.07–6.39]); thus, a CD4 count greater than or equal to 200 cells/mm 3 is a protective factor against infection (p=0.04; CI 95%; ORa=0.28 [0.07-0.87]). Table 5 reports the results of the multivariate analysis.

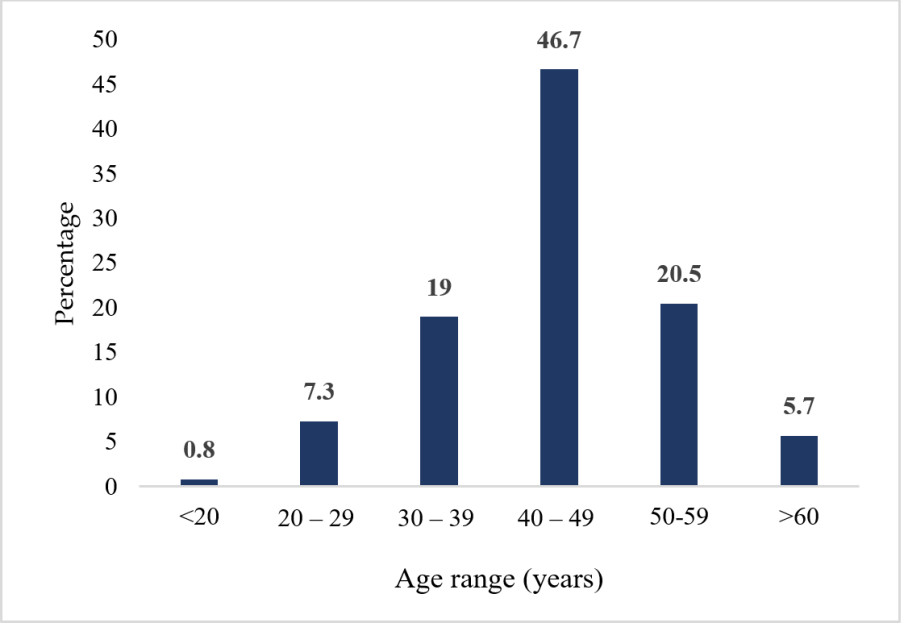

Figure 1. Distribution of 122 women living with HIV detected positive for HR-HPV according to age group

Table 3. Bivariate analysis of sociodemographic and behavioral factors in 276 women living with HIV

Variables	HR-HPV+ (n=122)	HR-HPV- (n=154)	OR [95% CI]	*p*
Mean age (years)	43.8 (10.0)	45.2 (8.0)	0.98[0.95;1.01]	0.22
Marital status				
Bachelor	73 (47.1)	82 (52.9)	1	0.466
Cohabitation	37 (39.4)	57 (60.6)	0.73 [0.43 – 1.23]	0.233
Bride	5 (35.7)	9 (64.3)	0.62 [0.20 – 1.95]	0.413
Divorcee	0 (0.0)	1 (100.0)	-	1.000
Widow	7 (58.3)	5 (41.7)	1.57 [0.48 – 5.17]	0.453
Level of education				
None	1 (20.0)	4 (80.0)	0.36 [0.04 – 3.49]	0.634
Primary	25 (62.5)	15 (37.5)	2.42 [1.03 – 5.69]	0.042*
Secondary	76 (41.8)	106 (58.2)	1.04 [0.55 – 1.97]	0.905
Academic	20 (40.8)	29 (59.2)	1	0.064
Age of first sexual intercourse (years)				
<13	2 (28.6)	5 (71.4)	0.60 [0.05 – 6.79]	1.000
13 – 16	62 (42.5)	84 (57.5)	1,11 [0.18 – 6.83]	1.000
17 – 21	56 (47.5)	62 (52.5)	1,35 [0.40 – 3.53]	1.000
>21	2 (40.0)	3 (60.0)	1	0.714
Frequency of sexual intercourse				
0	59 (41.0)	85 (59.0)	1	0.221
1	23 (41.1)	33 (58.9)	1.00 [0.54 – 1.88]	0.990
≥2	40 (52.6)	36 (47.4)	1.60 [0.91 – 2.80]	0.098
Number of sexual partners				
0	59 (41.5)	83 (58.5)	1	0.610
1	58 (47.5)	64 (52.5)	1.27 [0.78 – 2.08]	0.328
≥2	5 (41.7)	7 (58.3)	1.01 [0.30 – 3.32]	0.994
History of STIs				
Yes	5 (62.5)	3 (37.5)	2.15 [0.50 – 9.18]	0.308
No	117 (43.7)	151 (56.3)		
Condom use				
Yes	23 (48.9)	24 (51.1)	1.26 [0.67 – 2.36]	0.473
No	99 (43.2)	130 (56.8)		
Contraceptives				
Yes	1 (50,0)	1 (50,0)	1,26 [0.08 – 20.42]	1.000
No	121 (44.2)	153 (55.8)		

STI = Sexually Transmitted Infection

Table 4. Bivariate analysis of immunovirological factors in 276 women living with HIV

Variables	HPV+	HPV-	OR [95% CI]	*p*
CD4 (per mm³) n=268				
<200	11 (9.1)	4 (2.7)	1	
≥200	110 (90.9)	143 (97.3)	0.28 [0.70 – 0.84]	0.003*
Viral load n = 273				
Detectable	22 (19.8)	17 (11.2)	1.96 [1.002 – 3.86]	0.047*
Undetectable	97 (80.2)	135 (88.8)		

Table 5. Multivariate analysis of factors associated with the presence of HPV-HR in women living with HIV

Variables	HR-HPV+ (n=122)	HR-HPV- (n=154)	OR [95% CI]	*p*	ORa [95% CI]	*p*
CD4						
<200	11 (9.1)	4 (2.7)	1			
≥200	110 (90.9)	143 (97.3)	0.28 [0.70 – 0.84]	0.003	0.28 [0.07-0.87]	0.04*
Viral burden						
Detectable	24 (58.5)	17 (41.5)	1.96 [1.002 – 3.86]	0.047	1.80 [0.86 – 3.76]	0.121
Undetectable	97 (41.8)	135 (58.2)				
Level of education						
None	1 (20.0)	4 (80.0)	0.36 [0.04 – 3.49]	0.634	0.29 [0.03 – 2.86]	0.287
Primary	25 (62.5)	15 (37.5)	2.42 [1.03 – 5.69]	0.042	2.62 [1.07 – 6.39]	0.034*
Secondary	76 (41.8)	106 (58.2)	1.04 [0.55 – 1.97]	0.905	1.05 [0.53 – 2.08]	0.879
Academic	20 (40.8)	29 (59.2)	1	0.064		

4. Discussion

Our study focuses on the prevalence of high-risk human papillomaviruses and associated factors in women living with HIV at an HIV infection care center in Brazzaville, Congo.

4.1. Prevalence of HR-HPV

The study reveals a prevalence of HPV-HR of 44.2% in cervix samples, i.e., nearly half of the enrolled women. The most infected age group was between 40 and 49 years, representing 46.7% of the women who were detected positive for HPV-HR. Two HR-HPV prevalence studies were carried out among the general population in other departments in Congo. Nganga et al. revealed prevalences of 37.5% and 39.24% in the departments of Niari and Bouenza, respectively [14]. The study conducted by Boumba et al. estimated the prevalence at 44.1% in the Pointe-Noire department [15].

In Cameroon, another Central African country, Sosso et al. reported a prevalence of HR-HPV close to ours, estimated at 43.47% among FVVIH [16]. The proximity of Cameroon, bordering the Congo, suggests a similarity in sexual behaviors with respect to the risk of transmission of STIs, including HPV. In West Africa, the prevalence of HR-HPV is 35.9% in Nigeria and 59.1% in Burkina Faso [17,18]. In Southern Africa, prevalences of 79.1% and 72.9% were published for South Africa and Botswana, respectively [18,19]. In East Africa, prevalences of 24.9% and 35.2%, were reported for Zimbabwe and Ethiopia, respectively [20,21]. In North Africa, a study by Ouladlahsen et al. estimated the prevalence in Morocco at 74.5% [22]. A study conducted among seropositive women of African origin residing in Belgium reported a prevalence of 42.8% [23]. These observations show a great diversity in the prevalence of HR-HPV among persons living with HIV depending on the country and the sub-region of the African continent.

In Europe, particularly in Denmark, the authors reported a prevalence of 43.7% [24]. A review of the literature on work conducted in Latin America and the Caribbean among PLHIV showed a weighted prevalence of HR-HPV of 51% [25]. A meta-analysis of studies conducted in Asia showed a prevalence of HR-HPV infection of 34.6%, with extremes of 30.3 and 39.1% [26].

These observations testify to the circulation of HPV, particularly of genotypes at risk of causing precancerous and cancerous lesions. These findings corroborate data from the literature which reports a significant circulation of

HPV among people living with HIV in Africa [27]. The genital localization of HPV-HR infection, compatible with sexual transmission, suggests taking measures to curb the spread of sexually transmitted microorganisms. These measures also prevent HIV, with which HPV shares the same route of sexual transmission. Additionally, these measures help advance global strategy of the World Health Organization, which plans to eliminate cervical cancer as a public health problem by 2030, through prophylactic and therapeutic actions, particularly in women living with HIV [28].

4.2. Factors Associated with the Presence of HR-HPV

Regarding the associated factors, our study reveals a statistically significant association between the level of primary education of women living with HIV and the presence of HR-HPV in cervix samples (p=0.034; ORa=2.62 [95% CI 1.07 – 6.39]). This observation suggests that women with a low level of education could encounter difficulties accessing and understanding the information provided on sexually transmitted infections. These weaknesses could also concern the application of preventive methods, particularly the use of condoms. Our observations show low condom use (17%) by WLHIV. This constitutes a more effective means of prevention to fight against HIV than against HPV, because the surface contaminated by HPV is larger and extends beyond the area protected by the condom.

Other socio-behavioral factors associated with the detection of HR-HPV in FVVIH have been identified by various authors. For example, Masdoua et al. [29] in Algeria reported an association between the polygamy of the partner and the use of oral contraceptives. The women in our study population reported not using oral contraception. Additionally, we did not ask about the sexual behavior of their spouse. Megersa et al. [21] in Ethiopia identified the multiplicity of partners in WLHIV, as well as the history of STIs. Multiple partners and sexual intercourse with occasional partners are known risk factors for STIs [30]. Our study reveals a low frequency of sexual intercourse among women living with HIV followed at OTC. Due to the lack of control of the spouse's sexual behavior and the difficulty of adopting physical protective measures such as condoms, vaccination against HPV appears to be a useful means of strengthening the prevention of HPV-induced cervical cancer of the uterus. Abel et al. [31] reported a significant association between alcohol consumption and HR-HPV infection. None of the patients in our study reported regular alcohol consumption. The educational measures for PLHIV followed at the CTA in Brazzaville also include the prohibition of alcohol consumption. Mandiriri et al. [20] in Zimbabwe identified an association with early sexual intercourse between 13 and 16 years. In our study, there was no association with this factor.

Among the immunovirological factors, our study shows that a CD4 count greater than or equal to 200 cells/mm^3 constitutes a protective factor against HR-HPV infection (p=0.003 Ora 95% CI 0.28 [0.70 – 0.84]). Higher values have been reported in the work of Konopnicki et al. [32], who noted that a CD4 count >500 cells/µl for more than 18 months had a protective effect in FVHIV. Conversely, a low CD4 count has been reported as a factor associated with HR-HPV infection in several studies: a CD4 value of <350 cells/µl was noted in Denmark [24], whereas CD4 < 200 cells/mm^3 was identified in Burkina Faso, Ethiopia, and Tanzania [18,21,33]. Along the same lines, a meta-analysis of work conducted in Asia showed a high risk of HR-HPV infection when the CD4 value was below 200 cells/mm^3 [25]. The role of T lymphocytes in the immune response against infection is known. Regarding HPVs specifically, work on animal models has shown that the depletion of T lymphocytes reduces the control of HPV replication in the organism [34]. HIV leads to the failure of the immune system, which causes its ability to ensure the clearance of HPV to be lost. Antiretroviral therapy, aimed at restoring the immunity of people infected with HIV, is one of the means of ensuring the primary prevention of genital cancers induced by the virus.

Our study shows that the amount of circulating virus, expressed by the plasma viral load of RNA-HIV (PVL-HIV), does not influence the presence of HPV-HR on the cervix. This observation corroborates the work of Sosso et al., which states that HIV infection interferes with HPV due to the immunodeficiency induced by HIV and not by a direct inter-action between the viruses [16]. Detectable PVL-HIV (up to 50 copies/µl) was reported as an associated factor in Ethiopia [21]. In the West Indies, the protective role of an undetectable PVL-HIV (<50 copies/µl) has been identified [31].

5. Conclusions

Our study reveals a high prevalence of HR-HPV among women living with HIV. It shows a significant association between the low level of education and genital infection by high-risk HPV, as well as the protective role of the CD4 count greater than 200/mm^3, in this population group. These observations suggest the establishment of a system for the systematic and regular screening of HPV-HR in women affected by HIV and the strengthening of preventive measures for sexually transmitted infections. The means of communication used for prevention must be adapted to the target populations' level of education. Measures must also be taken to ensure compliance with antiretroviral treatment and to guarantee the restoration of the immunity of those affected.

Conflicts of Interest

The authors declare no conflict of interest.

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REFERENCES

[1] Egawa N., Egawa K., Griffin H., Doorbar J. "Human Papillomaviruses; Epithelial Tropisms, and the Development of Neoplasia", Viruses Vol.7, pp. 3863-3890, 2015.

[2] Frazer IH. "Interaction of human papillomaviruses with the host immune system: A well evolved relationship", Virology, Vol. 384, pp. 410-414, 2009.

[3] Cubie HA. "Diseases associated with human papillomavirus infection", Virology, Vol. 445, no. 1-2, pp. 21-34, 2013.

[4] Shanmugasundaram S, and You J. "Targeting Persistent Human Papillomavirus Infection", Viruses Vol.9, 229, 2017.

[5] Tummers B., van Der Burg SH. "High-Risk Human Papillomavirus Targets Crossroads in Immune Signaling", Viruses, Vol.7, pp. 2485-2506, 2015.

[6] Lu S., Cong X., Li M., Chang FX. and Ma L. "Distribution of high-risk human papillomavirus genotypes in HPV-infected women in Beijing, China", J Med Virol, vol.87, no.3, pp. 504-507, 2015.

[7] Yakub MM., Fowotade A., Anaedobe CG., Manga MM., Bakare RA. and Abimiku BA. "Human papillomavirus correlates of high grade cervical dysplasia among HIV-Infected women at a major treatment centre in Nigeria: a cross-sectional study", Pan Afr Med J, vol. 33, 125, 2019.

[8] World Health Organization. Immunization, Vaccines and Biologicals, 2023, Online available from https://www.who .int/publications/i/item/WHO-SRH-23.1 (accessed on 20 April 2023).

[9] Ahdieh L., Klein RS., Burk R., et al. "Prevalence, incidence, and type-specific persistence of human papillomavirus in human immunodeficiency virus (HIV)-positive and HIV-negative women", J Infect Dis, vol.184, pp. 682-690, 2001.

[10] Liu G., Sharma M., Tan N., and Barnabas RV. "HIV-positive women have higher risk of human papilloma virus infection, precancerous lesions, and cervical cancer", AIDS, vol. 32, no. 6, pp. 795-808, 2018.

[11] Okoye JO., Ofodile C., Adeleke OK. and Obioma O. "Prevalence of high-risk HPV genotypes in sub-Saharan Africa according to HIV status: a 20-year systematic review", Epidemiology and health, vol.43, e2021039, 2021.

[12] Ma X., Wang Q., Ong JJ., Fairley CK., Su S., Peng P. et *al*. "Prevalence of human papillomavirus by geographical regions, sexual orientation and HIV in China: a systematic review and meta-analysis", Sex Trans Dis, vol. 94, no. 6, pp. 434-442, 2018.

[13] Hesselink AT., Meijer CJL., Poljak M., Berkhof J., van Kemenade JF., van der Salm ML. et *al*. "Clinical Validation of the Abbott RealTime High Risk HPV Assay According to the Guidelines for Human Papillomavirus DNA Test Requirements for Cervical Screening", J Clin Microbiol, vol.51, no.7, pp. 2409-2410, 2013.

[14] Nganga PC., Boumba LMA., Tsimba CPL., Tchibinda FGL., Nkounkou RBB., Ataboho EE et al. "Prevalence and Genotyping of Human Papillomavirus among Women in the Departments of Niari and Bouenza, Republic of the Congo", Journal of Biosciences and Medicines, vol. 10, no1, pp. 64-77, 2022.

[15] Boumba LM., Qmichou Z., Mouallif M., Attaleb M., El Mzibri M., Hilali L., et al. "Human papillomavirus genotypes distribution by cervical cytologic status among women attending the General Hospital of Loandjili, Pointe-Noire, Southwest Congo (Brazzaville)", J Med Virol, vol.87, no 10, pp. 1769-1776, 2015.

[16] Sosso SM., Tchouaket MCT., Fokam J., Simo RK., Torimiro J., Tiga A., et al. "Human immunodeficiency virus is a driven factor of human papilloma virus among women: evidence from a cross-sectional analysis in Yaoundé, Cameroon", Virol J, vol. 17, no.1, 69, 2020.

[17] Yakub MM., Fowotade A., Anaedobe CG., Manga MM., Bakare RA., Abimiku BA. "Human papillomavirus correlates of high-grade cervical dysplasia among HIV-Infected women at a major treatment center in Nigeria: a cross-sectional study", Pan Afr Med J, vol. 33, 125, 2019.

[18] Kelly HA., Sawadogo B., Chikandiwa A., Segondy M., Gilham C., Lompo O. et al. "Epidemiology of high-risk human papillomavirus and cervical lesions in African women living with HIV/AIDS: effect of anti-retroviral therapy", AIDS, vol.31, no.2, pp. 273-285, 2017.

[19] McClung N., Mathoma A., Gargano JW., Nyepetsi NG, Querec TD, Onyekwuluge J et al. "HPV prevalence among young adult women living with and without HIV in Botswana for future HPV vaccine impact monitoring", BMC Infect Dis, vol.22, 176, 2022.

[20] Mandiriri AM., Pascoe MJ., Shamu T. and Lowe S. "Cervical human papillomavirus prevalence, risk factors and outcomes in a cohort of HIV-infected women in Harare, Zimbabwe", South Afr J HIV Med, vol.21, no.1, 1123, 2020.

[21] Megersa T., Dango S., Kumsa K., Lemma K. and Lencha B. "Prevalence of high-risk human papillomavirus infections and associated factors among women living with HIV in Shashemene town public health facilities, Southern Ethiopia", BMC Women's Health, vol.23, no.1, 125, 2023.

[22] Ouladlahsen A., Fayssel N., Bensghir R., Baba H., Lamdini H., Sodqi M. et al. "The Human papillomavirus among women living with Human Immunodeficiency Virus in Morocco: A prospective cross-sectional study", J Infect Dev Ctries, vol. 12, no. 6, pp. 477-484, 2018.

[23] Konopnicki D., Manigart Y., Gilles C., Barlow P., de Marchin J., Feoli et al. High-risk human papillomavirus infection in HIV-positive African women living in Europe. J Int AIDS Soc, vol. 16, 18023, 2023.

[24] Thorsteinsson K., Ladelund S., Storgaard M. et al. "Persistence of cervical high-risk human papillomavirus in women living with HIV in Denmark – the SHADE", BMC Infect Dis, vol.19, 740, 2019.

[25] Caicedo-Martínez M., Fernández-Deaza G., Ordóñez-Reyes C., Olejua P., Nuche-Berenguer B., Melle MB. et al. "High-risk human papillomavirus infection among women living with HIV in Latin America and the Caribbean: A systematic review and meta-analysis", International Journal of STD & AIDS, vol. 32, no.14, pp 1278-1289, 2021.

[26] Verrier F., Le Coeur S., and Delory T. "Cervical Human Papillomavirus Infection (HPV) and High Oncogenic Risk Genotypes among Women Living with HIV in Asia: A Meta-Analysis", J Clin Med, vol.10, 1911, 2021.

[27] Williamson AL. "The Interaction between Human Immunodeficiency Virus and Human Papillomaviruses in Heterosexuals in Africa", J Clin Med, vol.4, pp 579-592, 2015.

[28] World Health Organization. Global strategy to accelerate the elimination of cervical cancer as a public health problem. Geneva 2020. Online available from: https://www.who.int/publications/i/item/9789240014107. (accessed on 04 April 2023)

[29] Masdoua N., Boublenza L., Hassaine H., Ngou J., Nahet A., Segondy M., et al. "Characteristics of HPV infection in women at risk in Western Algeria", Med Mal Infect, vol. 47, no.1, pp. 38-41, 2017.

[30] Jung M. "Risk factors of sexually transmitted infections among female sex workers in Republic of Korea," Infect Dis Poverty, vol.8, no.1, 2019.

[31] Abel S., Najioullah F., Voluménie JL., Accrombessi L., Carles G., and Catherine D. "High prevalence of human papillomavirus infection in HIV-infected women living in French Antilles and French Guiana," PLoS ONE, vol.14, no.9, e0221334, 2019.

[32] Konopnicki D., Manigart Y., Gilles C., Barlow P., de Marchin J., Francesco Feoli F. et al. "Sustained Viral Suppression and Higher CD4+ T-Cell Count Reduces the Risk of Persistent Cervical High-Risk Human Papillomavirus Infection in HIV-Positive Women", J Infect Diseases, vol. 207, no.11, pp. 1723-1729, 2013.

[33] Mchome BL, Krüger Kjaer S, Manongi R, Swai P, Waldstroem M, Iftner T, et al. "HPV types, cervical high-grade lesions, and risk factors for oncogenic human papillomavirus infection among 3416 Tanzanian women", Sex Transm Infect, vol. 97, no.1, 56, 2021.

[34] Maglennon GA., McIntosh PB., Doorbar J. "Immunosuppression facilitates the reactivation of latent papillomavirus infections," J Virol, vol. 88, no. 1, pp. 710-716, 2014.