Rwanda Medical Journal RMJ

ORIGINAL ARTICLE Open Access

Genotypic distribution of penile and anal high-risk human papillomavirus among men who have sex with men in Rwanda

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ABSTRACT

INTRODUCTION: Men who have sex with men (MSM) experience a disproportionately high burden of high-risk human papillomavirus (hr-HPV) infection, particularly at anal and penile sites, increasing their risk of HPV-associated cancers. In Rwanda, data on the prevalence and genotype distribution of hr-HPV among MSM remain limited. The objective was to determine the prevalence and genotypic distribution of anal and penile hr-HPV infections among MSM in Rwanda, assess the frequency of multiple hr-HPV genotype co-infections, and compare genotype distribution between HIV-infected and HIV-uninfected MSM.

METHODS: A cross-sectional laboratory-based study was conducted among 120 MSM residing in Kigali, Rwanda. Anal and penile swab samples were collected and tested for hr-HPV using a real-time isothermal amplification genotyping assay capable of detecting 14 oncogenic HPV genotypes and HPV-53. Sociodemographic and clinical information, including HIV status, was obtained through a structured questionnaire. Data were analysed using STATA version 15.

RESULTS: The prevalence of hr-HPV was 28.3% in anal samples and 39.2% in penile samples. HPV-16 was the most frequently detected genotype at both sites, followed by HPV-53, HPV-31, HPV-51, and HPV-59. Multiple genotype infections were common among participants. HIV-positive MSM had a significantly higher prevalence of penile hr-HPV infection (65.0%) than HIV-negative MSM (34.0%) ($p < 0.05$).

CONCLUSION: MSM in Rwanda bear a substantial burden of anal and penile hr-HPV infection, characterized by the predominance of oncogenic genotypes and higher prevalence among individuals living with HIV. These findings highlight the need for targeted HPV prevention and control strategies, including expanded vaccination, integrated HPV screening, and strengthened HIV care services for this vulnerable population.

Keywords: High-risk human papillomavirus, Men who have sex with men, Genotypic distribution, Penile and anal infection; Human immunodeficiency virus, Rwanda

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Received: 14th June 2026; **Initial decision given:** 15th June 2026; **Revised manuscript received:** 16th Junel 2026; **Accepted:** 29th June 2026.
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Citation for this article: Patrick Nemeyimana et al. Genotypic distribution of penile and anal high-risk human papillomavirus among men who have sex with men in Rwanda. Rwanda Medical Journal, Vol. 83, no. 2, p. 48-55, 2026. https://dx.doi.org/10.4314/rmj.v83i2.6

INTRODUCTION

Human papillomavirus (HPV) is among the most prevalent sexually transmitted infections globally, with over 200 different genotypes discovered so far [1]. Among these About 40 genotypes target the anogenital region, and they are generally divided into low-risk and high- risk categories according to their cancer-causing ability. High-risk HPV strains, like HPV-16 and HPV-18, are closely linked to the onset of anogenital malignancies, including cancers of the anus, penis, cervix, and oropharynx. In contrast, low-risk strains, such as HPV-6 and HPV- 11, usually lead to non-cancerous growth like genital warts [2]. The impact of HPV infection is unevenly greater in specific groups, such as men who have sex with men (MSM), because of behavioral, immune system, and societal influences [3].

. Men who have sex with men (MSM) exhibit a notably greater rate of anal HPV infection than heterosexual men, which is linked to a higher likelihood of developing anal intraepithelial neoplasia and anal cancer [4]. HPV infection of the penis also plays a role in the development of penile intraepithelial neoplasia and invasive penile carcinoma, although in previous studies, it exhibited less frequently than anal cancer [5]. In addition, men who have sex with men (MSM) face a higher likelihood of acquiring HIV, which works in combination to heighten vulnerability to persistent HPV infection and its advancement to cancerous conditions [6].

The genetic variation of HPV differs by region and across various population groups, affecting the patterns of HPV-associated illnesses and the success of immunization efforts [7]. Worldwide, HPV-16 is the most common high-risk strain identified in anal and penile infections among men who have sex with men (MSM); however, other types like HPV-18, HPV-31, HPV-33, and HPV-45 also contribute significantly [8]. Identifying the particular genotype patterns within a specific population is essential for customizing prevention measures, such as choosing appropriate vaccines and designing effective screening initiatives [8].

In Rwanda and across sub-Saharan Africa, data regarding the distribution of HPV genotypes among MSM remains limited. This growing recognition of at-risk key populations highlights a lack of detailed information on the occurrence of anal and penile hr-HPV infections in MSM [9]. This knowledge gap poses a challenge to implementing effective public health strategies intended to lower HPV-related illness and death rates within this vulnerable population.

MSM encounter major obstacles in accessing healthcare services, largely due to societal stigma, prejudice, and restrictive legal frameworks [10]. This leads to insufficient diagnosis and inadequate treatment of HPV and other sexually transmitted infections, thereby sustaining their spread and contributing to negative health consequences [11]. Recent research has revealed a high rate of HIV among Rwandan MSM, suggesting a possible co-occurrence with hr-HPV infections that may elevate the risk of developing cancer [12].

Furthermore, with the rollout of HPV vaccines that target multiple high-risk strains, determining the prevalent HPV types within the MSM population is crucial for assessing vaccine reach and effectiveness [13] Existing vaccines mainly focus on HPV-16, HPV-18, and several other cancer-causing strains; however, the circulation of HPV genotypes not included in the vaccine may influence its overall effectiveness. Hence, monitoring HPV genotypes in anal and penile regions will generate valuable data to inform vaccination strategies and screening programs specifically designed for MSM in Rwanda.

At present, there is a major gap in knowledge regarding HPV infection patterns among MSM in Rwanda. There is virtually no published data on the prevalence of anal and penile HPV, the range of circulating genotypes in this group, or the extent of multiple genotype co-infections [14]. This lack of context-specific evidence limits the capacity of health authorities and policy designers to craft inclusive and evidence-based HPV prevention strategies, such as expanding vaccination to male populations or implementing targeted screening among MSM [15].

Gaining insight into the genotypic profiles of HPV in both anal and penile areas is essential, as these anatomical sites may harbor different HPV types or combinations, influencing disease progression and vaccine impact [15]. In light of these challenges, the current study aims to address a critical gap by evaluating the prevalence and genotype diversity of anal and penile HPV among MSM in Rwanda. By also exploring the occurrence of multiple genotype infections, this research will provide vital data to support inclusive, population-specific public health initiatives and guide future HPV control efforts in high-risk male populations.

METHODS

Study design and population

A cross-sectional study was conducted among 120 men who have sex with men (MSM) aged 18 years and above residing in Kigali, Rwanda. Upon getting the ethical approval Participants were recruited through purposive and snowball sampling techniques with the support of community-based organizations serving key populations.

Data collection and sources

Sociodemographic and clinical data, including HIV status, were collected using a structured interviewer-administered questionnaire. Anal and penile specimens were collected by trained personnel using sterile Dacron swabs following standardized procedures. Samples were placed in Preservative medium, stored at 2–8°C, and transported to the molecular biology laboratory for analysis. HPV genotyping was performed using the AmpFire® HPV Genotyping Detection assay, a rapid real-time isothermal amplification-based method capable of detecting 14 high-risk HPV genotypes and HPV-53 directly from clinical specimens. To ensure data reliability and laboratory quality, all specimen collection and processing procedures followed standard operating procedures, equipment was calibrated and maintained according to manufacturer recommendations, and positive and negative controls were included in each assay run.

Data analysis

Data were entered into Microsoft Excel, checked for completeness and consistency, and analyzed using STATA version 15. Descriptive statistics were used to determine the prevalence and genotype distribution of high-risk HPV infections, while comparative analyses were conducted to assess differences between HIV-infected and HIV-uninfected participants.

RESULTS

A total of 120 participants were included in the study. The mean age was 27.2 years with a standard deviation of 5.17 years, indicating a relatively young population. All participants were male. Out of the 120 participants, 83.3% (n=100) were HIV-negative while 16.7% (n=20) were HIV- positive. The majority of participants were born in Kigali City, accounting for 51.7% (n = 62) of the study population. This was followed by the Southern Province with 15.0% (n = 18), the Western Province with 11.7% (n = 14), and the Northern Province with 8.3% (n = 10). Participants born in the Eastern Province represented 5.8% (n = 7) of the sample. A smaller proportion were born in Bujumbura, accounting for 3.3% (n = 4), while 4.2% (n = 5) reported being born in other locations. Overall, more than half of the participants originated from Kigali City, indicating a predominance of urban-born individuals in the study population.

As per Table 1, the prevalence of hr-HPV infection differed significantly by HIV status at both anal and penile sites. Overall, hr-HPV was detected in 28.3% of participants at the anal site and 39.2% at the penile site. Among HIV-negative individuals, the anal hr-HPV prevalence was 25.0%, compared to a notably higher 45.0% in HIV-positive individuals. Similarly, at the penile site, hr-HPV prevalence was 34.0% in HIV-negative participants but rose sharply to 65.0% among those who were HIV-positive. These differences were statistically significant

Table 1: hr-HPV Prevalence by Site and HIV Status

Site	HIV Status	HPV Positive (n)	Total (n)	Prevalence (%)	95% CI
Anal	Overall	34	120	28.3	21.0 – 37.0
Anal	Negative	25	100	25.0	18.0 – 34.0
Anal	Positive	9	20	45.0	26.0 – 66.0
Penile	Overall	47	120	39.2	31.0 – 48.0
Penile	Negative	34	100	34.0	25.0 – 44.0
Penile	Positive	13	20	65.0	43.0 – 82.0

($p < 0.05$), indicating that HIV-positive status is associated with a higher likelihood of hr-HPV infection at both anatomical sites.

Table 2: Genotype frequencies

Number of Genotypes	Frequency	Percentage (%)
0	49	40.83
1	33	27.50
2	10	8.33
3	11	9.17
4	9	7.50
5+	8	6.67

Distribution of Total Number of hr-HPV Genotypes Detected

The number of HPV genotypes detected in each individual ranged from 0 to 5 or more (Table 2).

Notably, 40.8% of the participants had no detectable HPV genotypes, indicating a large proportion without infection or with undetectable viral presence. Among those with HPV, the majority had one genotype (27.5%), while smaller proportions harbored multiple genotypes: 8.3% had two genotypes, 9.2% had three, 7.5% had four, and 6.7% had five or more genotypes. This distribution highlights that while single-genotype infections are common, a significant subset of individuals is infected with multiple HPV genotypes, which may have implications for disease risk and progression.

Genotype Distribution

Anal hr-HPV Genotypes: A total of 85 genotype detections were made in the anal samples. The most frequent genotype was HPV-16 (16.5%), followed by HPV-53 (9.4%) and HPV-31 (8.2%).

A total of 85 HPV genotype detections were identified from anal samples collected in the study. Among these, HPV-16 was the most prevalent genotype, accounting for 16.5% of all detections. This was followed by HPV-53 at 9.4% and HPV-31 at 8.2%, indicating these genotypes are the predominant strains circulating in the anal region of the participants.

Other genotypes detected with notable frequency included HPV-52 (7.1%) and HPV-58 (5.9%). Several genotypes, including HPV-51, HPV-66, and HPV-56, were each detected 4 times, representing 4.7% individually. A group of genotypes such as HPV-39, HPV-59, HPV-45, HPV-35, and HPV-59 appeared 3 times each (3.5%), while genotypes like HPV-18, HPV-33, HPV-35, HPV-31, HPV-68, and HPV-18 were found twice each (2.4%). Finally, some genotypes including HPV-52, HPV-33, HPV-68, HPV-53, HPV-39, and HPV-66 were detected only once, making up 1.2% each.

Penile hr-HPV Genotypes

The penile samples showed a total of 89 genotype detections. HPV-16 was the most common (20.2%), followed by genotype 51 (9.0%) and HPV-59, HPV-39, and 53 (each about 6.7%).

Table 3: Penile genotype distribution

Genotype	Frequency	Percentage (%)
HPV-16	18	20.22
51	8	8.99
59, HPV-39	6 each	6.74 each
HPV-45, 66, 53, HPV-59, 18	4 each	4.49 each
HPV-66, HPV-31, HPV-52, 52, 45	3 each	3.37 each
35, HPV-56, 56, 58	2 each	2.25 each
16, 31, 54, HPV-16, HPV-51, HPV-66, 68, 39	1 each	1.12 each

A total of 89 HPV genotype detections were identified from penile samples. The most prevalent genotype was HPV-16, representing 20.2% of detections. This was followed by genotype 51 at 9.0%, and genotypes HPV-59, HPV-39, and HPV-53, each accounting for approximately 6.7% of detections. Other genotypes such as HPV-45, HPV-66, HPV-53, HPV-59, and HPV-18 were each detected 4 times (4.5%), while HPV-66, HPV-31, HPV-52, and HPV-45 appeared 3 times each (3.4%). Additionally, genotypes like HPV-35, HPV-56, and HPV-58 were detected twice each (2.3%), and a variety of genotypes including HPV-16, HPV-31, HPV-54, HPV-51, HPV- 66, HPV-68, and HPV-39 were each found once (1.1%) (Table 3.

Association Between HIV and hr-HPV Infections

Anal hr-HPV Infection by HIV Status: Among the study participants, HIV-negative individuals were more frequently HPV-negative (75) compared to HPV-positive (25) at the anal site. In contrast, HIV-positive individuals had a lower number of HPV-negative cases (11) and a smaller number of HPV-positive cases (9).
However, the difference in HPV infection status between HIV-negative and HIV-positive groups was not statistically significant (Chi-square = 2.3721, p = 0.1235). This indicates that, within this sample, HIV status was not significantly associated with HPV infection presence.

Penile hr-HPV Infection by HIV Status: The data show that among HIV-negative participants, 66 were HPV-negative and 34 were HPV- positive at the penile site. In contrast, among HIV-positive individuals, fewer were HPV- negative (7), while a higher proportion were HPV-positive (13). The difference in penile HPV infection status between HIV-negative and HIV-positive groups was statistically significant (Chi-square = 5.4841, p = 0.0192). This indicates that HIV-positive participants had a significantly higher prevalence of penile HPV infection compared to HIV-negative individuals, suggesting an association between HIV infection and increased susceptibility to penile HPV infection

Multiple hr-HPV Infections by HIV Status: The data reveal that among HIV-negative participants, 75 had no multiple hr-HPV infections, while 25 had multiple infections. Conversely, among HIV-positive participants, only 7 had no multiple infections, whereas 13 had multiple hr-HPV infections. The difference in the prevalence of multiple HPV infections between HIV-negative and HIV-positive groups was highly statistically significant (Chi-square = 10.5443, p = 0.0012). This indicates that HIV- positive individuals are significantly more likely to have multiple hr-HPV infections compared to HIV-negative individuals, suggesting that HIV infection may increase susceptibility to co- infection with multiple HPV genotypes.

DISCUSSION

The present study investigated the prevalence and genotype distribution of HPV infections among MSM in Rwanda, with particular attention to differences by HIV status and anatomical site of infection. The study population consisted of 120 young adult males, with a mean age of 27.2 years, predominantly originating from Kigali City. This demographic reflects a typical urban MSM cohort that may have unique risk profiles due to sexual behaviors and access to healthcare services.
The findings demonstrated a high prevalence of hr-HPV infections, with 28.3% of participants testing positive for anal hr-HPV and 39.2% for penile hr-HPV of the mean age 27.2 years as supported the study conducted in Serbia, which also specified that HPV in male is prevalent in age 27-29 years [16]. This agrees with another study conducted by Murenzi et al [10]. There was no significant difference in the HPV infection between anal and penile, suggesting that both anatomical regions are important reservoirs for HPV in this population [16]. Notably, our study showed that high-risk (HR) genotypes (16 and 18) were exclusively detected in all HPV- positive samples, highlighting the oncogenic potential of circulating HPV types and reinforcing the importance of targeted screening and vaccination strategies.

HIV status was found to be a critical determinant of HPV infection patterns. HIV-positive participants had significantly higher prevalence rates of HPV at both anal (56.3%) and penile (65.6%) sites compared to HIV-negative individuals. Research showed that the most prevalent HPV cases were found in HIV positive male who had sex with males [16]. Moreover, multiple HPV genotype infections were markedly more common among HIV-positive individuals (p = 0.0012), consistent with the

hypothesis that immunosuppression facilitates persistent and concurrent infections. These findings align with previous research in China demonstrating synergistic interactions between HIV and HPV, where compromised immune responses in HIV-infected individuals contribute to higher viral persistence, increased diversity of HPV genotypes, and elevated risk of HPV-associated malignancies [17].

The genotype distribution analysis revealed HPV-16 as the most prevalent type in both anal (16.5%) and penile (20.2%) samples, followed by other high-risk genotypes such as HPV-53, HPV-31, HPV-51, and HPV-59. The predominance of HPV-16 is of particular concern, given its established role in the pathogenesis of anal, penile, and oropharyngeal cancers. The detection of multiple high-risk genotypes further underscores the potential burden of HPV-related disease in this population. It was confirmed that among HIV positive and negative, HPV-16 was the most predominant.

Interestingly, while HIV status was strongly associated with penile HPV prevalence and multiple genotype infections, the association with anal HPV prevalence did not reach statistical significance in one analysis ($p = 0.1235$). This discrepancy may be attribute ed to the small sample size of HIV-positive participants, leading to limited statistical power, or to differences in exposure patterns and local immune responses between anatomical sites. The same study was conducted in Nigeria that the prevalence of anal HR-HPV was higher among HIV-positive than HIV-negative MSM (91.1% vs. 40.6%, $P < 0.001$) which is supportive for our findings [18].

These results highlight the need for comprehensive HPV prevention strategies among MSM in Rwanda, particularly among HIV-positive individuals who bear a disproportionate burden of high-risk and multiple HPV infections. Targeted interventions may include increased HPV vaccination coverage, routine screening for anal and penile lesions, and strengthened HIV care to mitigate immunosuppression-related vulnerability. The exclusive detection of high-risk genotypes also supports prioritizing vaccines that cover a broad range of oncogenic HPV types.

Future studies should aim to include larger and more diverse MSM populations across Rwanda to validate these findings and explore regional variations. Longitudinal research is warranted to assess the persistence of HPV infections, the progression to precancerous lesions, and the impact of antiretroviral therapy on HPV natural history. Additionally, molecular studies focusing on viral load, immune markers, and interactions between multiple genotypes could provide deeper insights into the pathogenesis of HPV in HIV-positive MSM. Evaluating the effectiveness of HPV vaccination programs in this high-risk group will also be essential for informing national policy.

CONCLUSION

This study provides critical insights into the prevalence, genotype distribution, and risk factors associated with hr-HPV infection among men MSM in Rwanda. The findings revealed a considerable burden of hr-HPV infections, with prevalence rates of 28.3% at the anal site and 39.2% at the penile site. Although statistical analysis did not show a significant difference between these anatomical sites, both were confirmed as important reservoirs for hr-HPV infection. High-oncogenic genotypes, particularly HPV16, were the most predominant across both sites, underscoring their oncogenic potential and their significance in cancer prevention strategies. The detection of multiple high-risk genotypes in a notable proportion of participants further suggests an elevated risk for HPV-related malignancies in this population.

HIV status was found to be a key determinant of hr-HPV infection patterns. HIV-positive participants had significantly higher prevalence of penile hr-HPV infections and were more likely to present with multiple hr-HPV genotypes compared to HIV-negative individuals. These findings highlight the synergistic interaction between HIV and hr-HPV, where immunosuppression in HIV-positive individuals facilitates persistent and diverse hr-HPV infections. Despite these alarming findings, hr-HPV infection among MSM in Rwanda remains largely underrecognized, with limited preventive measures and screening programs currently in place. This highlights the urgent need for targeted interventions to address this public health challenge.

Based on the findings of this study, several recommendations are proposed to mitigate the burden of HPV among MSM in Rwanda. First, HPV vaccination programs should be expanded to specifically include MSM, with priority given to HIV-positive individuals who are at an increased risk of persistent and multiple HPV infections.

Public health awareness campaigns should be strengthened to educate both MSM and healthcare providers about HPV transmission, associated risks, and preventive measures.

Second, routine screening for anal and penile HPV-related lesions should be incorporated into HIV care services to enable early detection and timely treatment of precancerous lesions, thereby reducing the risk of progression to malignancy. Strengthening antiretroviral therapy (ART) coverage and adherence is also crucial to decrease immunosuppression and reduce susceptibility to HPV persistence.

Third, comprehensive care models that integrate HIV and HPV management should be developed to address the dual burden of these infections. In addition, further large-scale research is warranted to confirm these findings across diverse MSM populations in Rwanda and to monitor longitudinal outcomes such as HPV persistence, progression to cancer, and the influence of ART on HPV natural history. Molecular studies focusing on viral load, immune responses, and genotype interactions would provide deeper insights into co-infection dynamics.

National HPV vaccination strategies should consider vaccines that cover a broader spectrum of high-risk HPV types, given the diverse genotype distribution observed in this study. Integrating HPV prevention and control into existing sexual health policies targeting key populations is essential for reducing the long-term burden of HPV-associated diseases in Rwanda.

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