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RESEARCH ARTICLE

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Prevalence of HPV types in HIV-positive and negative females with normal cervical cytology or dysplasia

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Abstract

The burden of HPV varies by country and HIV status. The study aimed to evaluate HPV types prevalent in HIV-positive females compared with HIV-negative females in the local population of the federal capital territory in Pakistan.

Method: The selected female population consisted of 65 already diagnosed HIV-positive females and 135 HIV-negative females. Cervical scrap was collected and analyzed for HPV and cytology.

Results: The prevalence of HPV in HIV-positive patients was 36.9%, higher than HIV-negative patients (4.4%). 12.30% had cervical cytology interpreted as "LSIL" and 87.69% had cytology interpreted as "NIL." The high-risk type was detected in 15.39% while 21.54% showed low-risk HPV types. Among the high-risk types, HPV18 (6.15%), HPV16 (4.62%), HPV45 (3.07%), HPV33 (1.53%), HPV58 (3.07%), and HPV68 (1.53%) were found. In patients with LSIL, high-risk HPV accounts for 62.5%. Risk factors, such as age, marital status, educational status, residence, parity, other STDs, and contraceptives, were analyzed to find the correlation with HPV infection Age ≤35 years (OR 1.21, 95% CI, 0.44–3.34), illiterate and incomplete secondary education (OR 1.08, 95% CI, 0.37–3.15), and those reported not to use contraceptives (OR: 1.90; 95% CI: 0.67–5.42) have an association for increased risk of HPV infection.

Conclusion: HPV18, HPV16, HPV58, HPV45, HPV68, and HPV33 were identified among high-risk HPV types. High-risk HPV was detected in 62.5% of low-grade squamous intraepithelial lesions. The data is useful for health policymakers to develop a strategy for HPV screening and prophylactic vaccination to prevent cervical cancer.

KEYWORDS
HPV, HPV and HIV, human papillomavirus, risk factors

1 | INTRODUCTION

Human papillomavirus infection (HPV) infection is a sexually transmitted infection across the world, affecting sexually active individuals. Approximately, 40 genotypes are involved in genital infections and classified as high-risk (HR-HPV) and low-risk HPV (LR-HPV) based on their oncogenic potential. The most common contributory agent for cervical cancer is HPV.[1,2]

Human papillomavirus prevalence has been reportedly higher in HIV-positive women.[3,4] One probable reason might be the inability of HIV compromised immune system to control the replication and expression of HPV in the body and this may lead to the faster progression of invasive cancer than in normal individuals. In HIV-positive patients, HPV infection appears to persist longer than among healthy individuals, thus increasing the risk of developing cancer. Females with multiple HR-HPV type infections are more susceptible to HIV infection, on the other hand, females positive for HIV are prone to acquiring HPV infection.[2,4] However, age, education level, lifestyle, marriage status or sexual activity, and other factors also have an effect on HPV infection.

Pakistan has a low prevalence but is a high-risk country for infection with HIV, with more than 100,000 patients living with AIDS. Epidemiological studies on these sexually transmitted illnesses have been hampered by social taboos (STDs).There is a need to encipher the status of HPV infection among HIV-positive females with normal cervical cytology as this data remain scarce. It is the first report of HPV prevalence in HIV-positive females from this region. Previously, there have been reports of HPV in the general population and with cervical cancer in Pakistan.[5,6] These reports indicated that HPV infection has a great risk to develop cervical cancer. However, none of the studies reports the prevalence of HPV in HIV-positive females with cervical dysplasia.[7]

The purpose of this study is to determine the prevalence of HPV type in HIV-positive and HIV-negative women with cervical dysplasia. Additionally, this study also analyzes the correlation of risk factors with HPV infection.

2 | MATERIALS AND METHODS

2.1 | Patient selection and study design

The study was carried out at Nuclear Medicine Oncology and Radiotherapy Institute (NORI). The study included 135HIV-negative and 65 HIV-positive female patients, aged between 18 and 80 years. These females visited the hospital with different complaints, such as vaginal discharge, postcoital bleeding, postmenopausal bleeding, infertility, and secondary amenorrhea for gynecological examination at Mother and Child Hospital, Pakistan Institute of Medical Science (MCH-PIMS), Islamabad between March 2017 and August 2019.

The study was approved by the ethical review committee of the institute. After signing the consent statement, a nurse interviewed the females and filled out the questionnaire related to sociodemographic, reproductive, and HIV history. In HIV-positive females, $CD4^{+}$T lymphocyte count was retrieved from their hospital medical record, and those who have a count of fewer than 200 cells/mm^3 indicate immunosuppression. The Pap smear test was performed and cytology slides were interpreted by a pathologist using the Bethesda 2001 report system.[8]

Cervical cell scrapes of the patients were collected using a sterile brush and transferred to a tube. A volume of 500 μL of sterile phosphate buffer saline was added and centrifuged at 3000 rpm for 10 min and then transferred the fluid containing cells into an Eppendorf tube and stored at −80°C until processed for DNA isolation. All samples were tested for cervical cytology. A High-risk genotype assay was performed on Abbott Real-time HPV assay. All the samples were further analyzed by sequencing using primer MY09/MY11.

2.2 | HPV DNA isolation and amplification

DNA extraction from cervical scrap was done using the m2000sp instrument and Abbot m sample preparation system DNA. In this procedure, magnetic particles were used to capture HPV DNA. Bound HPV DNA was eluted from the particles and collected in 96-well plates. Master mix prepared using a high-risk HPV typing kit and dispensed (amplified reagents) to the extracted DNA in 96 deep well plates.

Real-time detection and typing of HPV were performed on m2000rt. Probes were labeled with different fluorophores allowing their signals to be distinguishable in a single reaction. In addition, each detected signal has been reported individually. Assay results are automatically reported by the m2000rt at the end of real-time PCR. According to the manufacturer's instructions, the cut-off for cycles threshold is fixed at 32.

2.3 | Amplification of the L1 gene of HPV for sequencing

The MY09/MY11 primers were used to amplify the L1 gene region, yielding 450 bp products. The PCR product was sequenced and aligned by using the CLC Main workbench version 6 with reference sequence from the NCBI database, then HPV types identified.

2.4 | Statistics

A questionnaire was filled out by each individual to collect the information. Descriptive statistics were used to estimate the proportion. The Chi-square test was used to find the significant association between HPV status and study variables. A p-value <0.05 was considered significant. Logistic regression with the adjusted odd ratio at 95% confidence of interval was used to estimate the variables.

3 | RESULTS

The study was carried out on 200 females, 65 females are HIV-positive, and 135 females are HIV-negative. They have aged 18–80 years (mean age 35.12 ± 10.67). Baseline characteristics are given in Table 1.

Human papillomavirus was found in 36.9% (24/65) of HIV-positive females, while in the uninfected HIV group of females HPV was observed at 4.5% (6/135). High-risk HPV type was found in

TABLE 1 Baseline characteristics of HPV-positive and negative Females enrolled in the study.

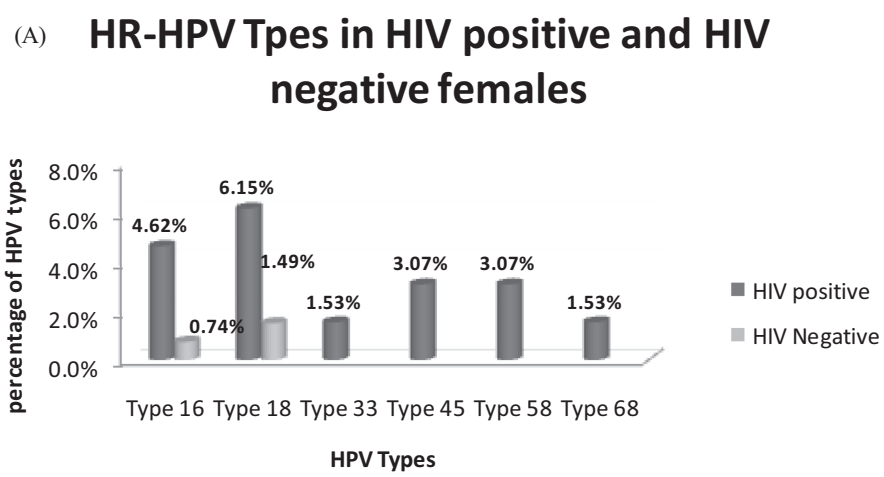
Characteristics	(Group A) HIV-negative			(Group B) HIV-positive			
	Sample size	*n* (%) HPV positive	*n* (%) HPV negative	Sample size	*n* (%) HPV positive	*n* (%) HPV negative	*p*-value
HPV	135	6 (4.4%)	129 (95.5%)	65	24 (36.9%)	41 (63.1%)	
Age (median = 36.43)							
≤35 years	58	3 (5.1%)	55 (94.8%)	36	14 (65.5%)	22 (61.1%)	0.03
>35 years	77	3 (3.9%)	74 (96.0%)	29	10 (34.5%)	19 (38.9%)	
Marital status							
Single, widow and separated	44	2 (4.5%)	42 (95.4%)	25	12 (48%)	13 (56%)	0.89
Married	91	4 (4.3%)	87 (95.6%)	40	12 (30%)	28 (70%)	
Parity (no. of pregnancies)							
≤3	56	2 (3.5%)	54 (96%)	34	10 (29.42%)	18 (52.9%)	0.76
>3	79	4 (5.0%)	75 (49.9%)	37	14 (37.84%)	23 (62.17)	
Educational status							
Literate (secondary and higher education)	62	2 (3.2%)	60 (96.7%)	21	8 (38.1%)	13 (61.9%)	0.002
Illiterate (incomplete secondary education)	73	4 (5.4%)	69 (94.6%)	44	16 (36.4%)	28 (63.3%)	
Residence							
Rural	97	3 (3.09)	93 (95.8)	48	17 (35.4%)	31 (64.6%)	
Urban	38	3 (7.8)	35 (92.1)	17	7 (41.2%)	10 (58.8%)	
Other STDs							
Yes	108	6 (5.55%)	102 (94.4%)	37	10 (27.02%)	27 (72.9%)	0.210
No	27	2 (7.4%)	25 (92.0%)	28	14 (50.0%)	14 (50.0%)	
Use of contraceptives							
Yes	102	5 (4.9%)	97 (95.0%)	37	8 (28.5%)	20 (71.42%)	0.04
No	33	2 (6.06%)	31 (93.9%)	28	16 (43.2%)	21 (56.7%)	
Cytology							
Normal	135	6 (4.4%)	129 (95.5%)	57	16 (28.07%)	41 (71.93%)	0.05
Abnormal	0	0	0	8	8 (100%)	0	

13/65 (19.97%) of HIV-positive females, while low-risk HPV types were found in 11/65 (16.92%). Among high-risk (HR) HPV types, HPV18 (6.15%), HPV16 (4.62%), HPV58 (3.07%), HPV45 (3.07%), HPV68 (1.53%), and HPV33 (1.53%) were detected. Low-risk HPV11 was the most common HPV type in the study (6.15%) followed by HPV6 (4.61%), HPV61 (1.54%), HPV54 (1.54%), and HPV55 (3.07%) (Figure 1A,B).

All the HIV-positive females were tested for colposcopy and Pap smear cervical cytology. Of these 12.30% (8/65) had abnormal cervical cytology and 87.69% (57/65) had normal cervical cytology. Histopathology results confirmed all eight patients had a low-grade squamous intraepithelial lesion (LSIL). Among these, high-risk HPV types account for 62.5%. The most prevalent high-risk HPV type 18 was accounting for 50% (4/8) followed by HPV16 with 12.5% (1/8). While in patients with normal cytology (no cell change) HPV 16 (2/16, 12.50%) along with other high-risk HPV types HPV33 (1/16, 6.25%), HPV45 (2/16, 12.50%), HPV58 (2/16, 12.50%), HPV68 (1/16, 6.25%) were observed. None of the patients with normal cytology had HPV18. The distribution of HPV type with normal and abnormal cytology results is shown in (Figure 2A,B).

Out of 135 uninfected HIV females, six (4.44%) were positive for the HPV DNA test on PCR and, 129 (95.5%) were negative for the HPV DNA test. Out of these 6 HPV positives, 2.2% (3/135) were positive for LR-HPV and 2.2% (3/135) were positive for HR-HPV. HR HPV16 (16.66%), HPV18 (33.33%), LR HPV11 (16.66%), and HPV6 (16.66%) were detected among HPV-positive cases (Figure 2A,B).

Univariate analysis was used to find the risk factor related to HPV infection. A significant variable associated with HPV infection was age ≤35 years (odd ratio 1.21, 95% confidence of interval [CI], 0.44–3.34) than patients aged >35 years. Illiterate (incomplete secondary education) than Literate (secondary and higher education) (OR 1.08, 95% CI, 0.37–3.15) was also associated with higher HPV infection. The regular use of contraceptive slows down the likelihood to get an HPV infection (OR: 1.90; 95% CI: 0.67–5.42). Single, widow, and separated had an increased Odd ratio for HPV infection as compared with married women (OR: 2.67, 95% CI, and 0.79–9.02).



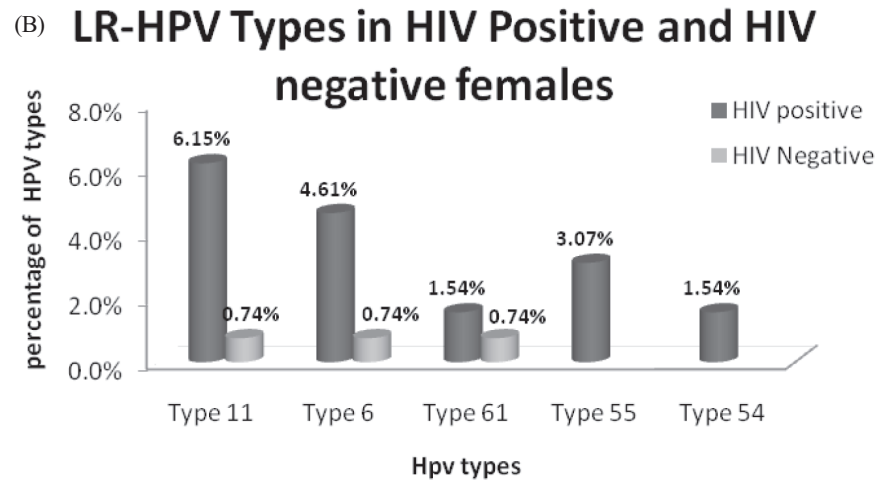


FIGURE 1 (A) Distribution of high-risk HPV genotypes in HIV-positive and HIV-negative patients. (B) Distribution of low-risk HPV types in HIV-positive and HIV-negative patients.

Rural background (OR: 0.78, 95% CI, 0.25–2.43), other STDs (OR: 0.63, 95% CI, 0.0.24–1.69), and parity (OR: 0.91, 95% CI, 0.33–2.53) does not show any association with an increased risk of getting an HPV infection in HIV-infected females (Table 2).

In HIV-negative group risk factors association was also analyzed and found that HPV infection was higher in the age group ≤35 years (OR 1.34, 95% CI, 0.26–6.29). Illiteracy rate (OR 1.74, 95% CI, 0.31–9.83), use of contraceptives (OR 1.25, 95% CI, 0.23–6.78), single, widow, and separated versus married (OR 1.04, 95% CI 0.18–5.88) were associated with HPV infection (Table 2). The presence and absence of other STDs, residence in rural, and urban, and parity were not associated with likely hood of HPV infection (Table 2).

Furthermore, the association of $CD4^+$ count and HPV infection among HIV-positive females was carried out. Patients with a cell count less than 200/μl have a 10 times higher chance of getting HPV infection as compared with those having a cell count >200–400/μL.

4 | DISCUSSION

Population-based HPV screening is the best way to prevent the development of cervical cancer. Findings show a high burden of HPV (36.9%) among HIV-positive females than among -negative HIV females. The HPV screening program is partially functional and therefore deter to estimating the true burden of infection.[9] The current study confirms a 36.9% incidence of HPV among HIV-infected patients, which is lower than the incidence reported by Clifford et al. who reported a 41% prevalence of HPV in HIV-infected females with normal cytology in the meta-analysis.[10] Other studies reported 38.9%[11] and 22%.[12] Moreover, studies reported a prevalence range of 20–70% in developing countries.[13]

The current study shows other high-risk HPV types along with reported types of HPV16 and HPV18. Among HR-HPV types, HPV18 was found in 6.15%, HPV16 in 4.62%, HPV58 in 3.07%, HPV45 in 3.07%, HPV33 in 1.53%, andHPV68 in 1.53% (Figure 1A,B). In agreement with the previous reports from surrounding countries that have similar cultures, reported several high-risk HPV types including HPV31, 33, 39, 45, 51, 52, 58, 59,66, and 68.[14] In a comparable study in India, high-risk HPV type 16/18 was found in 7.9%.[15] However, the current study reported a lower rate of HPV may be because of unique morals, laws, and beliefs followed in this part of the world, which assist to lower the burden of the disease. Low-risk HPV11 was found to be common (6.15%) along with other low-risk HPV types including HPV6, HPV54, HPV55, and HPV61 in the study, this may be due to the poor living standard and hygiene that weaken the immune system and make the person susceptible to carrying the infection.[15]

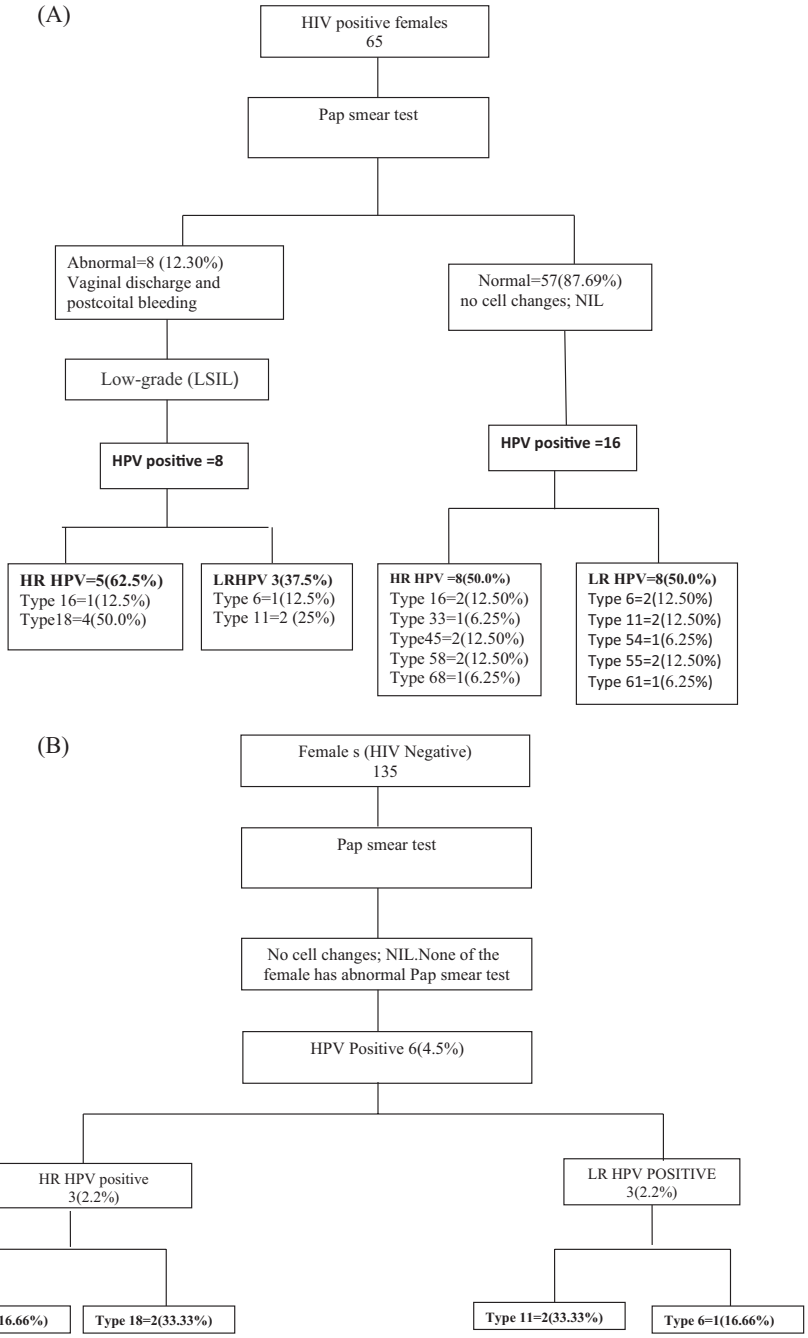


FIGURE 2 (A) HPV types in HIV-positive females with cervical Cytology. LSIL, Low-grade squamous intraepithelial lesion; NIL, Negative for Intraepithelial lesion. (B) Study diagram shows HPV types in HIV-negative females with cervical cytology.

In HIV patients with low-grade squamous intraepithelial lesions, HR-HPV accounts for 62.5%. HPV18 and HPV16 were detected in 50% and 12.5%, respectively of low-grade squamous epithelial lesion patients. HPV type 18 was predominant in consensus with other reports from the country[16,17] The prevalence of HPV 18 and HPV 16 differs from the studies reported on the general population in Pakistan because of the difference in the selection of the study population and the region within the country.[17] Similar to our observation Gul et al. reported a higher prevalence of HPV 18 15/33(45.5%) in squamous cell carcinoma patients and 7/18 (38.8%) in adenocarcinoma samples.[16] Moreover, the result of our study may not reflect the entire HIV population since the only federal area was covered. Studies reported that the distribution of HPV genotypes is heterogeneous, depending on the geographical region, social culture, and sexual habits. However, it is reported that HPV 18 was common in south East Asian countries.[18]

It has been observed that carcinogenic HPV infection predicts the progression of cervical dysplasia from normal to abnormal SIL.[19] The poor prognosis of HPV18 has been reported and is considered a transition to malignancy.[20,21] The reported data of HPV types show that HPV 16 is the most widely documented type followed by HPV 18 in patients with precursor lesions and carcinoma.[20] The low-risk HPV type was detected in 37.5% of this specific group. LR HPV11 was observed in 25% and LR HPV6 in 12.5%. The high incidence of HPV in HIV-positive females has a significant correlation. Because HIV damages the immune system and immunosuppression seem to

TABLE 2 Univariable logistic regression analysis of HPV positivity associated with different factors.

Variable	Odd ratio of HPV positivity	
	HIV-Negative	HIV-Positive
	OR (95% CI)	OR (95% CI)
Age (≤35 vs. >35)	1.34 (0.26–6.29)	1.21 (0.44–3.34)
Educational status (illiterate incomplete secondary education) vs. literate (secondary and higher education)	1.74 (0.31–9.83)	1.08 (0.37–3.15)
Marital status (single, widow and separated vs. married)	1.04 (0.18–5.88)	2.67 (0.79–9.02)
Parity (no. of pregnancies) (≤3 vs. >3)	0.60 (0.12–3.39)	0.91 (0.33–2.53)
Residence (rural vs. urban)	0.38 (0.07–1.95)	0.78 (0.25–2.43)
Other STDs (yes vs. no)	0.70 (0.14–3.86)	0.63 (0.24–1.69)
Use of contraceptives (yes vs. no)	1.25 (0.23–6.78)	1.90 (0.67–5.42)

favor the infection of HPV. Therefore, the higher prevalence may be because the immunocompromised HIV-infected women are unable to clear HPV infections easily.

Human papillomavirus infection has been linked to some risk factors. The prevalence of HPV DNA was higher in the age group ≤35 years, suggesting that they are sexually active and has exposure to HPV with an infected partner. Other studies also reported, an increased risk of HPV infection at age ≤35 years.[15] HPV infection prevailed 20% in HIV-negative and 50% in HIV-positive in single, widow, and separated females. Our findings were similar to the findings of Manhart et al. study, which reported that 10% of women with a single lifetime partner were infected with different high-risk HPV types.[22] Further, the predominant monogamous behavior in society may be a reason for the low report of HR-HPV prevalence in the population. The rate of HPV incidences among incomplete secondary education is higher as compared with secondary and higher education. This reinforces that knowledge gives better control over not only HPV infection but also other STDs.[23]

Human papillomavirus infection was observed more prevalent in patients with low $CD4^+$ count as compared with higher $CD4^+$count. HIV-infected females who have $CD4^+$ T Lymphocyte less than 200 cells/mm^3 have a threefold increased risk of contracting HPV infection.[24,25]

The present study reports HPV and its genotypes in HIV-positive females of the Pakistani population. Females infected with HIV are at a higher risk of developing HPV and other STD infections due to suppressed immune systems. It will help to inform health policymakers on HPV prevalence, and to develop a strategy for general screening of HPV for early detection of HR-HPV. These data also raise an awareness to educate the population, as well as to implement infection control measures and vaccination to reduce the burden of disease in the future.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

All data generated during the study are submitted in this article. The patient demographics are not presented publicly to maintain the privacy of research participants.

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