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Original Research Article

Prevalence of HPV Infection in Women at a Tertiary Care Hospital in Mumbai

Preeti U. Deshpande[1*], Preeti R. Mehta[2*], Shilpa C. Kerkar[3], Purva P. Sarkate[4*], Nayana A. Ingole[5*], Jayanti Mania-Pramanik[6**], Padmaja Samant-Mavani[5#], Urmila Parikh[7#]**

[1]Speciality Medical Officer, [2]Professor & Head, [3]Technician C, [4]Assistant Professor, [5]Associate Professor, [6]Scientist F, [7]Senior Scientific Officer,
[*]Department of Microbiology, Seth G.S Medical College and KEM Hospital, Mumbai.
[**]National Institute for Research in Reproductive Health, Mumbai.
[#]Department of Obstetrics and Gynaecology, Seth G.S Medical College and KEM Hospital, Mumbai.

Corresponding Author: Preeti U. Deshpande

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ABSTRACT

Background: Prevalence of Human Papilloma Virus (HPV), a known cause of cervical cancer has been reported to be more in HIV positive women. However, the HPV prevalence data in general population and HIV positive women in India is scarce. This hinders the development of policies regarding the screening, vaccination and follow- up of women for HPV infection

Aims: To estimate the prevalence of HPV in adult women and compare HPV prevalence in HIV infected and HIV non- infected women.

Methods: Women attending Gynaecology OPD with known HIV status were enrolled in this cross- sectional study. Their cervical specimens were collected for HPV DNA PCR and genotyping (HPV types 6, 11, 16 and 18). Statistical analysis was done using Chi- square/ Fisher's exact test.

Results: A total of 188 women were enrolled of which 38 were HIV positive and 150 were HIV negative. Of the total cervical specimens collected, 172 showed adequate DNA and HPV DNA PCR were positive in 15 of these samples (8.72%). HPV DNA positivity was 13.89% (5/36) in HIV infected and 7.35% (10/136) in HIV non- infected women (P=0.3147). High risk HPV genotypes 16/18 were found in 12/15 (80%) women.

Conclusions: Owing to the 8.72% prevalence of HPV, there is a need to screen all adult women for early detection of HPV infection and HPV vaccination should also be considered.

Key words: HPV, HIV, cervical cancer, screening, vaccination.

INTRODUCTION

Despite the high incidence of cervical cancer reported from India, large scale population based studies on the HPV prevalence and genotype distribution are few from this region. Since geographical variation may exist, knowledge about the distribution of HPV types circulating in the communities in different regions of India would be useful in understanding the prevalence of HPV genotypes and devising the optimum strategy for effective vaccination in India. [1-4] Hence, a study was carried out to estimate the prevalence of HPV in adult women in a tertiary care hospital setting and to compare HPV prevalence in HIV infected and HIV non-infected women.

MATERIALS AND METHODS

After Institutional Review Board approval, a cross- sectional study was carried out over a period of one year at a tertiary care teaching hospital in Mumbai. Adult women (≥18 years) attending gynaecology OPD referred for Pap smear were included in the study provided their HIV test report was known. Pap smear is a

screening test and is routinely recommended for all sexually active women attending the gynaecology OPD. Women who were pregnant or those having undergone pan-hysterectomy were excluded. Depending on their HIV report, the study participants were divided into two groups: HIV infected and HIV non- infected women. The sample size for both the groups was set at 150 with a precision of 5% and 95% confidence interval .

The selected participants' socio-demographic information and relevant sexual behaviour alongwith relevant clinical history was documented. These included their name, age, religion, education, socioeconomic status, marital status, age at marriage, parity, duration of sexual life, lifetime sexual partners, regular condom use, HPV vaccination status; ART status and CD4 counts in case of HIV positive women. The socio economic status was determined based on colour of the ration card that the women possessed in accordance with the Controller of Rationing & Director of Civil supplies (Govt. of Maharashtra). [5]

Cervical specimens were collected under aseptic precautions using a sterile cyto-brush, Hi-Cytobrush™ (HiMedia, India) for HPV DNA testing along with Pap smear using a wooden Ayre's spatula. Collected cervical cytobrush specimens were stored in a sterile tube containing 0.1 M Phosphate buffered saline (PBS) till DNA extraction. Pap smears were examined by cytopathologists and their results were noted.

DNA was extracted from the cervical specimens using modified non-enzymatic method. [6,7] The concentration of DNA was measured by using spectrophotometer (NanoVue Plus, GE Healthcare Life Sciences, USA). The quality and integrity of the extracted DNA was determined by PCR using primers directed towards detecting the β-globin gene segment. Once a band was seen for β-globin gene (200 bp), that specimen was tested for presence of HPV DNA using suitable degenerate primers (Forward: 5' CGT CCA AAA GGA TAC TGA TC 3' and Reverse: 5' GCA CAG GGA CAT AAC AAT GG 3'). [7] The PCR products obtained were analysed and compared with 1 kb universal ladder. If positive for HPV infection, a 450 bp product was obtained after amplification. [Figure 1] During the process of Southern blotting, DNA fragments were transferred from the electrophoresis gel to a membrane support and cross linked using UVC 500 UV crosslinker, GE Healthcare Life Sciences, USA. Digoxigenin (DIG), a steroid hapten was used to label DNA on genomic Southern blots for hybridisation and subsequent detection using PCR DIG Probe Synthesis Kit, Roche Diagnostics. The success of the labelling reaction was checked by Dot blot by using reagents provided in the DIG luminescent detection kit, Roche Diagnostics. Immunodetection of the hybridised probe was determined with enzyme conjugated anti-digoxigenin antibody and a chemiluminescent substrate. Signals in the form of spots were present in the slots, indicating positive result. If there were no spots, the result was negative. [Figure 2]

Specimens having a positive result for HPV- DNA by PCR were further subjected to hybridisation and immunodetection using type specific probes to determine the 4 HPV genotypes 6, 11, 16 and 18 which are present in the available licensed vaccines in India.

Type specific probes used to determine the high risk and low risk HPV types were as follows: [7]

HPV 16: 5'- CAT ACA CCT CCA GCA CCT AA- 3'

HPV 18: 5' CGA TGC TGC ACC GGC TGA- 3'

HPV 6: 5'- CAT CCG TAA CTA CAT CTT CCA- 3'

HPV 11: 5' TCT GTG TCT AAA TCT GCT ACA- 3'

These DIG labelled probes were synthesized in- house using DIG oligonucleotide 3' end labelling kit, Roche diagnostics, Indianapolis, USA. Detection

was carried out using the DIG luminescent detection kit, Roche Diagnostics, Indianapolis, USA.

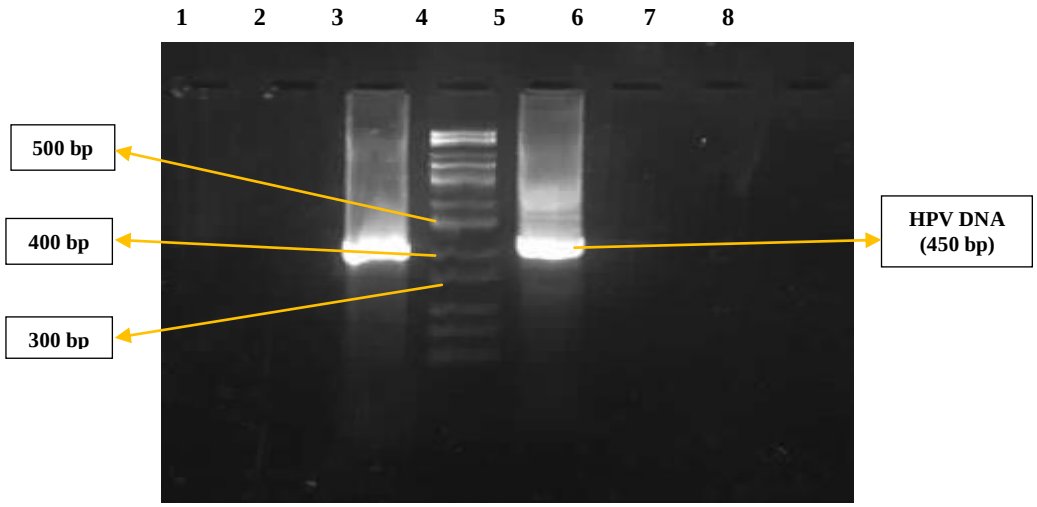

Figure 1: HPV DNA detection [Lanes 3 and 5 show presence of HPV DNA (450bp), Lane 4 is 1 kb DNA ladder

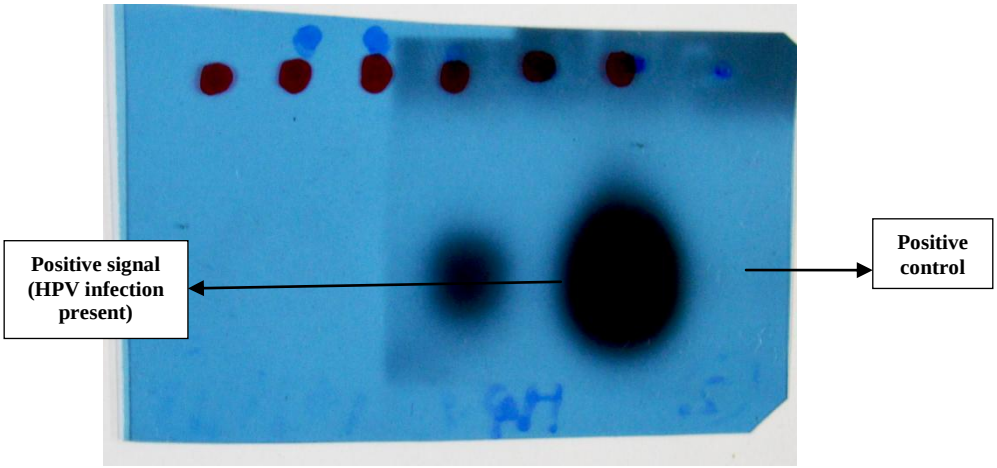

Figure 2: Immuno-detection of hybridised probes on X-ray film

Statistical methods

Statistical analysis was done using Open Source Epidemiologic Statistics for Public Health/OpenEpi, Version 2.3.1., applying Chi- square or Fisher's exact test. P-value < 0.05 was considered significant

RESULTS

A total of 252 women agreed to participate in the study. However, HIV status was known in only 188(74.6%) women who were enrolled after obtaining their written informed consent. Of these, 38 (20.21%) were HIV positive and 150 (79.79%) were HIV negative. Adequate quantity of DNA by Beta glob in estimation was observed in 172/188 (91.49%) specimens. Of the 172 women screened for presence of HPV DNA, 36 (20.93%) were HIV positive and 136 (79.07%) were HIV negative.

The overall HPV positivity rate was 8.72 % (15/172). There was no significant difference in HPV positivity in HIV positive (13.89%) and HIV negative group (7.35%) (P=0.3147). All HIV infected women were on highly active Anti-retroviral Therapy (HAART). The mean CD4 count of HIV positive women (n=36) was 559.78 (56.74-1062.82) cells/mm^3. Of these, HPV positive women (n=5) had mean CD4 count of 393 (139.68-646.32) cells/mm^3 and HPV negative women (n=31) had mean CD4 count of 586.68 (71.76- 1101.60) cells/mm^3

however the difference was not statistically significant (P=0.1380).

HPV 16 was found in 10/15 women (66.67%), HPV 18 in 7/15 (46.67%), HPV 6 in 1/15 (6.67%) and HPV 11 in 14/15 (93.33%) women. High risk HPV genotypes 16/ 18 were found in 12/15 (80%) women. Multiple genotypes were seen in both HIV infected and HIV non infected women (P=0.2418).

The HPV positivity was significantly higher in widowed/ separated/ single women as compared to married women (P=0.0291) [Table 1]. HPV positivity was highest (22.73%) amongst women who had no children (P=0.0436). There was no statistically significant difference observed between HPV positive and negative women based on their mean age, educational background, socio-economic status, age at first intercourse, duration of sexual life and regular use of condoms [Table 1, 2]. The Pap smear findings in HPV positive and HPV negative groups did not differ significantly [Table 3].

Table 1: Socio- demographic variables of women (n=172)

Factor	No.	HPV Negative n (%)	HPV Positive n (%)	P value
Age				
18-30	60	55 (91.67)	5 (8.33)	0.8921
31-40	71	64(90.14)	7 (9.86)	
>40	41	38 (92.68)	3 (7.32)	
TOTAL	**172**	**157**	**15**	
Religion				
Hindu	156	143 (91.67)	13 (8.33)	0.6344
Muslim	16	14 (87.50)	2 (12.50)	
TOTAL	**172**	**157**	**15**	
Education				
Illiterate	36	33 (91.67)	3 (8.33)	0.5142
Upto Secondary	108	100 (92.59)	8 (7.41)	
Higher sec or above	28	24 (85.71)	4 (14.29)	
TOTAL	**172**	**157**	**15**	
Economic status				
White	75	68 (90.67)	7 (9.33)	0.8257
Saffron	80	74 (92.50)	6 (7.50)	
Yellow	17	15 (88.24)	2 (11.76)	
TOTAL	**172**	**157**	**15**	

Table 2: Sexual history of women (n=172)

Factor	No.	HPV Negative n (%)	HPV Positive n (%)	P value
Marital Status				
Married	157	146 (92.99)	11 (7.01)	0.0291
Widowed/Separated/Single*	15	11 (73.33)	4 (26.67)	
TOTAL	**172**	**157 (91.28)**	**15 (8.72)**	
Parity				
No children	22	17 (77.27)	5 (22.73)	0.0436
1 child	40	37 (92.50)	3 (7.50)	
≥2 children	110	103 (93.64)	7 (6.36)	
TOTAL	**172**	**157**	**15**	
Age at 1st intercourse (years)				
≤19	102	92 (90.20)	10 (9.80)	0.5953
>19	70	65 (92.86)	5 (7.14)	
TOTAL	**172**	**157**	**15**	
Duration of sexual life (years)				
≤ 5	27	23 (85.19)	4 (14.81)	0.6319
6-10	34	31 (91.18)	3 (8.82)	
11-15	35	33 (94.29)	2 (5.71)	
>16	76	70 (92.11)	6 (7.89)	
TOTAL	**172**	**157**	**15**	
Regular Condom Use				
Yes	90	82 (91.11)	8 (8.89)	1
No	82	75 (91.46)	7 (8.54)	
TOTAL	**172**	**157**	**15**	
HIV Status				
Positive	36	31(86.11)	5 (13.89)	0.3147
Negative	136	126 (92.65)	10 (7.35)	
TOTAL	**172**	**157**	**15**	

*12 women were widowed, 2 were separated and 1 was single

Table 3: Pap smear findings and association with HPV (n=172)

Pap smear findings	HPV Positive n(%)	HPV Negative n (%)	Total	P-value
NILM*	14 (8.28%)	155 (91.72%)	169	0.2408
ASCUS†	1 (33.33%)	2 (66.67%)	3	
TOTAL	15	157	172	

*NILM = No evidence of Intraepithelial Lesion or Malignancy
†ASCUS= Atypical Squamous Cells of Undetermined Significance

DISCUSSION

Despite having the third largest burden of HIV-infected individuals and one-fourth of the global burden of cervical cancer, very few studies have addressed HPV prevalence, genotype distribution and cervical cancer prevalence in Indian women. [8,9] Considering this, our study was conducted to know the prevalence of HPV in adult women and comparison of HPV types was made between HIV positive and negative women. Though 252 women agreed to participate in the study, only 188 (74.60%) could be enrolled as the others were not aware of their HIV status as they were not tested. Integrated Counselling & Testing Centre (ICTC) and Prevention of Parent to Child Transmission (PPTCT) programme are important components for HIV / AIDS care, prevention and control. The testing for HIV is client initiated at the centre where the client can opt out from the testing. [10] The prevalence of HIV in Mumbai is reported to be more than 1% in the general population. [11] Moreover, known HIV infected women are not routinely referred to gynaecology OPD. No special efforts were made to enrol known HIV infected women.

In the current study, overall HPV prevalence was 8.72%. Similar findings (6.7%-10.4%) have been reported by other authors from India and different parts of the world. [4,12-14] However few studies are available from India defining the prevalence of HPV in women in general population. [8] In countries like Australia, UK, Sweden, Canada and USA, universal vaccination for HPV is recommended to all adolescent girls and women even though the prevalence of cervical cancer in them is similar to that of ours. [15] Hence, India should seriously consider including HPV vaccination in the Universal Immunisation Programme (UIP) in India. This can be linked and delivered through the strategic approach to Reproductive, Maternal, Newborn, Child & Adolescent Health (RMNCH+A) programme under the Ministry of Health and Family Welfare, Government of India. [16]

The primary immune response to HPV infection is cell mediated; therefore, conditions that impair cell-mediated immunity such as HIV infection increase the risk of acquisition and progression of HPV. [17-21] It has been reported that HIV-HPV co-infection leads to an increased risk of precancerous cervical lesions and a more rapid progression to cervical cancer. [22] HIV infected women are two to five times more likely to manifest cervical cancer, the incidence of HPV infection being as high as 95% in HIV positive women, compared with 22% in HIV negative women. [23,24]

Studies, both global and Indian have reported higher prevalence of HPV in HIV infected women. [12,14,22,25] However, in the current study, the correlation between HIV and HPV positivity was not significant and the results do not correlate with most of the reported studies. It has been reported that infections associated with HIV are pronounced when CD_4 count drops below 500 cells/mm^3 and this might possibly be one of the reasons of not having significant HPV positivity in HIV infected women in the current study as all the HIV positive women were on Highly active anti-retroviral therapy (HAART) and their mean CD_4 count was 559.78 cells/mm^3. [26]

Knowledge of the pattern of HPV type distribution of each region is useful for public health policy decisions concerning HPV vaccination and screening in the prevention and control of cervical cancer. [8] In the current study, most common high risk type found was HPV 16 in 10/15 (66.67%) of women. *Sarkar et al* (2011) and *Joshi et al* (2014) have reported similar findings. [8,12] In the current study, 80% of HIV positive women were also infected by High risk HPV type 16 & 18. *Rocha et al* (2014)

has reported that non-16 and non-18 high risk HPV genotypes are more commonly seen in HIV positive women. [14] However, these were not looked for in the current study. Also, infection with multiple types of HPV has been reported which can lead to increase in severity of cervical disease. [14] In the current study, non high risk HPV type 11 was seen in 93.33% of women and infection with multiple HPV types was seen in 80% women. There was no statistically significant difference between HIV infected and non-infected women. This could possibly be due to the inadequate sample size of HIV positive women in the current study.

The greatest risk of HPV infection coincides with greatest metaplastic activity which occurs at puberty and first pregnancy and declines after menopause. [20] HPV infection is most common in sexually active young women, 18-30 years of age. [20] There is a sharp decrease in prevalence after 30 years of age. However, cervical cancer is more common in women older than 35 years, suggesting infection at a younger age and slow progression to cancer. [20,21] In the current study, mean age of the HPV positive women was 36.93 years. It was observed that prevalence of HPV showed a rising trend till the age of 40 years after which rate of HPV infection decreased though this difference was not statistically significant (P=0.8921). [Table 1] *McDonald et al* (2014) has reported similar findings. [27] This might be because in immuno-competent women, HPV infection following first sexual exposure is cleared off with increasing age probably due to the development of acquired immunity to HPV infection following repeated exposure as proposed by *Sarkar et al* (2011). [12] Age wise difference was also not seen in a study conducted in Brazilby *Rocha et al* (2014). [14] However, it should be noted that in the current study as well as most other studies, study participants included were primarily married women. This is because in India, genital tract sampling of an unmarried woman is associated with cultural and social taboo. [12]

Worldwide, it has been reported that women of low socioeconomic status have a greater risk of having cervical cancer. [28] The risk of contracting genital HPV infection and cervical cancer is influenced by sexual activity. [29] The risk further increases with multiple sexual partners and sexual activity at an early age. Condom usage may not adequately protect individuals from exposure to HPV since HPV can be transmitted by contact with infected labial, scrotal or anal tissues that are not protected by a condom. [30] Also, multiple pregnancies is a significant independent risk factor among women with histopathological evidence of HPV infection. [30] In the current study, there was no correlation observed between HPV positivity and educational background, socioeconomic status, age at first intercourse, duration of sexual life and regular use of condoms. [Table 1, 2] Similar findings have been reported by *Rocha et al* (2014) and *Sarkar et al* (2011). [12,14] Although majority of women in the present study were married, a significant association was found between HPV positivity in widowed/ separated/ single women as compared to married women. [Table 2]This was because all of the widowed women were HIV positive and 3/12 gave history of having multiple sexual partners either prior to or after marriage. As a result, they had higher risk of exposure to sexually transmitted HPV.

A significant association was found between HPV positivity and nulliparous women (P=0.0436). [Table 2] However, most of the studies have reported positive or no association between multiparity and HPV positivity in contrast to the current study. [12,27] This difference in the current study could have been so because most of the nulliparous women were also widowed or separated.

There was no significant difference observed between Pap smear findings of HPV positive and negative women in the

current study. [Table 3] This might be because majority of women in the study population belonged to a relatively younger age group whereas cervical cancer is usually reported more commonly in women older than 35 years of age. [20,21]

The cross-sectional design of the current study did not lead to determining a causal relationship between the immunodeficiency caused by HIV and/or the prevalence and persistence of HPV and is a limitation of the study. Moreover, the genotyping was restricted to detection of HPV types 6, 11, 16 and 18 only.

CONCLUSION

Apart from the limitations, prevalence of HPV was considerably found to be 8.72% which indicates that measures need to be taken either in the form of early detection or inclusion of HPV vaccination in the Universal Immunisation Programme (UIP) in India.

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