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Research Article

HPV PREVALENCE IN ORAL CAVITY OF WOMEN WITH CERVICAL LESIONS AND ORAL SEX PRACTICES

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ABSTRACT
This investigation aimed to assess the prevalence of Human Papillomavirus (HPV) in the oral cavity of women with oral sex practices and cervical lesions. A questionnaire regarding oral sex practices was administered to 92 nonsmokers and non-alcoholics previously diagnosed with cervical intraepithelial neoplasia (CIN) within the past six months, followed by swab collection from the cheek and palate/gum for PCR analysis of HPV16, HPV18, and generic HPV. Results indicated that 73% of participants reported regular oral sex practices, with all showing positive HPV results in their oral mucus or palate. Overall, 35% had HPV16, with 27% of those with regular oral sex practices reporting frequent engagement and 9% reporting none. Oral HPV16 positivity correlated with advanced cervical CIN lesions. HPV18 was not detected, with a greater prevalence of HPV16 in buccal mucosa compared to palate/gum (23%).

Keywords: Cervical lesions, Oral sex practices, HPV, Oral cavity, PCR analysis.

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INTRODUCTION

Human papillomaviruses (HPVs) are a family of small (55 nm) icosahedral, non-enveloped virus with a circular double-stranded DNA genome of 7-8 kbp and with a special affinity for epithelial cells. Over 200 genotypes of papillomaviruses infect the skin and mucosal surfaces [1,2]. The most common oncogenic HPV are associated with leukoplakia and squamous carcinoma. While the majority of the HPV types have affinity to grow on skin, oral lesions, genitals, anal and larynx [3,4]. A number of HPV types are considered high risk, including 16, 18, 31, 33, 35, 39, 45, 52, and 58, which are necessary for the development of cervical cancer. Throughout the world, cervical cancer is a major public health issue; it is the most common cancer among women and the leading cause of cancer-related deaths [5,6]. Squamous cell carcinoma of the mouth has been linked to HPVs that are potentially oncogenic. Papillomavirus may be transmitted from the genital zone to the oral cavity through orogenital contact. HPV infections in the mouth are associated with age but are different from cervical infections [7,8]. In other studies, the Papanicolaou technique and cytological examinations have been used to detect HPV in women with genital HPV in the oropharynx. Researchers have discovered a potential natural reservoir of HPV outside the genital region, which may serve as a source of reinfection [9]. As well as this, even when tampons or digital penetration are used, HPV is rarely found in the vagina of virgin women. Cervical HPV infections can be translocated to oral HPV infections, despite their being recognized as a HPV-associated oral malignancy. Preventing oral mucosal infections with condoms and preventive vaccinations.

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A single oral sample was used to evaluate HPVs. [10, 11] HPV has been detected primarily in the tonsil and oropharynx in cancers of the upper aerodigestive tract. Our study found that the number of sexual partners did not seem to increase the risk of HPV-16 oral infection, although it is known that an increase in the number of sexual partners can lead to HPV infection in the cervix [13]. Both oral and cervical sites may be affected by oral sex practices. Infections caused by HPV at the cervix may be affected by local factors that have been found to affect their persistence. A number of factors contribute to this risk, including Chlamydia trachomatis or herpes simplex infection, smoking, age, HPV type, and hormones and contraceptives use. A questionnaire was not used in any of these reports to determine how HPV is transmitted. For this study, the cunnilingus and the incidence of mouth HPV infection were examined based on the sexual behaviors of the patients [14]. A significant association was found between HPV16 infections in mouth and the progression of genital CINs (Mann Whitney U test, p = 0.023), suggesting that women who developed persistent HPV16 infections and progressed to advanced genital lesions may be at a greater risk of HPV16 infection in mouth. We found HPV16 infection in a media of 37 years. Due to the cancer-related nature of these HPV types, dentists and pathologists should be aware of the fact that they are high-risk mucosal types of viruses. Previously, we discussed the possible role of men as HPV vectors [15]. HPV can be transmitted by oral contact as well as by sexual contact, according to our research. Women who refused to receive cunnilingus were HPV16 free, which suggests that their partner did not translocate the virus from the genital area to the mouth, but autoinoculation occurs more frequently than we realize [16].

METHODS

Patients and sampling

Summer was the time for this study to be conducted. A prior diagnosis of CIN was made by all women between six months prior to the study. A woman who attends the Dysplasia Clinic for any cervical problem, regardless of where she lives, must sign an informed consent and complete a questionnaire about her sexual habits to be in the study. A smoker and an alcoholic were excluded from the program. All women studied were above eighteen years old. To collect auto sampling, women were asked to swab their cheeks with cotton swabs and their palates/gums with another cotton swab. A minute was rubbed on tissues in both cases. After collection, cotton swabs were immersed in a 15 mL tube that contained 1 mL of transporting media (10 mM Trizma, pH 8.8, 1 mM EDTA; 0.01% sodium azide; 50 ng/mL ampicillin; 1 ng/mL proteinase K) and stored at -20° C between 24-hour intervals.

PCR for HPV samples was performed by centrifuging the samples at 3,500 rpm in a clinical centrifuge, removing the cotton swabs and centrifuging at 3,500 rpm for 5 minutes at 4°C. A new tube was filled with 300 mL of clear supernatant, and 25 mL of 5 M sodium acetate and 1 mL of isopropanol were added sequentially [17]. The mixture was centrifuged for five minutes at 14,000 rpm at 4° C. 70% ethanol was used to wash the pellets, and they were dried overnight at room temperature. An incubation period of 20 minutes at 65°C was performed with dissolved pellets in a 100-liter rehydration solution (Promega A7963).

5L DNA was used for the PCR, 12.5L 2X GoTaq Green Master Mix (Promega) for the PCR for the generic HPVs. In 5 liters of water, mix 2.5 MY11 and MY09 primers. We examined PCR products on two percent agarose gels using standard pairs base (Promega G7521) after 5 minutes of 94°C denaturation, followed by 7 minutes of 72°C extension, followed by 40 cycles of 94°C, 55°C, and 72°C. A human beta-globin gene primer and a human GH20 primer were used as internal controls. As described elsewhere, specific primers were used to detect HPV16 and HPV18.

RESULTS

A total of 86 women who participated in this study were non-smokers and non-alcoholics. Due to previous CIN alterations, female patients attend this clinic. The patients were instructed by imitation to auto-sample the oral cavity after they had completed the informed consent and questionnaire. To ensure the quality of PCR and to act as an internal control of human DNA, the human b-globin gene was amplified once in the laboratory. A wide range of HPV types were amplified with generic HPV rather than those that failed in the b-globin amplification. In the event that the generic HPV test was positive, the specific PCRs for HPV16 and HPV18 were conducted. Buccal mucosa was the first region sampled, followed by palate and gum (P/G) together. There was a prevalence of 100% for buccal cavity HPV in all women studied, either in mucosa or in P/G. According to Table 1, if mucosa and P/G are considered separately, the percentages of HPV infection are as follows: 86% for generic HPV in the buccal mucosa, 88% for P/G, and 23% for HPV16 in the mucosa and 16% for P/G. HPV16 is more common among women who have oral sex, but the number of cases is too small to see any significant risk. Study subjects were 63 percent married, 19 percent in common law marriages and 7% single. In the tables, the range of ages is not shown. However, the mean age was 35 years old (not shown in the tables). As a result of the questionnaire applied, the results are shown in Table 2. Both palate-gum and buccal mucosa were affected by generic HPV in all cases. 35% of all patients were positive for HPV16,

while 73% reported frequently practicing oral sex. It was not observed that HPV16 prevalence was associated with frequent oral sex practice. The total number of women who had sexual relations with each other was 53%. There is a higher likelihood of HPV16 infection among patients who practice oral sex (53%). It is interesting to note that the only three women (7%) who practiced fellatio but did not receive cunnilingus were generically positive, but did not test positive for HPV16. Consequently, all women were diagnosed with CIN within the past six months (according to Bethesda classification); oral HPV16 positivity appears to be associated with CIN progression in 51% of cases (Mann Whitney U test, p = 0.023); inflammatory changes were present in 28%; and there were no cervical changes in 21% of cases (Table 2). At the time of the gynecological visit, we observed two women who responded positively to the treatment. HPV16 was positive, but they did not have any cervical alterations. 60 percent of the patients said they used no condoms while practicing oral sex, and the majority did not use condoms while using oral sex. According to Table 2, condom use during oral sex prevents infection of the mucosa (Table 2). (Table 2) revealed that 47 percent of patients have only one sex partner, 23% have two partners, and 2% have more than two.

It was reported by 53% of the women that they did not share spoons, toothbrushes, or candy (Table 2). A significant fraction of those surveyed (40%) acknowledged sharing those objects occasionally (Table 2).

Table 1: Relationship between HPV prevalence and oral sex practices by oral region

Overall (N = 86)	Oral Sex	Genetic HPV		HPV16	
Buccal mucosa		n	(%)	n	(%)
	No	22	27	6	8
	Yes	52	61	14	17
	Total	74	88	20	25
Palate/gum		n	(%)	N	(%)
	No	18	22	2	3
	Yes	58	68	12	15
	Total	66	90	14	18

Table 2: Oral sex practices are associated with buccal HPV16.

	Over all (N=86)		HPV16 Negative (N=56)		HPV16 Negative (N=30)	
	n	%	n	%	n	%
Oral Sex frequency						
Never	24	29	16	28	8	28
Frequently	62	73	40	72	22	74
Oral Sex type						
Do not practice	2	3	2	5	0	0
To her partner	6	6	6	12	0	0
Both	46	54	30	55	16	54
Did not answer	32	38	18	33	14	48
Histological biopsy results						
Without alterations	18	22	14	26	4	14
Inflammatory Alterations	24	29	18	33	6	21
CIN-I & CIN-II	44	52	24	44	20	68
Use of condom while practicing oral sex						
Do not practice oral sex	24	29	16	28	8	28
Use of Condom	2	2	2	5	0	0
Do not use Condom	50	59	32	58	18	61
Did not answer	10	13	6	12	4	14
Number of Partners to whom practice oral sex						
Do not practice oral sex	2	2	2	5	0	0
One	40	48	24	44	16	54
Two	20	24	14	26	6	21
More than two	2	2	2	5	0	0

Did not answer	22	27	14	26	8	28
Personal objects sharing						
Do not practice	46	54	34	62	12	41
Occasionally	34	41	18	33	16	54
Frequently	4	6	2	5	2	8
Did not answer	2	2	2	5	0	0

DISCUSSION

Several factors affect the prevalence rate of HPV in normal oral mucosa, including sample types, methods of collection and detection, sensitivity level, PCR primers used, and PCR inhibitors. Healthy adults were found to have an 81 percent prevalence of oral HPV infection in a previous study. This study used the MY09/MY11 primer pair for HPV detection by PCR, which is widely used in epidemiological studies and has been shown to be similar in sensitivity to confirmatory nested PCR using GP5+/GP6+ primers with 94% correlation [18]. A 100% frequency of generic HPV was found in our sampled population.

Only oral samples were evaluated for HPVs. The oropharynx and tonsil are the most common sites of HPV detection in cancers of the upper aerodigestive tract. We found that the number of sexual partners does not increase the risk of HPV-16-infected oral cavity or of HPV-16-infected cervix in our study. Sexual partners are a risk factor for HPV-16-infected oral cavity, but not for HPV-16-infected normal cervix. The oral and cervical sites can be affected in a similar manner by oral sex practices. The natural history of oral HPV infections may be influenced by local factors that influence HPV persistence at the cervix. In addition to coinfections with Chlamydia trachomatis and herpes simplex virus, smoking, age, HPV type, and hormone and contraceptive use are all significant risk factors.

It should be noted, however, that none of these studies utilized any questionnaire to identify the factors that contribute to HPV transmission. The relationship between cunnilingus and mouth HPV infection has been examined by collecting information about sexual behavior of patients. A significant association was observed between HPV16 in mouth and the progression of genital CIN (Mann Whitney U test, $p = 0.023$), suggesting an elevated risk of HPV16 detection in the oral mucosa in women with persistent HPV16 infections and advanced genital lesions. A 37-year-old man was infected with HPV16. It is regarded as a mucosal type virus due to its association with cancer; and dental and pathological professionals should be aware of this phenomenon [19].

In the past, we have discussed the possibility of men acting as HPV vectors. The results of our study suggest that HPV can be transmitted orally as well as sexually. It is probably not their partner that transfers the virus from their genitals to their mouth, but probably autoinoculation is more prevalent than we realize. Women who failed to receive cunnilingus were HPV16 free; this may reflect that their partner is not transmitting the virus from women's genitals to their mouths. Therefore, oral contact with genitalia as well as autoinoculation should explain the presence of the virus in the oral cavity. Different populations and methodologies contribute to different prevalence data in oral HPV DNA detection. Therefore, parallel studies on the relationship between incident squamous epithelial lesions and persistent oral HPV will require comparative studies. By understanding the role of HPV in oral cancer development and cervical infection in the oral cavity, we will be able to better understand how HPV is involved in oral cancer development.

CONCLUSIONS

In this study, it was concluded that women with CIN who had genital HPV infection had HPV infection in their mouths as well, and that HPV16 detection in the mouth could be an indicator of CIN persisting and progressing.

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