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REVIEW

The impact of smoking on HPV infection and the development of anogenital warts

Reto Kaderli · Beat Schnüriger · Lukas E. Brügger

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Abstract
Purpose The worldwide prevalence of human papillomavirus (HPV) infection is estimated at 9–13 %. Persistent infection can lead to the development of malignant and nonmalignant diseases. Low-risk HPV types are mostly associated with benign lesions such as anogenital warts. In the present systematic review, we examined the impact of smoking on HPV infection and the development of anogenital warts, respectively.
Methods A systematic literature search was performed using MEDLINE database for peer-reviewed articles published from January 01, 1985 to November 30, 2013. Pooled rates of HPV prevalence were compared using the χ^2 test.
Results In both genders, smoking is associated with higher incidence and prevalence rates for HPV infection, whereas the latter responds to a dose-effect relationship. The overall HPV prevalence for smoking patients was 48.2 versus 37. 5 % for nonsmoking patients ($p<0.001$) (odds ratio (OR)=1.5, 95 % confidence interval (CI) 1.4–1.7). Smoking does also increase persistence rates for high-risk HPV infection, while this correlation is debatable for low-risk HPV. The incidence and recurrence rates of anogenital warts are significantly increased in smokers.
Conclusions Most current data demonstrate an association between smoking, increased anogenital HPV infection, and development of anogenital warts. These data add to the long list of reasons for making smoking cessation a keystone of patient health.

Keywords Smoking · Human papillomavirus · Anogenital warts

R. Kaderli · B. Schnüriger (✉) · L. E. Brügger
Department of Visceral Surgery and Medicine, Bern University Hospital, University of Bern, 3010 Bern, Switzerland
e-mail: beat.schnuriger@gmail.com

R. Kaderli
e-mail: reto.kaderli@insel.ch

L. E. Brügger
e-mail: lukas.bruegger@insel.ch

Introduction

According to the World Health Organization (WHO), 630 million people are infected with genital human papillomavirus (HPV), resulting in an estimated worldwide prevalence of 9–13 % [1, 2]. HPV is transmitted via genital contact and is the most common sexually transmitted infection worldwide [3, 4]. There is no consensus on a gender-specific risk of acquiring HPV [5]. Whereas at least 40 % of HPV infections are asymptomatic and transient, with subsequent clearance by the immune system, some infections persist [2, 5]. Persistent infection can lead to the development of malignant and premalignant or nonmalignant diseases, so-called "anal intraepithelial neoplasia" (AIN). Over 100 HPV types have been identified. Approximately 40 of these affect the anogenital region [2, 6]. HPV infections are detected by collecting material with brushes or swabs, followed by polymerase chain reaction analysis, genotyping, and HPV classification according to their risk of causing cervical cancer [2]: Low-risk HPV types are mostly associated with benign lesions such as anogenital warts (AIN grade I), and high-risk or oncogenic types are also associated with cancers and their precursors (AIN grade II/III) [6]. More than 90 % of anogenital warts are caused by low-risk HPV types 6 and 11 [7, 8]. Infected basal cells move toward the surface layer, where they ultimately develop to anogenital warts [2]. The worldwide prevalence of visible anogenital warts ranges from 0.13 to 5.1 % [9]. These may regress spontaneously, with reported

clearance rates varying widely from 0 to 55 % [10-12]. However, even with therapy (topical, surgical, and/or destructive), the recurrence rate within 3 months is 25–67 % [10].

Apart from genital contact, risk factors for HPVacquisition include younger age, early coitarche, number of lifetime sexual partners, failure to use condoms consistently, marital status, history of sexually transmitted infections, immunosuppression (including patients with human immunodeficiency virus), history of other HPV-mediated neoplasia, and low socioeconomic status [13, 4]. Furthermore, smoking is postulated to be an additional risk factor, due to increased susceptibility to the acquisition of HPV [14].

In the present systematic review, we examined the impact of smoking on the incidence, persistence, and prevalence of HPVand of anogenital warts.

Methods

A systematic literature search was performed using MEDLINE database for peer-reviewed articles published in English. The search was carried out for articles published from January 01, 1985 to November 30, 2013, using the following keywords: smoking AND human papillomavirus AND/OR incidence AND/OR prevalence AND/OR persistence AND/OR condylomata acuminata AND/OR anogenital warts AND/OR genital warts. The references in the identified articles were also reviewed. Titles and abstracts, when available, were scrutinized to select relevant studies addressing the relation of smoking and HPV infection and/or the occurrence of anogenital warts. All studies addressing the relation in the abstract and/or the full text manuscript were subsequently included. Case reports, letters to editors, and review articles were excluded. In addition, studies restricted to patients with cervical or AIN and cervical or anal cancer were excluded. Furthermore, studies examining HPV infection or warts in other regions than the genital or anal were excluded.

Pooled rates of HPV prevalence were compared between smoking and nonsmoking patients, using the χ^2 test. For this analysis, only prospective studies were included. As time intervals for incidence and persistence were different between studies with an analysis of different HPV types, comparison of these pooled rates was not practicable. To assess the clinical significance of differences in prevalence rates between smoking and nonsmoking patients, the 95 % confidence interval (CI) for the difference was derived. Statistical Package for Social Sciences (SPSS Windows) version 21.0 (SPSS, Chicago, IL) was used for all analyses.

Results

Incidence of HPV

Fifteen studies (14 prospective and one retrospective) are available on the impact of smoking on the incidence of HPV [15-29]. The retrospective study—including 1,880 patients—did not reveal an association [17]. Of the 14 prospective studies, 12 investigated genital [15, 16, 18-26, 29], one anogenital [27], and one solely anal [28] HPV infections. Nine of these 14 studies demonstrated a significant [15, 16, 18, 19, 21, 22, 24, 26, 27], one a nonsignificant [20], and four no association between HPV incidence and smoking (Table 1) [28, 30, 25, 29].

A gender-specific analysis revealed that three out of four prospective studies including a total of 5,376 male patients found a significant association between smoking and the incidence of HPV [16, 27, 21]. Only one prospective study with 374 male patients did not reveal an association [23]. Similarly, six out of ten prospective studies comparing 27,508 smoking and nonsmoking female patients found a significant association [15, 18, 19, 22, 24, 26]. Additionally, Oh et al. found a statistically nonsignificant trend toward an association among female patients [20]. Three remaining prospective studies, including 1,339 female patients, did not reveal an association [28, 25, 29].

Whereas the above-mentioned studies focused on the association of HPV incidence to current smoking, Partridge et al. found an association solely between past smoking and HPV infection (hazard ratio (HR)=1.6, 95 % CI 1.1–2.4) [21].

Persistence of HPV

Thirteen prospective studies including a total of 18,529 patients investigated the impact of smoking on the persistence rate of anogenital HPV during time intervals of 4 [31], 6 [27, 32-34, 23, 35, 16], or more [36, 37, 20, 38] months (one missing specification of time interval [39]). Nevertheless, the association between smoking and the persistence of HPV is not conclusive with regard to the number of patients in the two groups: Eleven of these 13 studies, including a total of 10,503 patients, found a statistically significant association (seven studies [27, 32, 33, 36, 31, 39, 38]) between HPV persistence and smoking or a trend (four studies [16, 20, 34, 23]) toward higher rates among smokers compared to nonsmokers [16, 27, 32, 33, 36, 31, 20, 34, 23, 39, 38]. Maucort-Boulch et al. even found a dose-effect relationship: Smoking >20 cigarettes per day was associated with a significantly increased risk of persistence of HPV infection among women, when compared with women who smoked <10 cigarettes per day (odds ratio (OR)=1.43, 95 % CI 1.02–2.01) [33]. The remaining two studies prospectively included a total of 8,026 patients but could not establish a positive relationship between smoking

Table 1 Summary of all prospective studies evaluating the association between the incidence of HPVand smoking

Author	Publication year	Study design	Population (sex, number of patients included)	Localization	Association with smoking	HPV incidence in nonsmoking subjects	HPV incidence in currently smoking subjects	OR or HR for currently smoking subjects	Specification of the association
Clarke et al. [15]	2013	Prospective	Female, *n*= 3,737	Genital	Yes	1,491/3,225 (46.2 %) [50.7 months]	317/499 (63.5 %) [50.7 months]	HR=1.2; 95 % CI 1.0–1.3	Evaluation of high-risk HPV infection
Schabath et al. [16]	2013	Prospective	Male, *n*= 4,026	Genital	Yes	812/2,326 (34.9 %) [12 months]	399/948 (42.1 %) [12 months]	HR=1.2; 95 % CI 1.0–1.5	Significantly higher association for current smokers compared to former and never smokers
Nyitray et al. [27]	2011	Prospective	Male, *n*= 1,110	Anogenital	(Yes)	–	–	–	Association in bivariate analysis among men who have sex with women
Nielsen et al. [18]	2009	Prospective	Female, *n*= 7,454	Genital	Yes	–	–	OR=1.5; 95 % CI 1.2–1.9	Association for acquiring a single high-risk HPV infection (≥10 cigarettes per day)
Sarian et al. [19]	2009	Prospective	Female, *n*= 12,114	Genital	Yes	–	–	OR=1.6; 95 % CI 1.2–2.1	Evaluation of high-risk HPV infection
Goodman et al. [28]	2008	Prospective	Female, *n*= 431	Anal	No	137/276 (49.6 %) [16 months]	29/61 (47.5 %) [16 months]	OR=0.9; 95 % CI 0.5–1.6	
Oh et al. [20]	2008	Prospective	Female, *n*= 197	Genital	(Yes)	16/131 (12.2 %) [18 months]	5/12 (41.7 %) [18 months]	OR=3.3; 95 % CI 0.7–14.6	Statistically nonsignificant trend
Partridge et al. [21]	2007	Prospective	Male, *n*= 240	Genital	Yes	–	–	HR=1.0; 95 % CI 0.4–2.2	Association with past smoking but not with current smoking
Syrjänen et al. [22]	2007	Prospective	Female, *n*= 3187	Genital	Yes	–	–	OR=1.5; 95 % CI 1.1–2.1	Evaluation of high-risk HPV infection
Kjaer et al. [23]	2005	Prospective	Male, *n*= 374	Genital	No	17/102 (16.7 %) [7 months]	6/42 (14.3 %) [7 months]	OR=0.6; 95 % CI 0.2–2.0	
Minkoff et al. [24]	2004	Prospective	Female, *n*= 2293	Genital	(Yes)	–	–	–	Association in HIV-infected but not in HIV-uninfected women
Sellors et al. [25]	2003	Prospective	Female, *n*= 307	Genital	No	19/178 (10.7 %) [14 months]	9/72 (12.5 %) [14 months]	OR=0.6; 95 % CI 0.2–1.9	Evaluation of high-risk HPV infection
Winer et al. [26]	2003	Prospective	Female, *n*= 603	Genital	Yes	–	–	HR=1.5; 95 % CI 1.0–2.3	Association with current smoking
Moscicki et al. [29]	2001	Prospective	Female, *n*= 601	Genital	No	–	–	–	

OR odds ratio, *HR* hazard ratio

and the persistence rate of HPV [37, 35]. Interestingly, Ho et al. even found that smoking >5 cigarettes per day was protective against persistent HPV infection (OR=0.3, 95 % CI 0.2–0.7) [35].

Prevalence of HPV

With respect to the prevalence of anogenital HPV, most studies showed an association with smoking for both men and women. Forty-seven studies (four prospective [40, 19, 41, 24], one retrospective [42], and 42 cross-sectional [43-68, 14, 69-83] studies)—including a total of 83,480 patients—found a significant association (35 studies [19, 40-44, 46, 48-53, 55-59, 61-64, 66-68, 14, 69-71, 75-77, 79, 80, 83]) with smoking or a trend (12 studies [24, 45, 47, 54, 65, 72-74, 60, 78, 81, 82]) toward an association, whereas 16 studies (one prospective [84] and 15 cross-sectional [17, 85-98] studies)—with a total of 12,188 patients—did not. Four out of five prospective studies—including 19,581 patients—found a significant association (three studies) between HPV prevalence and smoking or a trend (one study) toward higher rates among smokers compared to nonsmokers (Table 2) [40, 19, 41, 24]. The remaining prospective study with 576 patients did not reveal an association [84].

A statistical analysis of all prospective studies evaluating association between the prevalence of HPV and smoking showed that the overall HPV prevalence for smoking patients was 48.2 versus 37.5 % for nonsmoking patients ($p<0.001$) (OR=1.5, 95 % CI 1.4–1.7). For female patients only, the prevalence was 40.8 % for smokers versus 25.2 % for nonsmokers ($p<0.001$) (OR=2.0, 95 % CI 1.8–2.3) and for male patients 68.2 versus 63.2 % ($p=0.006$) (OR=1.2, 95 % CI 1.1–1.5).

There are ten studies comparing current with past smoking with regard to the prevalence of HPV [49, 51, 40, 61, 19, 14, 69, 72, 77, 90]. Except for one study, no impact of past smoking on the prevalence of HPV was shown [49, 51, 40, 61, 19, 14, 69, 72, 77].

Four cross-sectional studies among current smokers showed increasing prevalence of HPV with the number of cigarettes smoked [49, 50, 14, 69]. Nielson et al. found a stronger association between HPV detection and smoking ≥10 cigarettes than for smoking <10 cigarettes per day (OR=2.3, 95 % CI 1.0–5.3) [69]. In contrast, Roura et al. and Schabath et al. did not find any impact of the intensity of smoking on the prevalence of HPV [51, 40].

High-risk HPV

Four prospective studies focused on the incidence of high-risk HPV infection [15, 19, 22, 18]. These investigators

Table 2 Summary of all prospective studies evaluating the association between the prevalence of HPV and smoking

Author	Publication year	Study design	Population (sex, number of patients included)	Localization	Association with smoking	HPV prevalence in nonsmoking subjects	HPV prevalence in currently smoking subjects	OR or HR for currently smoking subjects	Specification of the association
Schabath et al. [40]	2012	Prospective	Male, *n*= 4,054	Genital	Yes	1,485/2,348 (63.2 %)	655/960 (68.2 %)	OR=1.2, 95 % CI 1.0–1.4	Association with current but not past smokers. No association between intensity of smoking and HPV prevalence
Sarian et al. [19]	2009	Prospective	Female, *n*= 12,114	Genital	Yes	444/2,699 (16.5 %)	215/990 (21.7 %)	OR=1.6, 95 % CI 1.2–2.1	Evaluation of high-risk HPV infection. Association with current but not past smokers
Kliucinskas et al. [41]	2006	Prospective	Female, *n*= 1,120	Genital	Yes	109/892 (12.2 %)	25/128 (19.5 %)	OR=1.8, 95 % CI 1.2–2.8	Evaluation of high-risk HPV infection
Minkoff et al. [24]	2004	Prospective	Female, *n*= 2,293	Genital	(Yes)	516/966 (53.4 %)	735/1,294 (56.8 %)	OR=1.1, 95 % CI 1.0–1.4	Association in HIV-infected but not in HIV-uninfected women
Feldman et al. [84]	1997	Prospective	Female, *n*= 576	Genital	No	174/370 (47.0 %)	93/206 (45.1 %)	OR=0.9, 95 % CI 0.7–1.3	
Total						**2,728/7,275 (37.5%)**	**1,723/ 3,578 (48.2%)**	**OR=1.5, 95% CI 1.4–1.7**	

OR odds ratio, *HR* hazard ratio

consistently found a significant association between smoking and high-risk HPV incidence.

Regarding persistence of high-risk HPV infection, three out of four prospective studies with a total of 827 patients revealed a significant association with smoking [32, 39, 38], whereas one study with 7,418 patients did not [37].

An analysis of HPV types in female patients revealed no significant difference in prevalence of high- or low-risk HPV genotypes when comparing female smokers to nonsmokers [99]. In two prospective studies including a total of 13,234 patients, a significant impact of smoking on the prevalence of high-risk HPV was found [19, 41].

Development of anogenital warts

Smoking has been shown to significantly increase the incidence of anogenital warts (Table 3). The risk of developing anogenital warts increases with the number

Table 3 Summary of all published studies evaluating the association between the incidence of anogenital warts and smoking

Author	Publication year	Study design	Population (sex, number of patients included)	Localization	Association with smoking	Incidence of anogenital warts in nonsmoking subjects	Incidence of anogenital warts in currently smoking subjects	OR or HR for currently smoking subjects	Specification of the association
Massad et al. [103]	2011	Prospective	Female, *n*=3766	Genital	Yes	–	–	HR=1.8, 95 % CI 1.4–2.4	Association with current but not with former smoking
Wiley et al. [113]	2009	Prospective	Male, *n*=2835	Genital	Yes	–	–	–	
Massad et al. [104]	2004	Prospective	Female, *n*=2031	Genital	Yes	–	–	–	Association with current but not with former smoking
Feldman et al. [84]	1997	Prospective	Female, *n*=576	Genital	Yes	–	–	–	
Hansen et al. [102]	2010	Retrospective	Female, *n*=58094	Genital	Yes	–	–	HR=1.3, 95 % CI 1.2–1.4.	Increasing risk with increasing number of cigarettes smoked per day
Kjaer et al. [114]	2007	Retrospective	Female, *n*=69147	Genital	Yes	2,936/35,799 (8.2 %)	2,251/15,965 (14.1 %)	OR=1.1, 95 % CI 1.0–1.2	Association with smoking for >59 pack-years
Wen et al. [13]	1999	Retrospective	Male and female, *n*=1954	Genital	Yes	89/631 (14.1 %)	154/644 (23.9 %)	OR=1.9, 95 % CI 1.0–2.3	Smokers of more than 10 cigarettes per day were twice as likely to have genital warts as were nonsmokers
Habel et al. [106]	1998	Retrospective	Female, *n*=282	Anogenital	(Yes)	45/118 (38.1 %)	35/67 (52.2 %)	OR=1.8, 95 % CI 1.0–3.3	Statistically nonsignificant association
Munk et al. [100]	1997	Retrospective	Female, *n*=10838	Genital	Yes	–	–	OR=1.5, 95 % CI 1.2–1.8	Increasing risk with increasing pack-years of cigarette smoking
Brisson et al. [101]	1988	Retrospective	Female, *n*=520	Genital	Yes	–	–	–	Increasing risk with increasing number of cigarettes smoked per day
Daling et al. [115]	1986	Retrospective	Female, *n*=245	Anogenital	Yes	–	–	–	

OR odds ratio, *HR* hazard ratio

of cigarettes smoked per day and the number of pack-years [13, 100-102]. However, this association only applies to current but not to past smoking [103, 104]. Luu et al. investigated the impact of current smoking on the size of anal warts but failed to find a relationship [105]. Smoking for 10 or more years has been found to increase the risk of recurrent genital warts (relative risk (RR)=4.5, 95 % CI 1.4–13.8) [106].

Discussion

In both genders, smoking is associated with higher incidence and prevalence rates for HPV infection, whereas the latter responds to a dose-effect relationship. Smoking does also increase persistence rates for high-risk HPV infection, while this correlation is debatable for low-risk HPV. The incidence and recurrence rates of anogenital warts are significantly increased in current smokers.

Smoking has deleterious effects on systemic and local immunity, as it suppresses both cell-mediated and humoral immune responses, which might lead to the present finding of increased susceptibility to HPV infection and development of anogenital warts [107]. Nicotine, the addictive substance in cigarette smoke, has been shown to be the main immunosuppressive constituent of cigarette smoke [107]. Moreover, smoking has been found to increase metaplasia and DNA damage in various tissues [108-111].

Although most studies adjusted for sexual behavior, unmeasured high-risk sexual behavior might be a potentially important confounder in the association between smoking and the incidence of HPV infection [16, 15, 26]. This is supported by Herrero et al., who found that sex with multiple partners is more prevalent among smokers [112].

The prevalence of HPV infection seems to decrease in patients who quit smoking, but the time period after which nonsmoker levels are reached is not yet clear.

The association of smoking with HPV infection and development of anogenital warts, respectively, is well supported by current data. However, the cascade from HPV infection to the development of anogenital warts is still not well understood. Furthermore, prospective data on the impact of smoking on the spontaneous recovery rate of anogenital warts are still lacking.

Studies are more often performed for female than male patients, probably due to regular gynecologic controls. On the other hand, comparison of the studies did not reveal gender differences in the impact of smoking.

Conclusion

Most current data demonstrate an association between smoking, increased anogenital HPV infection, and development of anogenital warts. These data add to the long list of reasons for making smoking cessation a keystone of patient health.

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