HUMAN PAPILLOMA VIRUS (HPV) PREVALENCE AND GENOTYPE DISTRIBUTION

HUMAN PAPİLLOMA VİRUS (HPV) PREVALANSI VE GENOTİP DAĞILIMI

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Öz

Human papillomavirus (HPV) prevelansını ve genotip dağılımının değerlendirilmesi, profilaktik HPV aşısının etkisinin izlenmesinde çok önemlidir. Bu çalışmanın amacı, Başkent Üniversitesi Adana Dr. Turgut Noyan Uygulama ve Araştırma Merkezinde, bayan hastalarda HPV prevalansı ve genotip dağılımını göstermektir. Nisan 2014 ve Kasım 2015 arasında, yaşları dağılımı 22-68 yıl olan 268 bayan hastadan servikal örnekler toplanmıştır. HPV DNA PCR ile çoğaltılmış ve HPV genotiplendirmesi Roche®linear array detection kit ile yapılmıştır. Histopatolojik inceleme 146 bayan hastaya yapılmıştır. Toplamda 268 uygun örneğin 124' ünde (%46.3) HPV için pozitif bulunmuştur ve bunların çoğu [84/124 (%67.7)] yüksek riskli (HR) HPV enfeksiyonlarıdır. HPV-16 pozitifliği %20.9 (n=26) ve HPV-18 pozitifliği %4 (n=5) olarak bulunmuştur. Grade I-III Servikal intraepitelyal neoplasiler (CIN) de HPV tip spesifik prevelansı sırasıyla %63.9, %53.8 ve %80' dir. Yüksek dereceli servikal lezyonlarda HPV-16 dışında diğer HR-HPV tipleri, HPV-31, 45, 51, 53 ve 56' yı içermektedir. Sonuç olarak, hastanemizde HPV-16 servikal lezyonlarla en sık ilişkili HPV genotipi olarak saptanmıştır. Bu çalışma, ayrıca yüksek ve düşük riskli HPV genotiplerini aynı zamanda birden fazla HPV enfeksiyonların prevalansı hakkında da bilgi vermektedir.

Anahtar Kelimeler

HPV; Genotiplendirme; İntra Epitelya Servikal Neoplaziler

Abstract

Assessment of Human papillomavirus (HPV) prevalence and genotype distribution is important for monitoring the impact of prophylactic HPV vaccination. This study aimed to demonstrate the HPV prevalence and type distribution in women from the Baskent University Adana Dr. Turgut Noyan Practice and Research Center. Cervical specimens from 268 women aged 22-68 years were collected between April 2014 and November 2015.Histopathological examinations were performedfor 146 women. HPV DNA was amplified by PCR and HPV and genotyping was undertaken using the Roche® linear array detection kit. In total, 124 out of 268eligible samples (46.3%) tested positive for HPV, with the majority of these [84/124 (67.7%)] having high-risk (HR) HPV infection; 20.9% were positive for HPV16 (n=26), and 4% for HPV18 (n=5). HPV type-specific prevalence was 63.9%, 53.8%,and 80% among cervical intraepithelial neoplasias (CIN) Grades I-III, respectively. The coverage of other HR-HPV genotypes apart from 16, included HPV31, 45, 51, 53, and 56in high-grade cervical lesions. In conclusion, HPV-16 was identified as the main HPV genotype associated with cervical disease in our hospital. The study reports the identification of high-and low-risk HPV genotypes as well as the prevalence of multiple HPV infections.

Keywords

HPV; Genotyping; Cervical Intraepithelial Neoplasms.

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Introduction

Human papilloma virus (HPV) is one of the most common causes of sexually transmitted disease in both men and women worldwide. Genital HPV infection is not a reportable disease, so actual incidence and prevalence figures are not known.Papillomaviruses are ubiquitous and have been detected in a wide variety of animals as well as in humans and are specific for their respective hosts. More than 200 types of HPV have been recognized on the basis of DNA sequence data showing genomic differences [1].

Cervical cancer (CC) represents the second-most common malignancy in women around the world and contributes to 9.8% of all female cancers. Increasing evidence suggests that multiple factors contribute to the development of cervical cancer, including genetic susceptibility or host genome, co-infection of HPV and other agents, and life-style factors. Based on their association with cervical cancer and precursor lesions, HPVs can also be grouped into high-risk and low-risk HPV types. Low-risk HPV types include types 6, 11, 40, 42, 43, 44, 54, 61, 70, 72, and 81. High-riskHPV types include types 16, 18, 31, 33, 34, 35, 39, 45, 51, 52, 56, 58, 59, 66, 68, and 70. Although all genotypes of HPV correlate with cervical cancers, HPV-16 and HPV-18 infection accounts for about 70% of all cases [1-5].

Although HPV-16, 18, 31, 52, and 58 are cited among the most common HPV genotypes in women with high-grade cervical lesions worldwide, there is significant variation in the reported prevalence of HR-HPV genotypes amongcountries.Since the establishment of HPV as the central cause of CC, data on HPV type distribution in CC have proven useful to predict the potential impact of HPV16 and 18 vaccines, as well as to determine priorities for inclusion of carcinogenic HPV types in future HPV vaccines and HPV-based screening tests [2,3,5,6].

The aim of the present study was therefore to provide an estimate of the background prevalence of HPV infection and age-specific HPV type distrubition, and to highlight the HPV genotypes most frequently present in cervical cancer tissue and precursor lesionsin Baskent University Adana Dr Turgut Noyan Practice and ResearchCenter, Adana, Turkey.

Materials and Method

Study Population

Between April 2014 and November 2015, an HPV prevalence study was conducted among the female patients who attended the Baskent University Adana Dr. Turgut Noyan Practice and Research Center. Samples were collected from women with routine cervical screening.

Cervical samples were collected with a cytobrush during gynecological exaninations. This was inserted into the endocervical canal and then placed into the transport medium (PreservCyt solution; Hologic, Bedford, MA, USA) and stored at 4°C until DNA extraction for HPV genotyping.

Clinical specimens

The 268 cervical cytology specimens collected were analyzed for the presence of DNA of HPV. Histopathological examinations were performed in 146 women by the pathologist according to the Bethesda Diagnostic Criteria. Lesions were classified as normal cytology, CIN I-III, or adenocarcinoma.

Linear Array HPV genotyping test

The Linear Array® (LA) HPV Genotyping test is registered for use in Europefor detecting 37 high- and low-risk HPV genotypes. The test is based on four major processes: 1) DNA extraction by the AmpliLute Liquid Media Extraction Kit; 2) PCR amplification of target DNA using HPV primers; 3) hybridization of the amplified products into oligonucleotide probes (Linear Array HPV genotyping test); and 4) detection of probe-bound amplified products by colorimetric determination (Linear Array Detection Kit). Briefly, using PGMY09/11 primers, a region approximately 450 base pairs in length within the L1 gene of the HPV genome was amplified by PCR. This assay simultaneously amplified a region within the human β globin gene as a control for cell adequacy, nucleic acid extraction, and PCR efficiency. PCR assays were performed in a reaction volume of 100 µl, using 50 µl of LA HPV master mix (Roche Molecular Systems) and 50 µ of DNA. The amplification parameters were: 2 min at 50°C and 9 min at 95°C; 40 cycles of 95°C for 30s, 55°C for 1 min, and 72°C for 5 min or until samples were collected. Nucleic acid hybridization using a reverse line blot system was then performed. Briefly, the PCR amplicons were denatured with the addition of 100 µl of LA denaturarion reagent (Roche Molecular Systems). After 10 min at room temperature, the denatured amplicons (100 µl) were hybridized and detected using the recommended LA protocol. Thirty-seven anogenital HPV genotypes were detected simultaneously, including 6, 11, 16, 18, 26, 31, 33, 35, 39, 40, 42, 45, 51-56, 58, 59, 61, 62, 64, 66-73, 81-84, IS39, and CP6108. The LA HPV genotyping strips were visually read using the HPV reference guide provided.

Results

In total, 268samples were received between April 2014 and November 2015. The 146samples were acquired through punch biopsies (54.5%).

Prevalence of HPV Infection

In total 124/268 samples tested positive for HPV (46.3%), with the majority of those (84/124,67.7%) having HR-HPV infection, 20.9% beingpositive for HPV-16 (n=26), and 4% being positive forHPV-18 (n=5). A total of 144/268 (53.7%) of samples were HPV negative.Figure outlines the overall HPV genotype profile among the cohort and HPV multiplicity. The six most common HPV genotypes detected across all samples examined were HPV-16, HPV-CP6108 (n=17), HPV-53 (n=17), HPV 56 (n=13), HPV 51 (n=10), and HPV-84 (n=10) (Fig.)

HPV Type-Specific Prevalence

The majority of cervical pathologies were from CIN I cases (36/146, 24.7%). HPV DNA was detected in 23/36 (63.9%) of CINI samples, 7/13 (53.8%) of CIN II lesions, and 12/15 (80%) of CIN III specimens. HPV DNA was not detected inAC samples (2/146, 1.4%). Table I details the number of HPV genotypes detected by pathological subtype. One-third of all samples (28.8%) had only one HPV genotype detected (n=19). HPV-16 DNA was present in 33.3% (5/15) of CIN III samples (Table II). Other HR-HPV genotypes were more prevalent in CIN I-II pathologies. LR-HPV genotypes were most common in CIN I lesions (Table II). Almost one-half (54.8%) of all pathologies were HPV negative.

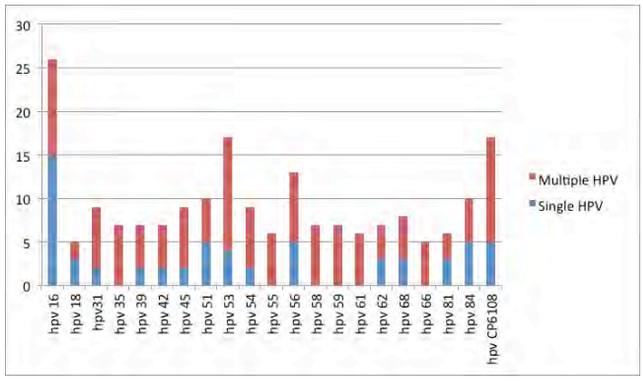


Fig. Overall HPV genotype profile denoting the prevalence of single and multiple HPV infections. HPV genotypes omitted above (no. of single/multiple infections): HPV 33 (1/1), HPV 40 (2/0), HPV 64 (0/1), HPV 67 (0/1), HPV 70 (0/4), HPV 72 (0/2), HPV 82 (1/2), and HPV 83 (1/3).

Table I. Percentage Distribution of the Number of HPV Genotypes Detected by Pathological Subtype and 5-Year Age Group

Number of HPV genotypes detected (% of each pathology)						
	HPV negative	1	2	3	4 or more	Total n (%)
Pathology						
CIN I	13 (36.1)	8 (22.2)	4 (11.1)	10 (27.8)	1 (2.8)	36 (24.7)
CIN II	6 (46.1)	3 (23.1)	2 (15.4)	1 (7.7)	1 (7.7)	13 (8.9)
CIN III	3 (20)	8 (53.4)	2 (13.3)	2 (13.3)	0	15 (10.3)
AC	2 (100)	0	0	0	0	2 (1.4)
Age group (yrs)						
Under 25	3 (60)	0	2 (40)	0	0	5 (1.9)
25-29	14 (45.2)	8 (25.8)	2 (6.4)	5 (16.2)	2 (6.4)	31 (11.6)
30-34	25 (58.2)	8 (18.6)	5 (11.6)	5 (11.6)	0	43 (16)
35-39	35 (60.3)	14 (24.1)	3 (5.2)	3 (5.2)	3 (5.2)	58 (21.6)
40-44	21 (42.8)	14 (28.6)	7 (14.3)	7 (14.3)	0	49 (18.3)
45-49	17 (56.7)	9 (30)	1 (3.3)	3 (10)	0	30 (11.2)
50-54	18 (62.1)	6 (20.7)	1 (3.4)	4 (13.8)	0	29 (10.8)
55-59	4 (30.8)	7 (53.8)	0	2 (15.4)	0	13 (4.8)
60-64	6 (75)	1 (12.5)	1 (12.5)	0	0	8 (3)
65+	1 (50)	0	0	0	1 (50)	2 (0.8)
Total	144	67	13	29	6	268

CIN, cervical intraepithelial neoplasia (Grades I-III); AC, adenocarcinoma.

The distribution of HPV genotypes detected within each cervical pathology is detailed in Table III. HPV-16 was the most common HPV detected across all cervical pathologies (Table III). We found that the prevalence of HPV infection without cervical abnormalities was 39.7% (58/146).

Age-Specific Prevalence for HPV Infection

The pathological distribution of samples by 5-year age groups is shown in Table IV. The mean age of women included in the study was 41 years (range 22-68 years, standard deviation (SD) 9.8 years). In total, 5 samples were from women aged 24 years and under,74 from thoseaged 25-34 years, and 189from those aged 35 years and over. CIN I was more common in women aged 50-54 years and CIN III most common in women aged 30-34 years (Table IV).

The number of HPV genotypes decreased with increasing age, with just over half (57.2%) of all HPV infections found in tissue from women aged between 40 and 44 years (Table I). Most tissue samples had a single HPV genotype and this was more

Table II. Type of HPV Genotypes Detected by Cervical Pathology and 5-Year Age Group

	HPV-16 and or HPV-18 n (%)	Other high-risk HPV genotypes n (%)	Low-risk HPV genotypes only n (%)	HPV negative n (%)	Total
Pathology					
CIN I	4 (11.1)	10 (27.7)	31 (86.1)	13 (36.1)	36
CIN II	2 (15.4)	7 (53.8)	4 (30.8)	6 (46.2)	13
CIN III	5 (33.3)	3 (20)	10 (66.7)	3 (20)	15
AC	0	0	0	2 (100)	2
Age group (yrs)					
Under 25	2 (6.4)	0	2 (1.9)	3 (60)	5
25-29	3 (9.7)	17 (23.6)	11 (10.5)	14 (48.4)	31
30-34	5 (16.1)	8 (11.1)	19 (18.1)	25 (60.5)	43
35-39	5 (16.1)	16 (22.2)	19 (18.1)	35 (60.3)	58
40-44	7 (22.6)	13 (18)	26 (24.8)	21 (44.9)	49
45-49	2 (6.5)	9 (12.5)	7 (6.6)	17 (56.7)	30
50-54	2 (6.5)	4 (5.6)	14 (13.3)	18 (62.1)	29
55-59	4 (12.9)	4 (5.6)	5 (4.8)	4 (12.5)	13
60-64	0	1 (1.4)	2 (1.9)	6 (75)	8
65+	1 (3.2)	0	0	1 (50)	2
Total n	31	72	105	144 (53.7)	268

CIN, cervical intraepithelial neoplasia (Grades I-III); SCC, squamous cell carcinoma; AC, adenocarcinoma.
[a]Including high-risk HPV genotypes other than HPV 16/18, that is: HPV-31, 33,35,39,45,51,52,56,58,59, and 68.

Table III. HPV Genotype Distribution by Cervical Pathology

Cervical histologya n (%)					
HPV genotype	AC	CIN I	CIN II	CIN III	Total
HPV 16	0	2 (5.6)	1 (7.7)	5 (33.3)	8 (12.1)
HPV 31	0	1 (2.8)	2 (15.4)	1 (6.7)	4 (2.7)
HPV 18	0	2 (5.6)	1 (7.7)	0	3 (2.1)
HPV 51	0	2 (5.6)	2 (15.4)	0	4 (2.7)
HPV 45	0	2 (5.6)	1 (7.7)	1 (6.7)	4 (2.7)
HPV 39	0	1 (2.8)	0	0	1 (0.7)
HPV 58	0	1 (2.8)	0	0	1 (0.7)
HPV 66	0	1 (2.8)	0	1 (6.7)	2 (1.4)
HPV 35	0	2 (5.6)	1 (7.7)	0	3 (2.1)
HPV 6	0	2 (5.6)	0	0	2 (1.4)
HPV 73	0	1 (2.8)	0	0	1 (0.7)
HPV 53	0	3 (2.8)	0	2 (13.3)	5 (3.4)
HPV 70	0	2 (5.6)	0	1 (6.7)	3 (2.1)
HPV 42	0	2 (5.6)	0	1 (6.7)	3 (2.1)
HPV 61	0	2 (5.6)	0	2 (13.3)	4 (2.7)
HPV 11	0	2 (5.6)	0	0	2 (1.4)
HPV CP6108	0	1 (2.8)	1 (7.7)	1 (6.7)	3 (2.1)
HPV 82	0	0	2 (15.4)	0	2 (1.4)
HPV 62	0	3 (2.8)	0	0	3 (2.1)
HPV 84	0	4 (11.1)	0	1 (6.7)	5 (3.4)
HPV 81	0	2 (5.6)	0	0	2 (1.4)
HPV 55	0	3 (2.8)	0	1 (6.7)	4 (2.7)
HPV 83	0	0	1 (7.7)	0	1 (0.7)
HPV 67	0	1 (2.8)	0	0	1 (0.7)
HPV 40	0	1 (2.8)	0	0	1 (0.7)
HPV 56	0	2 (5.6)	1 (7.7)	1 (6.7)	4 (2.7)
HPV 72	0	1 (2.8)	0	0	1 (0.7)

[a]Several patients may have had multiple HPV genotypes on testing.

Table IV. Age-Specific Prevalence for Cervical Pathology

Age group (yrs)	CIN I n, (%)	CIN II n, (%)	CIN III n, (%)	AC n, (%)	Total, n (%)
Under 25	2 (1.4)	0	0	0	2 (1.4)
25-29	2 (1.4)	4 (2.7)	0	0	6 (4.1)
30-34	3 (2.1)	2 (1.4)	5 (3.4)	0	10 (6.8)
35-39	3 (2.1)	4 (2.7)	3 (2.1)	1 (0.7)	11 (7.5)
40-44	7 (4.8)	2 (1.4)	3 (2.1)	0	12 (8.2)
45-49	7 (4.8)	0	0	0	7 (4.8)
50-54	10 (6.8)	1 (0.7)	0	0	11 (7.5)
55-59	1 (0.7)	2 (1.4)	0	0	3 (2.1)
60-64	1 (0.7)	0	1 (0.7)		2 (1.4)
65+	0	0	1 (0.7)	1 (0.7)	2 (1.4)
Total n	36 (24.7)	13 (8.9)	15 (10.3)	2 (1.4)	66 (45.2)

common in those aged over 55 years. Four or more HPV genotypes were detected in some women and this was more common under the age of 40 years (Table I). 17% of women with cervical pathology had HPV-16 or HPV-18 detected, and the highest proportion of HPV positive (16/18)were women aged 40-44 years (Table II). Other HR-HPV genotypes were more common in younger women, particularly those aged 25-29 years. LR-HPV genotypes were most common in those aged 40-44 years (Table II).

Discussion

This was a retrospective study investigating the HPV genotype distribution in women in Baskent University Adana Dr Turgut Noyan Practice and Research Center. HR-HPV was detected in 67.7% of all samples, with HPV-16 being the most common (20.9%) HPV genotype identified. This is consistent withprevalencesdescribed elsewhere across Europe in countries such as Spain, Germany, Finland, North Ireland, and internationally [3, 5, 7-9].The prevalence of HPV in CIN lesions in our study was 63.9% in CIN I, 53.9% in CIN II, and 80% in CIN III cases. We didn't detect any HPV DNA in AC samples across all cervical samples investigated. We found that HPV-16, CP6108, 53, 56, 51, and 84 were the most common genotypes in all cases and HPV-16, 31, 45, 51, 53, 56, and 61 were the most common genotypes in high-grade cervical lesions. The number of HPV genotypes detected in the current study varied across pathological grade, with the lowest percentage of single genotypes (22.2%) in CIN I lesions and the highest proportion (53.4%) in CIN III lesions.

In our study, the proportion of HPV positivity (67.7%) was similar to Europe (73.8%), Central/South America (64.2% vs. 67.3%), North America (76.4%), Asia (66.9%), Africa (70%), and Turkey (66%) [7, 11]. However, our prevalence rate of HPV 16/18 (25%) is lower than in Europe (57.6%) and Africa (67.7%) [6].

In general, precancerous cervical lesions, i.e. CIN II-III, have been accepted as a threshold of initiating definite treatment of precancerous lesion of squamous cell carcinoma (SCC). Identifying HPV genotype distribution in CIN II-IIIlesions that potentially progress to SCC is of utmost important in gaining insight into oncogenic potential of the different HPV genotypes, designing protocol for screening, and estimating the efficacy of type-specific HPV vaccines. As a substantial geographical variation in the HPV genotype distribution has been observed, data regarding HPV type-specific prevalence in each country are therefore required[11,12].

In an examination of FFPE tissue from more than 6,000 women from 17 European countries using the SFF10-LiPA25 assay, Tjalma et al. [13]found HPV-16 was the most frequent HPV type detected in both CIN and invasive cervical cancer. HPV-16 and/or HPV-18 prevalence (among HPV positive cases) was reported as 45.8% in CIN II and 67.3% in CIN III cases, higher than in our study. The prevalence of HPV-16 and/or-18 in our study was 15.4% in CIN II cases and 33.3% in CIN III cases. The authors reported HPV-31, 33, 35, 51, 52, 58, and 68 as the most frequently detected genotypes in women with high-grade CIN lesions. It has been reported that, worldwide, HPV-16 is the genotype with highest prevalence, followed by HPV-18 and HPV 31. Apart from HPV-16, we found that HPV- 31, 45, 51, 53, 56, and 61 were the most common genotypes identified in high-grade lesions. A study from Turkey by Ateşer et al. found that HPV- 16, 6, 11, 58, and 18 were the most common genotypes identified in high-grade lesions [3,10, 14, 15].

Clifford et al.[12]have suggested that worldwide, CIN II-III infected with HPV16, 18, or 45 are more likely to progress to SCC than CIN II-III infected with other HR types. They performed a meta-analysis of published data to compare HPV type distribution in CIN II-III and SCC. These data suggest that CIN II-III infected with HPV16, 18, and 45 more oftenprogress to SCC.Overall, HPV prevalence was slightly higher in SCC cases (87.6%) than in CIN II-III (84.2%). HPV 16 was the most common type in both SCC (54.3%) and CIN II-III (45%). HPV18 was also more prevalent in SCC (12.6%) than in CIN II-III (7%). When estimated from studies in Asia, Europe, and South/Central America, respectively, there was no material difference in SCC:CIN II-III ratios for HPV16, HPV18, HPV45, HPV33, HPV52, or HPV58. However, notably high ratios were observed for HPV31 in South/Central America in comparison toEurope and Asia, and for HPV58 in China (including Taiwan and Hong Kong) in comparison tonon-Chinese Asian countries, raising the possibility of localised variation in the malignant potential of particular HPV types [2,11].

There were only two cases of AC included in the current investigation. HPV DNA was not detected in AC samples. Therefore our study is probably not powerful enough to investigate HPV prevalence in this subgroup. A recent study in FFPE tissue detected using the SPF10-DEIA/LiPA25-PCR assay reported thatthe prevalence of HPV-16 and/or HPV-18 was 64.3%, lower than reported in an English multi-site investigation of HPV DNA in cervical cytology and cervical cancer biopsies using the Roche Linear array typing system (81.9%, among HPV positive cases) and among AC cases from other European studies (94.6%)[3].

We found that the prevalence of HPV infection without cervical abnormalities was 39.7%. Forman et al. reported that, worldwide, the prevalence of HPV infection without cervical abnormalities is 11% to 12%. De Sanjosé et al. found that, overall, in Asia and China the prevalence of HPV in females without cytological abnormalities was 8.0% and 11.4-20.3%, respectively (Zeng2016).Ogembo et al. found that HPV infection among women in Africa with normal cervical cytology was 57.3% in Southern Africa, followed by Eastern Africa (42.2%), Western Africa (7.8%), and Northern Africa (12.8%) [6].

A previous meta-analysis showed that, worldwide, HPV prevalence was highest in the younger age categories (<34 years), with a second peak in the older age categories (≥45 years). We observed a similar pattern, with peaks of prevalence in subjects aged <34 years and these older than 40 years. The mechanism of the association between HPV infection and age is not clear. Lee et al. showed that changes in vaginal microbiota in postmenopausal women made them more susceptible to HPV infection. Other research found that bacterial vaginosis was associated with susceptibility to HPV infection. Moreover, the social activities of the younger age groups can lower immunity to HPV and increase chance exposure [15, 16].

In this study, multiple HPV infections were identified in 38.7% of the positive specimens, and the age-specific prevalence of multiple HPV infections also showed peaks at ages 25-29 and 55-59 years. Zeng et al.[15] speculated that the decline of immunosurveillance in the younger and older age groups may increase the risk of multiple HPV infections. Also it should be noted that HPV has the ability to evadehost defenses. According to previous studies, people infected with >2 HPV genotypes might have an increased risk of developing cervical cancer. However, the interaction af various genotypes in co-infections remains unclear, and future studies are needed to verify whether coordinated mechanisms in co-infections exist.

Although we achieved a few novel findings in the present study, there remain other relevant factors that should be considered. Firstly, we collected the samples from the general female population, and not specifically cervical cancer patients. Therefore the findings and conclusions drawn from this study may not be applicable for estimating HPV genotype-specific prevalence in woman affected by cervical cancer. Secondly, HPV also causes diseases in men, including cancer of the penis; Smith et al. [17] reported that the prevalence of overall HPV was 16% in men. However, the objectives of our study did not include an evaluation of gender-specific prevalence of HPV infection. It would be interesting in future studies to investigate whether the prevalence of HPV infection in men matches that of women, since vaccinating males is also considered important[15].

In conclusion, HPV-16 was identified as the main HPV genotype associated with cervical disease in our hospital. The study reports the identification of high-and low-risk HPV genotypes as well as the prevalence of multiple HPV infections. When comparing the HPV prevalence between countries it is important to consider that variations in HPV positivity may be explained by differences in the quality and type of samples analyzed (biopsies, surgical specimens, or fresh tissue), as well as the methods of HPV detection and assessment.

Competing interests

The authors declare that they have no competing interests.

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