International Journal of Infectious Diseases 93 (2020) 22–27

Contents lists available at ScienceDirect

International Journal of Infectious Diseases

journal homepage: www.elsevier.com/locate/ijid

Prevalence of human papilloma virus in Oman: Genotypes 82 and 68 are dominating

Zainab Al-Lawati[a], Faryal A. Khamis[b], Aisha Al-Hamdani[d], Moza Al-Kalbani[f], Fatma Ali Ramadhan[c], Thuria Rashid Al-Rawahi[e], Muhannad F. Al-Kobaisi[a,*]

[a] Department of Microbiology & Immunology, College of Medicine and Health Sciences, Sultan Qaboos University, Oman
[b] Department of Medicine, Royal Hospital, Oman
[c] Department of Pathology, Royal Hospital, Oman
[d] Department of Pathology, Sultan Qaboos University Hospital, Oman
[e] Department of Obstructive and Gynecology, Royal Hospital, Oman
[f] Department of Obstructive and Gynecology, Sultan Qaboos University Hospital, Oman

ARTICLE INFO

Article history:
Received 12 September 2019
Received in revised form 22 December 2019
Accepted 31 December 2019

Keywords:
High risk HPV
Low risk HPV
Genotypes
Normal cervical cytology
Abnormal cervical cytology
HPV prevalence

ABSTRACT

Background: Persistent infection with high-risk (HR) HPV genotypes has been associated with cervical cancer, the third cancer affecting women in Oman with a crude incidence rate of 4.7 and mortality rate of 2.5 respectively. Other types of lower-risk (LR) HPV are associated with warts in both genders worldwide.
Objectives: To assess the prevalence and genotype distribution of HPV and the risk factors among women with normal and abnormal cytology.
Methods: A cross sectional study conducted between September 2014 and April 2015. 258 cervical samples were obtained from women aged 18–68 years attending the Gynaecology Out-patient Clinic. HPV genotyping was performed using a multiplex real time-polymerase chain reaction (RT-PCR) assay.
Results: 22 different HPV genotypes were detected in 46 women (17.8%) and included 15 HR and 7 LR genotypes. Human immunodeficiency virus (HIV) patients (P = 0.052) and oral contraceptives users (P = 0.016) showed significant association with HPV infection.
Conclusion: The most frequently observed HPV types were HR HPV 82 and LR HPV 54. These findings show that the predominant HPV genotypes in Oman are different from those seen in worldwide studies. This finding is important to determine the potential impact of preventive measures especially new vaccines to reduce the burden of cervical cancer.

Introduction

Human papillomavirus (HPV) is a sexually transmitted infection and most sexually active individuals acquire it at some point in their lifetime (Crosbie et al., 2013). Evidence suggests that cervical cancer is restricted to high risk HPV, such as (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58 and 59) or possibly carcinogenic types: (68, 26, 30, 34, 53, 66, 67, 69, 70, 73, 82, 85 and 97) (Doorbar et al., 2012). However, de Sanjosé and colleagues concluded that the evidence is sufficient to consider HPV genotypes 26, 30, 67, 69, and 82 as carcinogenic (de Sanjosé et al., 2010). The most common HR-HPV 16, 18, 45, 31 and 33 account for 80% of cervical cancer occurrence, while in normal cytology the most common HPVs are 16, 18, 31, 58 and 52 (de Sanjosé et al., 2007). Vaccines have been targeting most of the above-mentioned HR-HPV, including the approved 9 valent vaccine GARDASIL® 9 () (Petrosky et al., 2015).

Several studies on HPV infection in the Middle East and North Africa (MENA) region showed that its prevalence is low compared to western countries (Vaccarella et al., 2013).

No studies on the prevalence and genotypes of HPV in Oman have yet been performed (Bruni et al., 2014), hence the objective of this study was to determine the prevalence of HPV infection, and HPV genotypes in women with normal and abnormal cytology.

Methods

Study group

A cross-sectional study based on convenience sampling was conducted at two tertiary care hospitals in Oman: Sultan Qaboos

* Corresponding author at: Department of Microbiology & Immunology, College of Medicine and Health Sciences, P.O. Box.: 35, Sultan Qaboos University, Al Khod, P. C.123, Oman.
E-mail address: kobaisi@squ.edu.om (M.F. Al-Kobaisi).

https://doi.org/10.1016/j.ijid.2019.12.038

University Hospital (SQUH) and Royal Hospital (RH). The study group were married Omani women attending the Gynaecology Out-patient Clinic for either follow-up (i.e. previous dysplasia) or for gynaecologic symptoms or routine screening. Non-Omani women and post total hysterectomy women were excluded from the study. Cervical specimens were collected from 1st September 2014–30th April 2015.

A total of 258 Omani women with an age range of (18–68 years) were enrolled in this study. The age cluster was stratified into 4 groups: ($\leq$25, 25–34, 35–44 and $\geq$45) years. An informed consent form was given to those who participated in the study and were interviewed to obtain demographic, reproductive and sexual history along with risk factors. The risk factors included: history of sexually transmitted diseases (HIV and others), age of first sexual intercourse, multiple marriages, contraceptive methods used and multiparity. The study was approved by the Ethical Committee of the College of Medicine and Health Sciences (COMHS) joint with SQUH (MREC #871) and the Research and Ethics Committee (MESR #36) of RH.

Cytology

Samples were taken from the cervical squamous columnar junction area using a cervical brush and the exfoliated cells were placed into Preserve Cyt media (Thin Prep Pap Test; Cytyc Corporation, Boxborough, Mass). Cervical slides were prepared using liquid-based cytology and the cytological classifications were made according to Bethesda 2001 criteria (Cubie, 2013). Residual exfoliated cells from Preserve Cyt media were used for HPV testing.

DNA extraction

Genomic DNA was extracted from Thin Prep samples by QIAamp DNA Mini Kit, following manufacturer instructions (Qiagen, CA, USA, cat No 51304).

Multiplex real time PCR

HPV detection and genotyping were performed with a multiplex real-time PCR kit, following manufacturer instructions (Anyplex™ II HPV28 Seegene, Seoul, Korea) using a CFX96™ real-time thermocycler (Bio-Rad, Hercules, CA, USA). The Anyplex™ II HPV28 detection assay permits simultaneous amplification, detection and differentiation of target nucleic acids of 19 HR-HPV genotypes (16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 69, 73, 82) in addition to 9 LR-HPV genotypes (6, 11, 40, 42, 43, 44, 54, 61, 70).

The real-time PCR efficiency and amplification process were monitored throughout the study by the use of different controls, including an internal control (Human housekeeping gene), incorporated into the samples as an endogenous whole process control (Schrader et al., 2012).

Statistical analysis

Statistical analysis was performed using IBM SPSS version 22.0 (Statistical Package for Social Sciences).

The prevalence of HPV positivity was assessed using the chi-square test. A *p-value* of less than 0.05 was considered significant. Odd ratios (OR) for HPV positivity were calculated with their 95% confidence intervals.

Results

Overall HPV detection by real time PCR

Among the 258 patients, 46 were positive for HPV DNA, demonstrating a prevalence of 17.8%; 95% confidence interval 13.1–22.5. The HPV genotyping revealed 22 genotypes among the samples studied. Those included 15 HR-HPV genotypes (HPV16, 18, 31, 33, 35, 39, 51, 52, 53, 56, 58, 66, 68, 82, 73) and 7 LR-HPV genotypes (HPV6, 11, 54, 44, 43, 42, 70) (Figure 1). The most prevalent HR-HPV genotypes were HPV82 (10.77%), HPV 68 (7.69%), while HPV 56, HPV 53 and HPV 18 (6.15%) each. The most prevalent LR-HPV genotypes were HPV 54 (12.31%), HPV 42 (7.69%), and HPV 44 (6.15%). Single, double and multiple HPV types that were present in a single infection accounted for 35 (76.1%), 7 (15.22%) and 4 (8.7 %) of HPV prevalence respectively. According to HPV risk types, HR types were detected in 27 (58.7%) women, while LR types were detected in 15 (32.6%), and 4 (8.7%) infected women had both HR and LR- HPV.

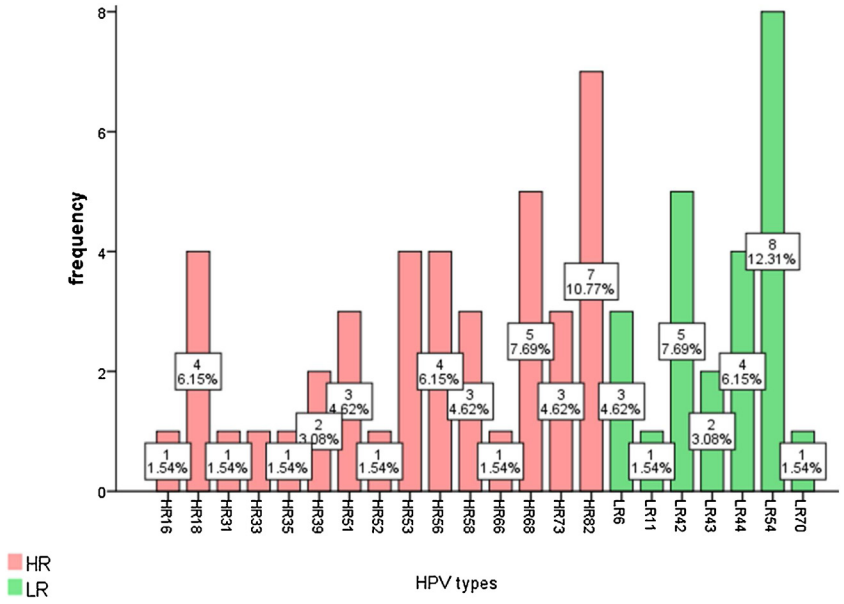

Figure 1. The 22 different HPV genotypes detected in the study population.

Cytology results

Based on cervical cytology test reports, 231 (89.5%) of the females had normal cytological results. In contrast, 16 (6.2%) had abnormal cytology results and 11 (4.3%) had unsatisfactory smears. Among the 16 women with abnormal cytology results, 13 (5%) showed atypical squamous cell of unknown significance (ASCUS), while 3 (1.2%) were of low-grade squamous intraepithelial lesion (LSIL).

HPV DNA versus cytological results

Overall HPV prevalence in women with normal cytology was 39/231 (16.9%), while in women with abnormal cytology, it was 6/16 (37.5%) (P = 0.010). In the abnormal cytology group, HPV prevalence was 23.1% (3/13) in (ASCUS) and 100% (3/3) in (LSIL) (Table 1).

Prevalence of single and multiple HPV infections according to cytological results

In the normal cytology group, 31/39 (79.5%) smears had a single infection, compared with 4/6 (66.7%) women with abnormal cytology with a single infection. The prevalence of multiple HPV infections was 2/39 (5.1%) in the normal cytology group, compared to 2/6 (33.3%) in the abnormal cytology group. This association between the type of infection (single/ multiple) and cytology results (normal/abnormal) was marginally significant (P = 0.058) (Table 2).

Distribution of high and low risk HPV infections according to cytology results

Among the normal 39 cytology cases which showed HPV positive, 22 (56.4%) were infected with HR-HPV, 14 (35.9%) with LR-HPV and 3 (7.7%) with both HR-HPV and LR-HPV. The 6 abnormal smears revealed: 4 (66.7%) infected with HR-HPV, one (16.7%) with LR-HPV and one (16.7%) with both. (Table 3).

Distribution of HPV type in women with normal/abnormal cervices

The most frequent HR-HPV strains among 39 normal smears were five cases of HPV 82 (10%), followed by four cases of HPV 68 (8%). The most frequent LR-HPV strains were seven cases of HPV 54 (14%), five of HPV 42 (10%) and three of HPV 44 (6%) (Figure 2).

In the abnormal smear group, HR-HPV 82 was the most frequent type (2 cases). The remaining HR genotypes were HPV 51, 56, 68, 33, 18, 53 and 58 (1 case each).

Association between HPV infection and various characteristics of the studied population

A strong correlation between HIV infection and HPV positivity was observed (OR = 2.741; 95% CI: 0.958–7.84). In addition, HPV positivity was significantly high in women using oral contraceptives (OR = 2.518; 95% CI: 1.183–5.360).

Table 1
Difference between HPV positive and HPV negative cases, according to the cytological result of the study group. Data are presented as numbers (%).

Variables	HPV +	HPV −
Normal cytology (n = 231)	39 (16.9%)	192(83.12%)
[a]ASCUS (n = 13)	3 (23.1%)	10 (76.9%)
[b]LSIL (n = 3)	3 (100%)	0 (0%)

[a] ASCUS: Atypical squamous cell of unknown significance.
[b] LSIL: Low-grade Squamous Intraepithelial Lesion.

Table 2
Prevalence of single and multiple HPV infections according to cytological results.

Type of infection	Cytology results			Total
	Normal	Abnormal	Unsatisfactory	
Single	31	4	0	35
	79.5%	66.7%	0.0%	76.1%
Double	6	0	1	7
	15.4%	0.0%	100.0%	15.2%
Multiple	2	2	0	4
	5.1%	33.3%	0.0%	8.7%
Total	39	6	1	46
	100.0%	100.0%	100.0%	100.0%

Table 3
Risk types distribution according to cytology result cross tabulation.

Risk type	Cytology results			Total
	Normal	Abnormal	Unsatisfactory	
Count	22	4	1	27
HR	56.4%	66.7%	100.0%	58.7%
% within cytology results				
Count	14	1	0	15
LR	35.9%	16.7%	0.0%	32.6%
% within cytology results				
Count	3	1	0	4
HR AND LR	7.7%	16.7%	0.0%	8.7%
% within cytology results				
Count	39	6	1	46
Total				
% within cytology results				
	100.0%	100.0%	100.0%	100.0%

There was no significant association with age, education level, age at sexual debut, marital status and multiparity.

Characteristics of HPV infection in HIV patients

A total of seventeen women (6.6%) had HIV infection, of whom 6 (35.3%) had HPV co-infection (P = 0.052 with OR = 2.74). (Table 4).

The prevalence of HR-HPV genotypes was higher among HIV infected women (P = 0.057), while LR-HPV genotypes were more predominant in the HIV negative group. (See appendix D-24 b). Additionally, patients infected with HIV were substantially more likely to be infected by multiple HPV types than those without HIV infection (P = 0.000). The commonest HR-HPV genotypes detected in HIV infected patients were HPV 53 and HPV-18. (Table 5).

Discussion

The prevalence of HPV in the present study (17.8%) is much higher than in studies from neighbouring countries, such as Iran (7.8%) (Khodakarami et al., 2012), Qatar (6.1%) (Bansal et al., 2014), and KSA (9.8%) (AlObaid et al., 2014), that were conducted on normal and abnormal cytology. Moreover, the HPV prevalence in normal cytology samples in our study is (17%), which is higher than previous reports from studies conducted in the regional countries such as Kuwait and Bahrain on women with normal cytology as the prevalence was (2.4%) and (9.8%) respectively (Al-Awadhi et al., 2011; Moosa et al., 2014).

This high prevalence may be due to the presence of certain factors: First, SQUH and RH are tertiary care hospitals where the majority of women are referred for further clinical management, including HIV cases. Second, the methodological techniques used in this study are among the few assays capable of identifying 28 HPV genotypes that include HR-HPV, LR-HPV and uncommon HR- HPV. These assays are more sensitive than the assays used by

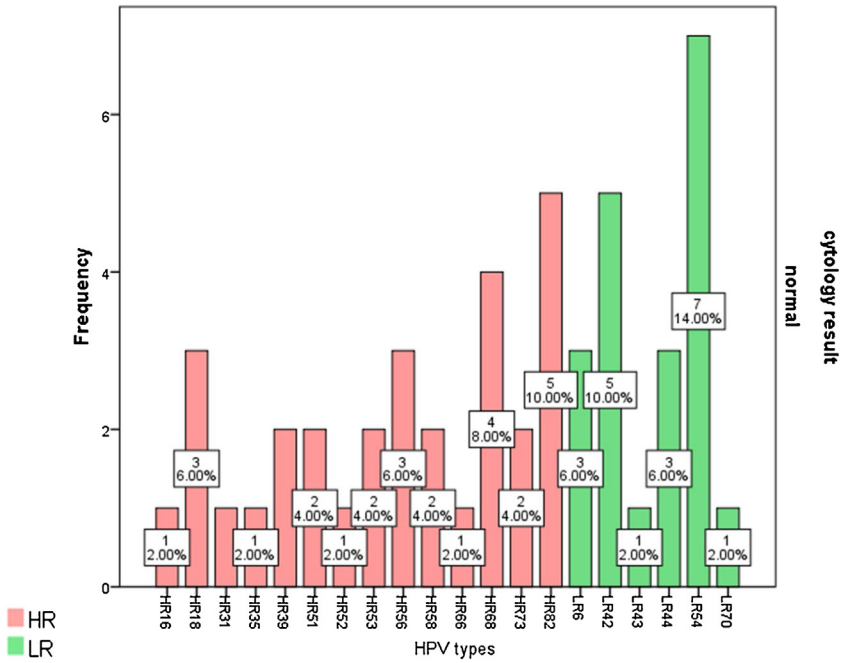

Figure 2. HPV genotypes detected in normal cytology.

Table 4
Association between HPV infection and HIV.

HIV status	# women	HPV positive # %		OR	CI
HIV POS	17	6	35.3%	2.741	0.958-7.84
HIV NEG	241	40	16%	1.00	Ref
Total	258	46			
X^2 for trend					$P = 0.052$

Table 5
HPV genotype distribution among HIV patients.

HIV with HPV	HPV genotypes	Type of infection
1	HPV 56, 68, 33, 43, 44 & 11	Multiple HR and LR
2	HPV 16,18,39 & 35	Multiple HR
3	HPV 42	Single LR
4	HPV 51	Single HR
5	HPV 53 & 73	Double HR
6	HPV 18, 53 & 58	Multiple HR

earlier studies from the region. In addition, the high sensitivity of the test used permits the detection of HPV-DNA in samples with low HPV viral load (Estrade and Sahli, 2014), which might score as HPV negative in alternative assays.

One of the important findings in this study is that the predominant HPV genotypes in Oman are 82 and 54, unlike those seen worldwide, where HPV 16, 18 and other HR-HPV (i.e. 31, 33, 35, 45, 52, and 58) types are the most common HPV infections in women with both normal and abnormal cervices (de Sanjosé et al., 2007). Several studies from the region showed that the distribution of HR- HPV genotypes differ from the global distribution. A study from KSA revealed that HPV 68 and 73 were predominant (AlObaid et al., 2014), and recently HPV 33, 52, 58 and 16 were also detected among the population (Mansour et al., 2018), while in Bahrain the most prevalent HR- HPV found in normal cytology was HPV 52, 16, 31 and 51 (Moosa et al., 2014). In a study from Kuwait conducted among women with abnormal cytology, HR- HPV16 followed by 66 and 33 were detected (Al-Awadhi et al., 2013). However, recently HR genotypes other than types 16, 18, 45 were also identified in Kuwait (Mallik et al., 2018). A study from Qatar revealed that HR HPV 16, 56 and 59 were the most prevalent in their general population (Bansal et al., 2014).

The large meta-analysis study done by de Sanjosé and colleagues classified HPV 82 as a rare oncogenic HPV, but little is known about the exact mechanism of transformation, because of insufficient epidemiological evidence of its presence (de Sanjosé et al., 2010). Interestingly, our finding is in agreement with a Tunisian study which showed high prevalence of HPV 58 and HPV 82 and lack of HPV 18 among female prostitutes (De Marco et al., 2006). The interpretation of rare genotypes in other studies was most likely hampered because of different detection methods used. Majority of these genotype assays can only detect the most common HR and LR types, not the rare or unknown risk types or even the novel types (Co et al., 2013). For instance, the commercially available Hybrid Capture II (HC II, Digene) HR-HPV test does not include the rare HPV, but includes 13 HR- and 5 LR-HPVs, which are most frequently isolated (Coutlée et al., 2005). Other validated molecular assays, such as Abbott Real-Time HR-HPV and Roche Cobas 4800 HPV tests, work as partial HPV genotyping, distinguishing HPV16 or 18 from the other HR types (HPV 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, and 68) as a group (Estrade and Sahli, 2014).

Multiple HPV infections, unlike single infections (Monsonego et al., 2012) have been reported previously and are thought to be associated with increased risk of high-grade lesions and invasive cancer. In the present study, females with normal cytology had mostly single HPV type infections (79.9%), but in women with cytological abnormalities, infections with multiple HPV types were frequently observed (33.3%). Our findings are in line with a previously reported study (Iwasaki et al., 2014).

In the present study, HIV patients and those taking oral contraceptives are the only risk factors associated with HPV infection. HIV showed a significant association with HPV infection ($P = 0.052$), and women with a positive serology for HIV had a significantly higher prevalence of HR-HPV, as observed in other studies conducted among HIV patients (Clifford et al., 2006; Joshi

et al., 2014). Moreover, HIV positive females were substantially infected with multiple HPV types, compared to those who were HIV negative ($P < 0.001$). This finding is in agreement with Levi and colleague's findings, which state that frequent exposure of patients to multiple HPV genotypes is due to unprotected sexual contacts, or failure of the immune system to clear the infections that cause an increase in the persistence rate of HPV (Levi et al., 2002).

In the current study, a statistically significant association was found between HPV infection and oral contraceptive use ($P = 0.016$), a finding similar to studies from Kuwait (Al-Awadhi et al., 2011) and Morocco (Alhamany et al., 2010). However, some studies have shown no association between oral contraceptive and the risk of HPV infection (Baseman and Koutsky, 2005), in addition, there was no association between HPV infection and the stratified age groups ($\leq$25, 25–34, 35–44 and $\geq$45 years). Human papillomavirus prevalence in the developed countries peaks in young women and decreases after 35 years of age (Crosbie et al., 2013). Moreover, other studies showed that there is a second peak in the postmenopausal age groups in some parts of the world, such as Latin America, which are examples of the U-shaped curve of age-specific prevalence (Franceschi et al., 2006; Stanley, 2010).

In this study, there was no evidence of higher prevalence in younger women, confirming the flat age-specific curve across the age groups seen in other low-resource countries in Asia and the EMENA region (Franceschi et al., 2006; Vaccarella et al., 2013).

The main reason that might explain the difference in HPV prevalence and the age distribution between the EMENA countries and other parts of the world is the sexual conservative norms. Most women in the EMENA region are Muslims and typically they get married at an age between 20–26 years, and they continue their life in a monogamous relationship. However, this is not the condition in other countries were sexual activity starts at the age of 18 years with multi-partners. The present study has limitations, whereby the recruited females were attending tertiary care hospitals for different gynecological symptoms which might not be a complete representative of the entire female population, although most were asymptomatic, and the majority had normal cytology and low-grade lesions. In addition, a fraction of HIV-seropositive women has been included that characteristically has been associated with high HPV prevalence, large spectrum of HPV types other than HPV16 and 18, and high frequency of multiple infections. However, the number of HIV infected patients included in this study is too small to affect the prevalence and types of HPV genotypes seen.

The current available vaccines such as the bivalent vaccine Cervarix®, the quadrivalent vaccine Gardasil® and the very recent 9 valent vaccine GARDASIL®9 (Human Papillomavirus 9-valent Vaccine Recombinant) aim to prevent cervical cancer caused by HPV, but not for treatment of an HPV infection. These vaccines target HPV Types 6, 11, 16,18, 31,33, 45, 52, and 58, and genital warts caused by HPV types 6 and 11. All three vaccines address the HR and LR HPV's of western countries. Our study did not include patients with cervical cancer; thus, it is difficult to make any recommendation regarding the best vaccine type for our population. There is an urgent need to conduct a larger scale study both in healthy women with normal cytology and women with abnormal cervical cytology that identifies the predominant genotypes present in the region.

Conclusion

This study is the first in Oman to determine HPV infection, genotypes and its epidemiology. The relatively high prevalence of HPV in women in the region requires further steps to control the spread of high-risk HPV in the future. Our findings indicate that the predominant HPV genotypes in Oman might be different from those seen in worldwide studies. The study highlights the importance of using molecular techniques to enhance cervical screening triage and the need to increase awareness of sexually transmitted diseases and protection measures required to avoid such infections. A larger population-based study is highly recommended, to assess the predominate HPV genotypes, the importance of HPV vaccination for teenage girls, as well as the need to adopt national cervical screening programs in the region.

Funding

Department resources.

Competing interests

None declared.

Ethical approval

The study was approved by the Ethical Committee of the College of Medicine and Health Sciences (COMHS) joint with Sultan Qaboos University Hospital (SQUH) in the letter (MREC #871) and the letter of Research and Ethics Committee (MESR #36) of the Royal Hospital (RH).

Acknowledgements

The authors are grateful to Dr Alia Al-Ansari for offering the use of the new Bio-Rad real time PCR machine in her laboratory and would also like to acknowledge the cytology lab and gynaecology clinic staff for helping in sample collection.

References

Al-Awadhi R, Chehadeh W, Kapila K. Prevalence of human papillomavirus among women with normal cervical cytology in Kuwait. J Med Virol 2011;83(March (3)):453–60.
Al-Awadhi R, Chehadeh W, Jaragh M, Al-Shaheen A, Sharma P, Kapila K. Distribution of human papillomavirus among women with abnormal cervical cytology in Kuwait. Diagn Cytopathol 2013;41(2):107–14.
Alhamany Z, El Mzibri M, Kharbach A, Malihy A, Abouqal R, Jaddi H, et al. Prevalence of human papillomavirus genotype among Moroccan women during a local screening program. J Infect Dev Ctries 2010;4(11):732–9.
AlObaid A, Al-Badawi IA, Al-Kadri H, Gopala K, Kandeil W, Quint W, et al. Human papillomavirus prevalence and type distribution among women attending routine gynecological examinations in Saudi Arabia. BMC Infect Dis 2014;14(1) 643. . Available from: http://bmcinfectdis.biomedcentral.com/articles/10.1186/s12879-014-0643-8.
Bansal D, Elmi A, Skariah S, Haddad P, Abu-Raddad LJ, Al Hamadi A, et al. Molecular epidemiology and genotype distribution of human papillomavirus (HPV) among Arab women in the state of Qatar. J Transl Med 2014;12(1):300–9.
Baseman JG, Koutsky LA. The epidemiology of human papillomavirus infections. J Clin Virol 2005;32:16–24.
Bruni L, Albero G, Serrano B, Mena M, Gómez D, Muñoz J, et al. Human papillomavirus and related diseases report. ICO HPV Information Centre 2014;1–156.
Clifford GM, Gonçalves MAG, Franceschi S. Human papillomavirus types among women infected with HIV: a meta-analysis. AIDS 2006;20(18):2337–44.
Co NNC, Chu L-O, Chow JKF, Tam JWO, Ng EKO. HPV prevalence and detection of rare HPV genotypes in Hong Kong women from Southern China with cytological abnormalities. ISRN Virol 2013;2013:1–5.
Coutlée F, Rouleau D, Ferenczy A, Franco E. The laboratory diagnosis of genital human papillomavirus infections. Can J Infect Dis Med Microbiol 2005;16(2):83–91.
Crosbie EJ, Einstein MH, Franceschi S, Kitchener HC. Human papillomavirus and cervical cancer. Lancet 2013;382(9895):889–99.
Cubie HA. Diseases associated with human papillomavirus infection. Virology 2013;445(1–2):21–34.
De Marco F, Houissa-Kchouk F, Khelifa R, Marcante ML. High-risk HPV types in Tunisia. A pilot study reveals an unexpectedly high prevalence of types 58 and 82 and lack of HPV 18 among female prostitutes. J Med Virol 2006;78(7):950–3.
de Sanjosé S, Diaz M, Castellsagué X, Clifford G, Bruni L, Muñoz N, et al. Worldwide prevalence and genotype distribution of cervical human papillomavirus DNA in women with normal cytology: a meta-analysis. Lancet 2007;7(7):453–9.
de Sanjosé S, Quint W, Alemany L, Geraets D, Klaustermeier J, Lloveras B, et al. Human papillomavirus genotype attribution in invasive cervical cancer: a retrospective cross-sectional worldwide study. Lancet Oncol 2010;11(11):1048–56.

Doorbar J, Quint W, Banks L, Bravo IG, Stoler M, Broker TR, et al. The biology and life-cycle of human papillomaviruses. Vaccine 2012;30(Suppl. 5):F55–70.
Estrade C, Sahli R. Comparison of seegene anyplex II HPV28 with the PGMY-CHUV assay for human papillomavirus genotyping. J Clin Microbiol 2014;52(2):607–12.
Franceschi S, Herrero R, Clifford GM, Snijders PJF, Arslan A, Anh PTH, et al. Variations in the age-specific curves of human papillomavirus prevalence in women worldwide. Int J Cancer 2006;119(11):2677–84.
Iwasaki R, Galvez-Philpott F, Arias-Stella J, Arias-Stella J. Prevalence of high-risk human papillomavirus by cobas 4800 HPV test in urban Peru. Braz J Infect Dis 2014;18(5):469–72.
Joshi S, Babu JM, Jayalakshmi D, Kulkarni V, Divate U, Muwonge R, et al. Human papillomavirus infection among human immunodeficiency virus-infected women in Maharashtra, India. Vaccine 2014;32(9):1079–85.
Khodakarami N, Clifford G, Yavari P, Farzaneh F, Salehpour S, Broutet N, et al. Human papillomavirus infection in women with and without cervical cancer in Tehran, Iran. Int J Cancer 2012;131(2):E156–61.
Levi JE, Kleter B, Quint WGV, Fink MCS, Canto CLM, Matsubara R, et al. High prevalence of human papillomavirus (HPV) infections and high frequency of multiple HPV genotypes in human immunodeficiency virus-infected women in Brazil. J Clin Microbiol 2002;40(9):3341–5.
Mallik MK, Alramadhan B, Dashti H, Al-Shaheen A, Al Juwaiser A, Das DK, et al. Human papillomaviruses other than 16, 18 and 45 are the major high risk HPV genotypes amongst women with abnormal cervical smear cytology residing in Kuwait: implications for future vaccination strategies. Diagn Cytopathol 2018;46:1036–9.
Mansour MA, Elhassan MM, Eltom FM, Yousif HM, Missawi HM, Mohamed EO, et al. Genotyping of high-risk human papilloma virus (HR-HPV) and its role in cervical cancer among suspected women at reproductive age. Afr J Microbiol Res 2018;11(48):1693–8.
Monsonego J, Zerat L, Syrjänen K, Zerat JC, Smith JS, Halfon P. Prevalence of type-specific human papillomavirus infection among women in France: Implications for screening, vaccination, and a future generation of multivalent HPV vaccines. Vaccine 2012;30(35):5215–21.
Moosa K, Alsayyad SS, Quint W, Gopala K, DeAntonio R. An epidemiological study assessing the prevalence of human papillomavirus types in women in the Kingdom of Bahrain. BMC Cancer 2014;14(1):1–8.
Petrosky E, Bocchini JA, Hariri S, Chesson H, Curtis RC, Saraiya M, et al. Use of 9-valent human papillomavirus (HPV) vaccine: updated HPV vaccination recommendations of the advisory committee on immunization practices. Morb Mortal Wkly Rep 2015;64:300–4.
Schrader C, Schielke A, Ellerbroek L, Johne R. PCR inhibitors - occurrence, properties and removal. J Appl Microbiol 2012;113:1014–26.
Stanley M. Pathology and epidemiology of HPV infection in females. Gynecol Oncol 2010;117:S5–S10.
Vaccarella S, Bruni L, Seoud M. Burden of human papillomavirus infections and related diseases in the extended middle east and North Africa Region. Vaccine 2013;31(S6):G32–44.