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ETIOLOGICAL ROLE OF PAPILLOMAVIRUS INFECTION IN WOMEN

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Abstract:
Human Papillomavirus (HPV) is a common virus, transmitted through skin to skin sexual contact. HPV can infect both women and men. HPV infection usually clears on its own with the body's immune system. However, in some people, HPV infection may persist over time

Keywords: Human Papillomavirus

INTRODUCTION: Human papillomavirus (HPV) is a sexually transmit-ted double-stranded DNA virus responsible for the devel-opment of anogenital (cervical, vaginal, vulvar, penile and anal) and oropharyngeal diseases in both women and men. The International Agency for Research on Cancer (IARC) considers it a human carcinogen and classifies HPV 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 and 66 as high-risk (HPV-hr) and HPV 6, 11, 42, 43, 44, 54, 61, 70, 72, 81 as low risk (HPV-lr) types. Some genotypes have an uncertain oncogenic potential. High-risk types 16 and 18 account for almost 70% of all cervical cancers, as well as about 90% of the anal cancers and a variable fraction of vulvar, vaginal, penile and oropharyngeal cancers. HPV genotypes 31, 33, 45, 52 and 58 are the etiological agent for more than 20% of cervical cancers. Low-risk genotypes 6 and 11 mainly cause cervical, vulvar and vaginal low-grade lesions, and are responsible for 90% of the anogenital warts or condy-loma acuminatum [12, 22, 30].

Vaccination represents one of the most cost-effective public health measures. The development of the prophylactic vaccine against HPV, with the aim of preventing the initial infection and subsequent neoplastic transformation, has generated some controversy. Therapeutic vaccines capa-ble of inducing the regression of existing lesions are under development [5, 24, 29].

Human Papillomavirus (HPV) is a common virus, transmitted through skin to skin sexual contact. HPV can infect both women and men. HPV infection usually clears on its own with the body's immune system. However, in some people, HPV infection may persist over time

There are more than 100 HPV types, and each type is identified by a number. Some HPV types can cause genital or anal warts. Other HPV types can cause cancer, most commonly cervical cancer. HPV infection is also a risk factor for vaginal, penile, anal, mouth and throat cancers. Human papillomavirus (HPV) is a common sexually transmitted infection (STI) and a well-known cause of cervical cancer. Several recent studies have demonstrated that HPV infection may be involved in the development of malignant tumors other than cervical cancer, including oral, pharyngeal, anal, and skin cancers. In fact, it has been estimated that approximately 10% of cancer cases worldwide are associated with HPV infection. Among urogenital malignancies, penile cancer is most likely to be associated with HPV infection, as confirmed by many epidemiological studies.

ETIOLOGY: Invasive cervical cancer (ICC) is the third most common cancer among women worldwide. In the middle of the 1970s, the hypothesis that cervical cancer may arise from virus infection was established, and in the 1990s, the causal relationship between genital human papilloma virus (HPV)

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infection and cervical cancer was confirmed. The most significant etiological factor, HPV in the development of both invasive cervical cancer (ICC) and its precursor lesions (cervical intraepithelial neoplasia, CIN) has been well established. It is now widely recognized that HPV infection is a necessary cause for over 99% of cervical cancer cases, and nearly all invasive cervical cancers are indeed positive in HPV DNA test. HPV is a large of group of epitheliotropic viruses of more than 200 different subtypes. Among them, only 40 HPV subtypes could infect the human being. According to its potential to induce carcinogenesis, HPV types have been classified as low-risk oncogenicity (LR-HPV) and high-risk oncogenicity (HR-HPV). Previous studies showed that HR-HPV plays a crucial role in the etiology of anogenital cancer, especially cervical cancer. Many studies had showed that 13 HR-HPV subtypes were significant connected with cervical cancer, namely HPV16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 and 68. Among the high-risk types, infection with HPV16 and 18 are associated with significantly higher risk of disease progression, and consequently these two types together cause approximately 70% of invasive cervical cancer worldwide

The distribution of HPV genotypes in invasive cervical cancer is crucial to guide the introduction of prophylactic vaccines as well. Two prophylactic HPV vaccines are currently available and protect against two carcinogenic HR-HPV types (HPV16 and HPV18). One is a Merck's quadrivalent vaccine preventing infection from 4 HPV types (HPV 6, 11, 16, and 18) (Gardasil), and was approved by FDA in 2006. The second is a bivalent vaccine preventing infection from 2 high-risk oncogenic HPV types (HPV16 and 18) (Cervarix)

CONCLUSION: HPV is a sexually transmitted pathogen responsible for almost all cases of cervical cancer, as well as an important fraction of preinvasive and invasive anogenital and oro-pharyngeal lesions in both sexes. The total fraction of malig-nant and pre-malignant lesions attributed to HPV genotypes contained in the nonavalent vaccine is significant in both women and men, which turns this vaccine into a great asset in terms of Public Health

Currently, three different vaccines are commercialized, varying in the number of HPV genotypes contained. All vac-cines protect against HPV types 16 and 18. The quadriva-lent vaccine also targets types 6 and 11. The nonavalent vaccine, available in Europe since 2015, in addition to the genotypes present in the quadrivalent vaccine, also co-vers types 31, 33, 45, 52, and 58, predicting 90% efficacy in reducing the overall incidence of cancers associated with HPV infection. In Portugal, this vaccine has been part of the National Immunisation Schedule since 2008. The nonava-lent vaccine in the two-dose scheme replaced the quadriva-lent vaccine in January 2017, and has been covering girls aged 10 years.2-6 Vaccination in males is recommended by several national and international scientific societies, and its introduction into the immunisation schedule is currently being discussed in Portugal [16, 25, 28].

CLINICS: The objective of this article is to review the incidence of non-cervical lesions attributed to HPV genotypes contained in the nonavalent vaccine, and to assess its potential impact in terms of Public Health. Though the incidence of cervical cancer in the Extended Middle East and North Africa (EMENA) shows lower rates compared to the rest of the world [2, 7], the burden of HPV infection still warrants public health interventions. In addition, the age specific HPV prevalence has varied widely across different population and showed two peaks of HPV positivity in younger and older women [8,9]. Among the general population of Arab women with normal or abnor-mal cytology residing in Qatar, we recently estimated an HPV prevalence rate of 6.1% [10]. We also identified the presence of a varied genotypic profile of HPV with a high prevalence of low-risk HPV genotype 81. However, HPV DNA testing cannot differentiate between cur-rent and previous infection and does not reflect the lifetime risk of HPV infection. Moreover, despite much progress, risk factors influencing the epidemiology of HPV infection are not yet fully understood [11].

The HPV infection is one of the major concerns nowadays. The virus can be spread by very common routes such as hand contact, sharing objects, sexual contacts, blood, and most surprisingly through inhalation also [30]. Protection from this virus is available only by vaccination. On the clinical trials (phase II) of CervarixTM, the efficacy of preventing HPV18 and 16 infection was 92% while efficacy in the prevention of obstinate infection was 100%. On the other hand, the efficacy of preventing HPV 6, 11, 16, and 18 with GardasilTM was 90% in Phase II and 100% (for HPV 16 and 18) in phase III clinical trial [31,32]. However, vaccination also associated with many limitations like it should be given within 13– 19 before any kind of sexual exposure though it can be spread through hand contact also. All kind of HPV cannot protect by this vaccines. Only a few can be protected. The use of condoms shows very little protection.

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Treatment is also unavailable till date. However, recent studies show some hope to as through surgery and adjuvant therapy or antiretroviral therapy is found to cure HPV-positive oropharyngeal, anal, and cervical cancers [25, 39].

REFERENCES

1. Larke NL, Thomas SL, dos Santos Silva I, Weiss HA. Male circumcision and penile cancer: a systematic review and meta-analysis. Cancer Causes Control 2011;22:1097–110.
2. May M, Burger M, Otto W, Hakenberg OW, Wieland WF, May D, et al. Ki-67, mini-chromosome maintenance 2 protein (MCM2) and geminin have no independent prognostic relevance for cancer-specific survival in surgically treated squamous cell carcinoma of the penis. BJU Int 2013;112:E383–90.
3. Polman NJ, Uijterwaal MH, Witte BI, Berkhof J, van Kemenade FJ, Spruijt JW, et al. Good performance of p16/ki-67 dual-stained cytology for surveillance of women treated for high-grade CIN. Int J Cancer 2017;140:423–30.
4. Sand FL, Rasmussen CL, Frederiksen MH, Andersen KK, Kjaer SK. Prognostic significance of HPV and p16 status in men diagnosed with penile cancer: a systematic review and meta-analysis. Cancer Epidemiol Biomarkers Prev 2018; (July), doi:http://dx.doi.org/10.1158/1055-9965.
5. Shigehara K, Sasagawa T, Kawaguchi S, Nakashima T, Shimamura M, Maeda Y, et al. Etiologic role of human papillomavirus infection in bladder carcinoma. Cancer 2011;117:2067–76.