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Prevention of High Risk HPV Infection Related to Cervical Cancer

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Cervical Cancer; HPV; Prevention; Vaccine.

INTRODUCTION

"It is better to prevent than to cure" is one of the best known maxims of the father of clinical medicine Hippocrates of Kos (460-377 BCE) [1]. For more than two millennia physicians have learned to consider the prevention of any disease a better strategy than treating the disease. The advent of experimental science and the era of evidence-based medicine has led to best ever recognition of disease causes and therefore best ever opportunities of prevention.

Cervical cancer is the third most frequent cancer worldwide in terms of mortality [2]. It is estimated that globally up to 2% of women annually develop advanced precancerous neoplasia of the cervix of CIN stages 2 and 3, cytologically characterized as high-grade squamous intraepithelial lesions, which if undetected or untreated can lead to invasive cervical cancer [2-5]. Almost all such advanced precancerous or cervical cancer cases are caused by infection with high-risk (for cancer) types of the human papillomavirus (HPV) [2-5].

HPV infection is globally the most common sexually transmitted infection, accounting for approximately 270,000 deaths annually, with 88% of them occurring in developing countries [2, 5]. Mounting evidence indicates that high-risk HPV types are the etiologic agents in virtually all cervical cancer cases, in approximately 90% of anal cancers and in a significant percentage (but less than 50%) of vulvar, penile, oral and pharyngeal cancers [3].

Evidence from several studies indicates that most sexually active adults will be infected at least once with HPV in their lifetimes, mostly in genital-tract and mouth in which a concordance both in prevalence rate and HPV type has been noted [6]. HPV infections are known to be most common in young women and to be related to the number of sexual partners [7-9]. Usually these HPV infections appear to be transient, with a typical clearance period within two years [10]. Nevertheless, there are HPV infections that do persist over two years and those are the ones that might be conferring increased risk to women for developing high-grade precancerous lesions or invasive cervical cancer [11,12].

Early detection and treatment of precancerous lesions can obviously prevent cervical cancer. The Pap-smear test developed by George Papanicolaou in the 1950s has been utilized in routine screening programs of many developed countries in order to identify high-grade cervical intraepithelial neoplasia (CIN2+), treat it and prevent its progression to invasive cancer. The realization of such national screening programs has over the years increased the coverage of women taking a Pap-smear test and as a consequence has reduced the occurrence rate of cervical cancer [13].

Molecular characterization of tens of HPV types for more than twenty years has yielded a great amount of data regarding their neoplastic ability and resulted in their classification as high-risk and low-risk types. High-risk HPV types 16 and 18, in particular, appear to be responsible for about 70% of cervical cancer cases and especially HPV 16 for a large percentage of other cancers [2,6-8]. The best prevention strategy for cervical cancer screening seems to be the combination of HPV typing with colposcopy, which provides a more sensitive and efficient detection approach than do methods based solely on cytology [5,14-18].

In recent years, the development of prophylactic vaccines against the most common HPV types has increased hopes

for prevention of viral infection by immunization of early adolescents before any sexual activity [19]. Prophylactic vaccination is expected to have a major impact on the burden of cervical cancer as well as that of other HPV-related cancers [19]. Despite the fact that the currently available vaccines may protect against only a few high-risk HPV types (including 16 and 18), several studies have suggested that vaccination would prevent more than 80% of cervical cancers worldwide [20,21].

Eventually, the future development of new vaccines targeting a broader range of high-risk-HPV types based on the distribution of viral types across populations may protect virtually all women at risk. Until then, cervical neoplasia screening programs should continue to prevent new cases, either by using cost-effective but less efficient cytology, or by using a highly effective combination of colposcopy and HPV typing.

REFERENCES

1. Yapijakis C. (2009). Hippocrates of Kos, the father of clinical medicine, and Asclepiades of Bithynia, the father of molecular medicine. In Vivo. 23(4), 507-514.

2. McGraw SL and Ferrante JM. (2014). Update on prevention and screening of cervical cancer. World J ClinOncol. 5(4), 744-752.

3. Schiffman M, Castle PE, Jeronimo J, Rodriguez AC, et al. (2007). Human papillomavirus and cervical cancer. Lancet. 370(9590), 890-907.

4. McCredie MR, Sharples KJ, Paul C, Baranyai J, et al. (2008). Natural history of cervical neoplasia and risk of invasive cancer in women with cervical intraepithelial neoplasia 3: a retrospective cohort study. Lancet Oncol. 9(5), 425-434.

5. Santesso N, Mustafa RA, Schünemann HJ, Arbyn M, et al. (2016). World Health Organization Guidelines for treatment of cervical intraepithelial neoplasia 2-3 and screen-and-treat strategies to prevent cervical cancer. Int J Gynaecol Obstet. 132(3), 252-258.

6. Adamopoulou M, Vairaktaris E, Nkenke E, Avgoustidis D, et al. (2013). Prevalence of human papillomavirus in saliva and cervix of sexually active women. GynecolOncol. 129(2), 395-400.

7. Vaccarella S, Franceschi S, Herrero R, Muñoz N, et al. (2006). Sexual behavior, condom use, and human papillomavirus: pooled analysis of the IARC human papillomavirus prevalence surveys. Cancer Epidemiol Biomarkers Prev. 15(2), 326-333.

8. de Sanjosé S, Diaz M, Castellsagué X, Clifford G, et al. (2007). Worldwide prevalence and genotype distribution of cervical human papillomavirus DNA in women with normal cytology: a meta-analysis. Lancet Infect Dis. 7(7), 453-459.

9. Datta SD, Koutsky LA, Ratelle S, Unger ER, et al. (2008). Human papillomavirus infection and cervical cytology in women screened for cervical cancer in the United States, 2003-2005. Ann Intern Med. 148(7), 493-500.

10. Plummer M, Schiffman M, Castle PE, Maucort-Boulch D, et al. (2007). A 2-year prospective study of human papillomavirus persistence among women with a cytological diagnosis of atypical squamous cells of undetermined significance or low-grade squamous intraepithelial lesion. J Infect Dis. 195(11), 1582-1589.

11. Castle PE, Rodríguez AC, Burk RD, Herrero R, et al. (2009). Short term persistence of human papillomavirus and risk of cervical precancer and cancer: population based cohort study. BMJ. 339, b2569.

12. Kjær SK, Frederiksen K, Munk C and Iftner T. (2010). Long-term absolute risk of cervical intraepithelial neoplasia grade 3 or worse following human papillomavirus infection: role of persistence. J Natl Cancer Inst. 102(19), 1478-1488.

13. Peto J, Gilham C, Deacon J, Taylor C, et al. (2004). Cervical HPV infection and neoplasia in a large population-based prospective study: the Manchester cohort. Br J Cancer. 91(5), 942-953.

14. Adamopoulou M, Kalkani E, Charvalos E, Avgoustidis D, et al. (2009). Comparison of cytology, colposcopy, HPV typing and biomarker analysis in cervical neoplasia. Anticancer Res. 29(8), 3401-3409.

15. Saslow D, Solomon D, Lawson HW, Killackey M, et al. (2012). American Cancer Society, American Society for Colposcopy and Cervical Pathology, and American Society for Clinical Pathology screening guidelines for the prevention and early detection of cervical cancer. CA Cancer J Clin. 62(3), 147-172.

16. Ibáñez R, Autonell J, Sardà M, Crespo N, et al. (2014). Protecting the underscreened women in developed countries: the value of HPV test. BMC Cancer. 14, 574.

17. Cuzick J, Myers O, Hunt WC, Saslow D, et al. (2015). Human papillomavirus testing 2007-2012: co-testing and triage utilization and impact on subsequent clinical management. Int J Cancer. 136(12), 2854-2863.

18. Castle PE, Stoler MH, Wright TC Jr, Sharma A, et al. (2011). Performance of carcinogenic human papillomavirus (HPV) testing and HPV16 or HPV18 genotyping for cervical cancer screening of women aged 25 years and older: a subanalysis of the ATHENA study. Lancet Oncol. 12(9), 880-890.

19. Tota JE, Chevarie-Davis M, Richardson LA, Devries M, et al. (2011). Epidemiology and burden of HPV infection and related diseases: implications for prevention strategies. Prev Med. 53 Suppl 1, S12-21.

20. Wheeler CM, Castellsagué X, Garland SM, Szarewski A, et al. (2012). Cross-protective efficacy of HPV-16/18 AS04-adjuvanted vaccine against cervical infection and precancer caused by non-vaccine oncogenic HPV types: 4-year end-of-study analysis of the randomised, double-blind PATRICIA trial. Lancet Oncol. 13(1), 100-110.

21. Lehtinen M, Apter D, Baussano I, Eriksson T, et al. (2015). Characteristics of a cluster-randomized phase IV human papillomavirus vaccination effectiveness trial. Vaccine. 33(10), 1284-1290.