HPV18 DNA levels are not prognostic for precancerous cervical lesions

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Perhaps surprisingly, the number of copies of the carcinogenic human papillomavirus type 18 (HPV18) relative to cellular DNA is not associated with the likelihood of progression to advanced precancerous lesions of the cervix, according to a study in the January 27 online issue of the Journal of the National Cancer Institute.

Two types of HPV are most frequently associated with cervical cancer, HPV16 and HPV18. Previous studies showed that the number of HPV16 copies per cell correlated with an increasing risk of progression to cervical intraepithelial neoplasia grade 2 or 3 (CIN2-3). The prognostic significance of HPV18 DNA level is not known.

In the current study, Long Fu Xi, M.D, Ph.D., of the University of Washington in Seattle, and colleagues compared the number of copies of HPV18 DNA relative to cellular DNA at baseline with a woman's risk of progressing to CIN2-3. The 303 study participants were drawn from the Atypical Squamous Cells of Undetermined Significance and Low-Grade Squamous Intraepithelial Lesion Triage Study.

During the 2-year study period, 92 women were diagnosed with CIN2-3. Among women with a cytologic diagnosis of low- or high-grade squamous intraepithelial lesions at enrollment, HPV18 DNA level was lower in women with CIN2-3 than those without CIN2-3.

"In summary, our data indicated that HPV18 DNA levels were highest among women with evidence of a benign squamous intraepithelial lesion, intermediate among those with CIN2-3, and lowest among those with normal cytological findings," the authors write. "Thus, testing for high levels of HPV18 DNA does not appear to be clinically useful."

In an accompanying editorial, Eduardo Franco, Dr.P.H., and François Coutlée, M.D., of McGill University in Montreal, agree with the authors' conclusion and applaud their effort to elucidate the underlying biology of cervical cancer. "As far as clinical practice is concerned, the obvious conclusion from the study of Xi et al. is that quantifying the HPV18 DNA load may not have the same value as for HPV16," the editorialists write. "That said, the findings from this study considerably extend our appreciation for the heterogeneity of molecular events and their cellular targets in cervical carcinogenesis."

Citations:


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