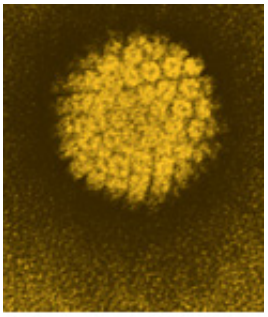
High-risk HPV present in subset of penile carcinomas

August 13 2012



Electron micrograph of HPV. Photo courtesy: U.S. National Institutes of Health.

High-risk human papillomavirus infection is found in a subset of penile squamous cell carcinomas that may develop from undifferentiated penile intraepithelial neoplasia, according to a study published online Aug. 6 in the *Journal of the American Academy of Dermatology*.

(HealthDay) -- High-risk human papillomavirus (hrHPV) infection is found in a subset of penile squamous cell carcinomas (PSCCs) that may develop from undifferentiated penile intraepithelial neoplasia (PeIN), according to a study published online Aug. 6 in the *Journal of the American Academy of Dermatology*.

Carla Ferrándiz-Pulido, M.D., from the Hospital Universitari Vall d'Hebron in Barcelona, Spain, and colleagues examined the prevalence of HPV in a retrospective series of 82 patients with PSCC (69 invasive

and 13 PeIN). Polymerase chain reaction assay with SPF-10 broad-spectrum primers followed by DNA enzyme immunoassay was used for HPV detection, and genotyping was performed using a reverse hybridization line probe assay.

The researchers identified HPV DNA in 31 of 77 (40.2 percent) PSCC cases (22 of 67 invasive and nine of 10 PeIN). HPV-16 was identified in 25 of 31 (80.6 percent) cases. Most basaloid and warty tumors were hrHPV positive, while only 15 percent of usual PSCC were hrHPV positive. All hrHPV-positive PSCC had an adjacent undifferentiated PeIN. The researchers found that hrHPV infection correlated with strong $p16^{INK4a}$ immunostaining, and $p16^{INK4a}$ immunohistochemical overexpression was present in most undifferentiated PeIN. There was better overall survival, although not statistically significant, in both hrHPV-positive and $p16^{INK4a}$-positive tumors.

"In this study, we detected hrHPV in 28 percent of PSCC and in 90 percent of PeIN," the authors write. "These results allow identification of a subset of PSCC in which HPV would play a triggering role and give support to the bimodal etiopathogenic hypothesis that distinguishes two different subsets of PSCC."

More information: Abstract
Full Text (subscription or payment may be required)