Condylomata Acuminata and Human Genital Cancer

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The subsequent review by Goldberg and Gravel (8) stresses the possible role of HSV-2* infections in human cervical cancer. Although seroepidemiological studies do suggest an association of HSV-2 with this type of cancer (12, 14), repeated attempts, with 1 exception (6), have failed to demonstrate viral DNA by nucleic acid hybridizations in cervical cancer cells (Refs. 19 and 21; H. Schulte-Holthausen and H. zur Hausen, unpublished data), even when assays were performed under very sensitive conditions. Although these negative results are not yet conclusive, additional arguments have been raised that do not support the HSV-2 etiology of cervical cancer (19).

It is the intention of this note to draw attention to a virus-induced and virus particle-containing tumor revealing a similar epidemiological pattern, as does cervical cancer: the genital warts or condylomata acuminata. Condylomata acuminata have been shown to contain a papilloma virus (5, 13), which appears to differ from human wart virus when tested biochemically (20) or immunologically (1). These particles can be detected by electron microscopy in about 50% of all condylomas examined (13). Attempts to grow this virus in tissue culture or to isolate it directly from condylomata have not been very successful in the past (unpublished data).

Genital warts seem to be transmitted primarily by sexual contact and are predominant in populations of high sexual promiscuity. In contrast to common warts, condylomata may become malignant (although rarely), and there exist a number of clinical observations on malignant transition of this tumor (3, 4, 7, 9–11, 15, 17). In particular, penile carcinomas have been observed to develop within or at a site of condylomata acuminata (2, 16, 18).

The condyloma agent has been entirely neglected thus far in all epidemiological and serological studies relating not only to cervical and penile, but also to vulvar and perianal, carcinomas. This is particularly unusual in view of the localization of genital warts, their mode of venereal transmission, the number of reports on malignant transition, and the presence of an agent belonging to a well-characterized group of oncogenic DNA viruses.

References


* The abbreviation used is: HSV-2, herpes simplex virus type 2.
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