Genital Warts

Of genital warts, 90% are caused by HPV 6 or 11. HPV types 6 or 11 are commonly found before, or at the time of, detection of genital warts (406). HPV types 16, 18, 31, 33, and 35 are found occasionally in visible genital warts (usually as coinfections with HPV 6 or 11) and can be associated with foci of high-grade intraepithelial neoplasia, particularly in persons who are infected with HIV infection. In addition to warts on genital areas, HPV types 6 and 11 have been associated with conjunctival, nasal, oral, and laryngeal warts.

Genital warts are usually asymptomatic, but depending on the size and anatomic location, they can be painful or pruritic. Genital warts are usually flat, papular, or pedunculated growths on the genital mucosa. Genital warts occur commonly at certain anatomic sites, including around the introitus in women, under the foreskin of the uncircumcised penis, and on the shaft of the circumcised penis. Genital warts can also occur at multiple sites in the anogenital epithelium or within the anogenital tract (e.g., cervix, vagina, urethra, perineum, perianal skin, and scrotum). Intra-anal warts are observed predominantly in persons who have had receptive anal intercourse, but they can also occur in men and women who do not have a history of anal sexual contact.

Diagnosis of genital warts is usually clinical, made by visual inspection. Genital warts can be confirmed by biopsy, which might be indicated if 1) the diagnosis is uncertain; 2) the lesions do not respond to standard therapy; 3) the disease worsens during therapy; 4) the lesion is atypical; 5) the patient has comprised immunity; or 6) the warts are pigmented, indurated, fixed, bleeding, or ulcerated. Genital warts are usually asymptomatic, but depending on the size and anatomic location, they might be painful or pruritic. The use of HPV DNA testing for genital wart diagnosis is not recommended, because test results would not alter clinical management of the condition.

The application of 3%–5% acetic acid, which causes skin color to turn white, has been used by some providers to detect HPV-infected genital mucosa. However, acetic acid application is not a specific test for HPV infection. Therefore, the routine use of this
procedure for screening to detect mucosal changes attributed to HPV infection is not recommended.

Treatment
The primary reason for treating genital warts is the amelioration of symptoms (including relieving cosmetic concerns) and ultimately, removal of the warts. In most patients, treatment can induce wart-free periods. If left untreated, visible genital warts can resolve on their own, remain unchanged, or increase in size or number. Available therapies for genital warts likely reduce, but probably do not eradicate, HPV infectivity. Whether the reduction in HPV viral DNA resulting from treatment reduces future transmission remains unclear. No evidence indicates that the presence of genital warts or their treatment is associated with the development of cervical cancer.

Regimens
Treatment of genital warts should be guided by the preference of the patient, available resources, and the experience of the health-care provider. No definitive evidence suggests that any of the available treatments are superior to any other, and no single treatment is ideal for all patients or all warts. The use of locally developed and monitored treatment algorithms has been associated with improved clinical outcomes and should be encouraged. Because of uncertainty regarding the effect of treatment on future transmission of HPV and the possibility of spontaneous resolution, an acceptable alternative for some persons is to forego treatment and wait for spontaneous resolution.

Factors that influence selection of treatment include wart size, wart number, anatomic site of the wart, wart morphology, patient preference, cost of treatment, convenience, adverse effects, and provider experience. Factors that might affect response to therapy include the presence of immunosuppression and compliance with therapy, which can consist of either a single treatment or complete course of treatment. In general, warts located on moist surfaces or in intertriginous areas respond best to topical treatment. The treatment modality should be changed if a patient has not improved substantially after a complete course of treatment or if side effects are severe. Most genital warts respond within 3 months of therapy. The response to treatment and any side effects should be evaluated throughout the course of therapy.

Complications occur rarely when treatment is administered properly. Patients should be warned that persistent hypopigmentation or hyperpigmentation occurs commonly with ablative modalities and has also been described with immune modulating therapies (imiquimod). Depressed or hypertrophic scars are uncommon but can occur, especially if the patient has had insufficient time to heal between treatments. Rarely, treatment can result in disabling chronic pain syndromes (e.g., vulvodynia and hyperesthesia of the treatment site) or, in the case of anal warts, painful defecation or fistulas. A limited number of case reports of severe systemic effects resulting from treatment with podophyllin resin and interferon have been documented.

Treatment regimens are classified into patient-applied and provider-applied modalities. Patient-applied modalities are preferred by some patients because they can be
administered in the privacy of the patient’s home. To ensure that patient-applied modalities are effective, patients must comply with the treatment regimen and must be capable of identifying and reaching all genital warts. Follow-up visits are not required for persons using patient-applied therapy. However, follow-up visits after several weeks of therapy enable providers to answer any questions patients might have about the use of the medication and any side effects they have experienced; follow-up visits also facilitate the assessment of a patient’s response to treatment.