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GENITAL WARTS (CONDYLOMATA ACUMINATA): CURRENT PERSPECTIVES ON EPIDEMIOLOGY, DIAGNOSIS, TREATMENT AND PREVENTION. – A REVIEW

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ABSTRACT

Human papillomavirus (HPV) is the most prevalent sexually transmitted infection globally, responsible for a wide spectrum of epithelial lesions ranging from benign warts to invasive malignancies of the cervix, vulva, anus, and oropharynx. More than 200 genotypes have been identified, with low-risk types such as HPV-6 and HPV-11 linked to genital warts, and high-risk types, notably HPV-16 and HPV-18, associated with oncogenic transformation. Transmission occurs primarily through direct sexual contact, although nonsexual and vertical routes have been described. The majority of infections remain asymptomatic and transient, yet persistent infection with high-risk genotypes can lead to neoplastic progression. Diagnosis is mainly clinical, supported by histopathology and molecular HPV DNA detection when indicated. Current management focuses on lesion removal through topical agents (podophyllotoxin, imiquimod, sinecatechins) and ablative or surgical techniques, though recurrence remains frequent. Preventive strategies are central to disease control, with prophylactic HPV vaccination demonstrating remarkable efficacy in reducing infection rates and HPV-related disease burden worldwide. The bivalent, quadrivalent, and nonavalent vaccines provide durable protection for at least 10–14 years and are recommended before sexual debut. Expanding global vaccine coverage, combined with continued screening and education, remains vital to reducing HPV-related morbidity and mortality.

KEYWORDS

Human Papillomavirus (HPV), Genital Warts, HPV Vaccination, Prevention, Diagnosis, Treatment, Condyloma Acuminata, Sexually Transmitted Infections

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1. Introduction

Human papillomavirus (HPV) is a widespread sexually transmitted infection responsible for a broad spectrum of epithelial lesions affecting both cutaneous and mucosal surfaces, and is also a well-established etiological factor in the development of several anogenital and oropharyngeal cancers. There are more than 200 identified HPV types. [1,2] Only around 10% of individuals infected with HPV are capable of transmitting the virus. The infection can be spread through direct skin-to-skin contact with an infected person, typically during sexual interaction. One person may contract multiple HPV genotypes at the same time.[3] During their lifetime, most sexually active men and women will contract HPV at least once, although the majority will not develop any clinical disease. [5] Depending on the viral genotype, HPV infections may manifest as benign warts or progress to malignant lesions. Low-risk types, such as HPV-6 and HPV-11, are primarily associated with genital warts, whereas high-risk types, notably HPV-16 and HPV-18, are strongly linked to intraepithelial neoplasia and cancers of the cervix, vulva, anus, and oropharynx [1,2]. Factors such as age, lifestyle habits, and sexual behavior influence an individual's susceptibility to developing genital warts. Various topical therapeutic agents are available, including podophyllotoxin in solution or cream form, imiquimod cream, and sinecatechins ointment. Additional treatment options include ablative methods such as cryotherapy, chemical destruction with trichloroacetic acid, and different surgical approaches. Recurrence after topical therapy is common, whereas surgical excision demonstrates the highest clearance rates, approaching nearly 100%. The currently available HPV vaccines - bivalent, quadrivalent, and nine-valent - have demonstrated efficacy in reducing both HPV infection rates and the incidence of HPV-related diseases across multiple regions worldwide. [5] Patient education regarding treatment choices, the necessity of regular follow-up, and the practice of safe sexual behaviors is essential for effective management and prevention of reinfection. [4]

2. Etiology

Human papillomavirus (HPV) is a small, non-enveloped, double-stranded circular DNA virus belonging to the Papillomaviridae family [2]. The viral particle measures approximately 50–60 nm in diameter and has an icosahedral structure composed of 72 capsomeres made of L1 and L2 proteins. The HPV genome contains about 8,000 base pairs and encodes eight early (E1–E7, E8) and two late (L1, L2) proteins, in addition to a long control region (LCR) that regulates viral replication and transcription. The L1 protein, the major capsid component, forms pentameric units that assemble into virus-like particles (VLPs). It is highly immunogenic due to the presence of surface loops that determine antigenic specificity and induce neutralizing antibodies, which is why it serves as the basis for current HPV vaccines [6].

Over 200 HPV genotypes have been identified, of which approximately 40 infect the anogenital tract [4]. Based on their oncogenic potential, HPV types are classified as low-risk or high-risk. Low-risk genotypes, mainly HPV-6 and HPV-11, are responsible for about 90% of cases of genital warts whereas high-risk types, such as HPV-16 and HPV-18, are strongly associated with cervical, anal, vulvar, penile, and oropharyngeal cancers [1,4]. Other high-risk types - including HPV-31, -33, -35, -39, -45, -51, -52, -56, -58, and -59 may also contribute to anogenital and mucosal malignancies [4]. According to the International Agency for Research on Cancer (IARC), HPV is among the seven viruses classified as carcinogenic to humans [1]. HPV infection is primarily acquired through sexual contact, including both penetrative and non-penetrative activity, with the virus entering the host through microscopic abrasions in the skin or mucosa [3]. Multiple sexual partners, early sexual debut, smoking, long-term oral contraceptive use, and immunosuppression have been identified as major risk factors for HPV acquisition and persistence [1,2].

3. Epidemiology

Human papillomavirus (HPV) infection is the most common sexually transmitted infection worldwide, affecting both men and women across all regions and age groups [1,2]. Globally, the prevalence of HPV infection is estimated at 9–13% in the general population, with higher rates in low- and middle-income countries [5]. Among women with normal cervical cytology, prevalence ranges from 10% to 12%, but can exceed 20% in Sub-Saharan Africa and Oceania [5]. The majority of infections occur in individuals aged 17 to 33 years, with a peak incidence between 20 and 24 years [3]. In men, genital HPV infection affects approximately 20–50% of sexually active individuals, with prevalence rates similar to or slightly lower than those observed in women [5]. HPV transmission occurs primarily through sexual contact, although non-sexual routes such as self-inoculation, vertical transmission from mother to child, and contact with contaminated medical instruments have been reported [1,6]. Most infected individuals remain asymptomatic, facilitating silent viral spread. Clinical manifestations such as genital warts develop in about 1% of infected persons [3]. HPV types 6 and 11 cause approximately 90% of genital warts, while high-risk oncogenic types 16 and 18 are implicated in the majority of cervical, anal, and oropharyngeal cancers [2,5]. The regional distribution of HPV infection shows considerable variability. The highest rates are observed in Sub-Saharan Africa (24–33%), Eastern Europe (21%), and Latin America (15–20%), while developed regions such as Western Europe and North America show lower prevalence, often below 10%, largely due to effective vaccination programs and screening initiatives [5]. Recent studies demonstrate that HPV vaccination has significantly reduced the incidence of genital warts, particularly among young women and heterosexual men through herd immunity [3,9]. However, in areas with limited vaccine coverage, the burden of HPV-related diseases continues to rise, highlighting the importance of preventive measures and global vaccination access [4,5]

4. Pathophysiology

Human papillomavirus (HPV) is a non-enveloped, icosahedral, double-stranded DNA virus with a genome of approximately 8 kb consisting of eight or nine open reading frames (ORFs) [10]. The viral genome is divided into three major regions: the early (E) region, encoding proteins responsible for replication and transcriptional regulation (E1–E7); the late (L) region, which encodes structural capsid proteins (L1 and L2); and the long control region (LCR), which contains regulatory sequences essential for viral DNA replication [10]. The HPV life cycle begins when the virus gains access to the basal layer of the squamous epithelium, typically through microabrasions that expose basal epithelial stem cells to infection. As a highly epitheliotropic virus, HPV establishes infection within the nucleus of differentiating keratinocytes. The infection can remain latent for several months, corresponding to an incubation period ranging from one month to two years [4].

During the early phase of infection, viral genes E1 and E2 initiate replication, while E5, E6, and E7—known as oncoproteins—play key roles in cellular transformation [10]. E6 and E7 are particularly important

in oncogenesis: E6 promotes degradation of the tumor suppressor protein p53 and enhances telomerase activity, while E7 inactivates the retinoblastoma protein (pRb), leading to dysregulation of the cell cycle and uncontrolled proliferation of infected epithelial cells [2,10]. These changes prevent the infected keratinocytes from entering the resting (G0) phase, maintaining them in a proliferative state that allows viral replication to persist [2]. E7 also induces epigenetic reprogramming and cell cycle re-entry, while the combined actions of E6 and E7 facilitate viral genome amplification in suprabasal epithelial layers [10].

As infected basal cells migrate upward, viral DNA replication continues, resulting in hypertrophy and acanthosis of the affected epithelium, which manifest clinically as warty lesions such as condylomata acuminata [4]. In most immunocompetent individuals, the immune system eventually halts viral replication, leading to spontaneous regression of lesions [4]. However, persistent infection with high-risk HPV types, especially 16 and 18, can lead to genomic instability and neoplastic transformation of the epithelium [2,10]. The completed viral particles are assembled in the upper epithelial layers and released as the keratinocytes undergo terminal differentiation and desquamation [10]. HPV transmission between individuals occurs mainly through direct skin-to-skin or mucosal contact during vaginal, anal, or oral sex, and can also occur through autoinoculation from one site to another [11].

5. Histopathology

Histopathological examination of lesions caused by human papillomavirus (HPV) infection reveals characteristic microscopic changes within the stratified squamous epithelium. Genital warts are exophytic lesions that arise from proliferation of the dermal papillae and hyperplasia of the overlying squamous epithelium [3]. These lesions are typically diagnosed clinically, as their morphological features are distinctive, and biopsy is rarely necessary for confirmation [4].Under the microscope, condyloma acuminatum demonstrates **acanthosis**, **papillomatosis**, and **hyperkeratosis**, often accompanied by **parakeratosis**—retention of nuclei within the stratum corneum [2,4]. The elongated rete ridges tend to converge toward the center of the lesion, and small capillaries within the dermal papillae are frequently dilated or thrombosed [2]. The most pathognomonic finding in HPV-induced lesions is the presence of **koilocytes**—large squamous epithelial cells with perinuclear halos, irregular and hyperchromatic nuclei, and cytoplasmic vacuolization [3,4]. These cells, which result from viral replication and cytopathic effect, are typically located in the upper layers of the epithelium.condyloma acuminatum can be distinguished histologically from **verruca vulgaris** (common warts) by the pattern of epithelial hyperplasia: verruca vulgaris exhibits pointed, spiked rete ridges, whereas condyloma acuminatum shows broad-based papillomatosis with blunted projections [4]. In lesions associated with low-risk HPV types (6 and 11), atypia is minimal or absent, and the basement membrane remains intact, confirming their benign nature. Conversely, infection with high-risk HPV genotypes may induce nuclear atypia and increased mitotic activity, signifying progression toward dysplastic or neoplastic transformation [3].

6. Diagnosis

Accurate diagnosis of genital warts (condylomata acuminata) is crucial for appropriate management and differentiation from other genital lesions. Clinically, warts may appear as flat, dome-shaped, pedunculated, keratotic, or cauliflower-like lesions and may present singly, in clusters, or as confluent plaques [12]. Their size and appearance can vary — from small papules less than 5 mm in diameter to large exophytic masses that may involve the perianal or genital regions [3]. The color of these lesions typically matches the surrounding skin but can occasionally be darker or erythematous. Some patients experience itching, pain, or mild bleeding, while others remain asymptomatic; in fact, visible warts are often the only clinical manifestation of HPV infection [3].

The diagnosis of external or anogenital warts is primarily clinical, based on direct visual inspection under adequate illumination and magnification [12]. In men, the meatus and fossa navicularis should be carefully examined using an otoscope or small speculum to ensure complete mucosal assessment. Routine biopsy is not recommended. However, histopathological examination should be performed when lesions exhibit atypical features — such as pigmentation, rapid growth, ulceration, fixation to deeper tissues, or failure to respond to standard treatment — or when malignancy is suspected [8,12]. This is particularly important in immunocompromised individuals (e.g., patients with HIV infection), who present a higher risk of intraepithelial neoplasia or squamous cell carcinoma associated with HPV [8].

The differential diagnosis includes other dermatologic and infectious conditions that can mimic genital warts: condyloma lata in secondary syphilis, seborrheic keratoses, molluscum contagiosum, pearly penile

papules, and various benign or dysplastic nevi [12]. Moreover, certain neoplastic processes such as Bowen's disease, Bowenoid papulosis, and Buschke–Löwenstein tumors may resemble genital warts but require histologic confirmation due to their malignant potential [12].

Although acetowhite testing and HPV genotyping assays have been historically proposed as diagnostic adjuncts, they are not recommended for routine clinical use, as their diagnostic specificity is low and they rarely alter management decisions [12]. Similarly, molecular methods like hybrid capture assays are not part of standard diagnostic protocols for genital warts, as their clinical benefit in visible lesions is limited [12].

However, in broader HPV-related disease surveillance, HPV DNA testing has become an important molecular diagnostic tool, particularly in screening for oncogenic HPV infections of the cervix [13,14]. Current evidence supports its use as a primary screening method for women aged 30 years and older, with testing intervals of five years [13]. When oncogenic HPV types are detected, reflex cytological evaluation (Pap smear) should be performed to assess for dysplasia [13]. A significant advancement in molecular diagnostics is the introduction of self-sampling techniques, which demonstrate detection rates comparable to clinician-collected specimens, thereby improving access to screening in resource-limited settings [13].

From a molecular standpoint, detection and genotyping of HPV DNA rely primarily on polymerase chain reaction (PCR)-based methods, targeting specific viral genes such as L1, L2, and E1 [14]. These genes encode viral capsid or replication-associated proteins and are key markers used in commercial diagnostic assays. Molecular techniques remain essential in research and in cases requiring precise HPV typing, given that HPV cannot be efficiently cultivated in traditional culture systems [14].

7. Treatment

The management of human papillomavirus (HPV) infections and associated lesions, such as condyloma acuminata (genital warts), focuses primarily on the removal of visible lesions, alleviation of symptoms, and improvement of the patient's quality of life. None of the currently available therapies eradicate the virus completely, as HPV can remain latent within epithelial cells; thus, recurrence after treatment is common [15–17].

In many young, immunocompetent individuals—including adolescents and children—genital warts may spontaneously regress over months or years. Therefore, treatment can be delayed unless the lesions persist for more than two years, cause discomfort, or pose cosmetic concerns. Therapy is indicated for symptomatic, progressive, or psychologically distressing lesions [4].

Topical therapies

Topical therapy remains the first-line treatment for many patients due to its accessibility, non-invasive nature, and ability to be self-administered. Commonly used topical agents include **podophyllotoxin, imiquimod, sinecatechins**, and **trichloroacetic acid (TCA)** [15,17].

Podophyllotoxin (0.5% solution or 0.15% cream) is a plant-derived antimitotic compound that disrupts microtubule polymerization, leading to localized tissue necrosis within several days. The recommended regimen involves application twice daily for three consecutive days, followed by a four-day rest period; this cycle is repeated for up to six weeks. Lesions often resolve within four weeks of therapy. It is considered safe and effective but may cause local erythema, edema, and erosions. Podophyllotoxin should not be used during pregnancy due to potential systemic toxicity [4,15].

Imiquimod, available as a 5% cream, is an immune response modifier that acts as a toll-like receptor 7 (TLR7) agonist. It induces the release of pro-inflammatory cytokines such as interferon-alpha and tumor necrosis factor-alpha, thereby stimulating a Th1-mediated immune response against HPV-infected cells. It is applied three times per week and typically achieves lesion clearance within 12–16 weeks. Recurrence rates are lower than with podophyllotoxin. Local irritation, burning, erythema, and mild flu-like symptoms are the most common side effects [4,15].

Sinecatechins (15% ointment) are a botanical extract derived from green tea (*Camellia sinensis*) that contains catechins with anti-inflammatory, antioxidant, and antiviral properties. The preparation inhibits HPV E6 and E7 oncoprotein expression and induces apoptosis in infected keratinocytes. It is applied three times daily for up to 16 weeks, with efficacy comparable to imiquimod but fewer local side effects [4,17].

Trichloroacetic acid (TCA) (80–90%) acts as a chemical cauterizing agent that destroys infected epithelium via protein coagulation. It is typically applied weekly for 8–10 sessions. TCA is cost-effective and widely available but carries a recurrence rate of 30–40%. Healing generally occurs without scarring. Due to its corrosive properties, TCA should be administered only by trained healthcare professionals [4,15,20].

Physical and surgical methods

In cases of extensive, keratinized, or treatment-resistant lesions, **ablative procedures** such as **cryotherapy, electrocautery, CO_2 laser therapy, curettage, or surgical excision** are indicated [4,15,17].

Cryotherapy with liquid nitrogen or nitrous oxide is a safe, inexpensive, and effective treatment, including for pregnant patients. The lesion is frozen for approximately 20 seconds, repeated three to five times per session, with weekly treatments for 6–10 weeks. The process causes necrosis and immune-mediated clearance of infected tissue. Although recurrence rates are low, the main drawbacks are pain during the procedure and the need for multiple sessions [4,15].

CO_2 laser therapy uses focused infrared light to vaporize lesions with excellent precision and minimal bleeding. The method is effective for refractory or large lesions and provides rapid healing with minimal scarring. However, it is expensive, requires specialized equipment and training, and carries a risk of aerosolized viral particles; therefore, appropriate protective measures (filtration masks and smoke evacuation systems) are necessary [15].

Electrocautery and **simple surgical excision** are alternative options for large or recurrent lesions. Surgical methods achieve clearance rates approaching 100% but are associated with postoperative pain and scarring. **Photodynamic therapy (PDT)** using **5-aminolevulinic acid (ALA)** has recently emerged as an effective alternative. PDT selectively destroys HPV-infected cells via reactive oxygen species generation and has demonstrated lower recurrence rates and better cosmetic outcomes compared with CO_2 laser ablation. It also treats subclinical lesions, reducing the risk of reinfection [4,20].

Adjunctive and emerging therapies

For patients with recalcitrant or widespread disease, particularly those with immunosuppression, adjunctive therapies may be considered. These include **interferon therapy**, **intralesional vitamin D injections**, and **isotretinoin**, which has demonstrated anti-inflammatory and antiproliferative properties [17].

Recent research focuses on the development of **molecular and immunotherapeutic strategies** targeting viral oncoproteins **E6, E7, and E1** to inhibit viral replication and induce apoptosis of infected cells [17,18]. Other promising approaches include plant-derived compounds such as **curcumin, resveratrol, silymarin**, and **berberine**, as well as nanotechnology-based agents like **gold nanoparticles** and **cell-penetrating peptides** that prevent viral entry or replication.

Immunotherapeutic combinations—integrating checkpoint inhibitors, therapeutic HPV vaccines, or cytokine-based therapies with standard treatments—are being explored to enhance local and systemic antiviral immunity [18].

8. Prevention and Vaccination

Human papillomavirus (HPV) infection remains one of the most common sexually transmitted diseases worldwide, with a well-established role in the pathogenesis of cervical, anogenital, and oropharyngeal cancers. Since there is currently no curative therapy for HPV infection itself, prevention plays a pivotal role in controlling its spread and reducing the burden of HPV-related diseases [16,23].

Primary Prevention

Primary prevention aims to reduce exposure to the virus and includes behavioral, hygienic, and immunological strategies. Sexual abstinence is the only method that completely eliminates the risk of HPV transmission; however, it is not a realistic approach for most individuals. Consistent condom use has been shown to provide partial protection against HPV infection and related lesions, including cervical intraepithelial neoplasia and genital warts, although it does not offer complete prevention due to viral transmission through uncovered skin and mucosa [16]. Good personal hygiene and avoidance of sharing contaminated items may further reduce indirect transmission.

Vertical transmission of HPV from mother to child during delivery is uncommon, occurring in approximately 5% of cases when the mother is HPV-positive. Cesarean section can slightly lower this risk, but vaccination and education of women prior to pregnancy remain the most effective preventive measures [16].

Vaccination

The introduction of HPV vaccines represents the most significant advancement in primary prevention. The first prophylactic vaccines were approved in 2006, and three main formulations are now widely available: the bivalent (HPV 16 and 18), quadrivalent (HPV 6, 11, 16, 18), and nonavalent (HPV 6, 11, 16, 18, 31, 33, 45, 52, 58) vaccines [16]. These vaccines consist of non-infectious virus-like particles (VLPs) made from the L1 capsid protein, which elicit a strong neutralizing antibody response without containing viral DNA [15].

The Centers for Disease Control and Prevention (CDC) recommends routine vaccination for boys and girls aged 11–12 years, with catch-up vaccination available up to age 26. For individuals who begin vaccination before the age of 15, two doses administered six to twelve months apart are sufficient, while those starting later or with immunosuppression should receive a three-dose series [16]. The World Health Organization (WHO) now supports one- or two-dose regimens for girls aged 9–20 years, based on studies demonstrating comparable immunogenicity and protection [24].

Long-term studies have confirmed the durable efficacy of HPV vaccination. Follow-up data from multiple large-scale trials demonstrated that the quadrivalent HPV vaccine (qHPV) provides sustained protection for at least 10–14 years, with no breakthrough infections by HPV 6, 11, 16, or 18 among vaccinated individuals [21]. Furthermore, vaccinated women and men showed significantly lower rates of cervical intraepithelial neoplasia, genital warts, and anal dysplasia compared to unvaccinated populations [21,23]. Antibody levels peak after the third dose and remain stable over time, ensuring long-lasting immunity [21].

Secondary Prevention and Public Health Perspective

In addition to vaccination, secondary prevention relies on cytological and molecular screening. Routine cervical cytology (Pap test) and HPV DNA testing are crucial for early detection of high-grade lesions and prevention of cervical cancer [12,24]. Screening is recommended even for vaccinated women, as vaccines do not cover all oncogenic HPV types.

Despite the proven efficacy of HPV vaccines, global coverage remains suboptimal—particularly in low- and middle-income countries—due to high costs, limited access, and vaccine hesitancy among parents and healthcare providers [25]. Increasing awareness, strengthening healthcare worker education, and incorporating HPV vaccination into national immunization programs are essential to improving uptake and achieving global eradication of HPV-related malignancies.

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