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Review

Human Papillomavirus in Men: An Updated Review of its Clinical and Epidemiological Relevance

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ABSTRACT

The human papillomavirus (HPV) is the most common sexually transmitted infection worldwide and also represents an important public health issue in men. This review synthesizes current evidence on epidemiology, clinical manifestations, and preventive strategies for HPV in men, highlighting key differences compared to females. Prevalence remains high throughout adult life, and the penile shaft is the anatomical site with the highest viral load. HPV is implicated in a significant proportion of penile, anal, and oropharyngeal cancers, with genotype 16 being the most prevalent. Limited seroconversion and the absence of screening protocols hinder early detection in men, especially in vulnerable populations such as men who have sex with men and immunocompromised individuals. Male vaccination has demonstrated efficacy in reducing infections and associated neoplasms, although its coverage remains uneven. The essential role of the dermatologist in the comprehensive management of HPV in men is highlighted, from education and prevention to diagnosis and treatment, as well as the need for public health policies that promote gender equity in access to vaccination.

Introduction

Anogenital infection with human papillomavirus (HPV) constitutes a field infection and is the most common sexually transmitted disease worldwide. It is estimated that between 85% and 90% of sexually active individuals will acquire HPV at some point in their lives.[1–3] In most cases, these infections are transient and asymptomatic.

HPV is a small DNA virus with tropism for cutaneous and mucosal epithelia. More than 200 genotypes have been identified, of which at least 12 are considered oncogenic.[4,5] It is estimated that more than 95% of cervical cancers are related to HPV infection, as well as 90% of anal cancers, 36–40% of penile cancers, 40–64% of vaginal cancers, 40–51% of vulvar cancers, and between 12% and 63% of oropharyngeal cancers.[6,7] According to the International Agency for Research on Cancer, in 2018 approximately 69,400 cases of cancer attributable to HPV were estimated in men.[8] Similarly, data from the Global Cancer Registries database (GLOBOCAN, Global Cancer Observatory) predict an increase of more than 56% in the incidence rate of penile cancer by 2040.[9]

On the other hand, HPV is responsible for all cases of genital warts. In Europe, between 379,000 and 510,000 new cases are reported annually in women, and between 377,000 and 428,000 in men. Although these lesions are benign, they affect quality of life and their treatment entails a significant cost for health care systems.[10]

Historically, HPV research has focused on women and its association with cervical cancer.[11] However, in recent years, interest in understanding infection in men has increased, revealing relevant differences between sexes regarding the natural history of the virus and the anatomical sites affected.[12] Although this review focuses on the term “men” in a biological sense, some of the clinical signs and risks associated with HPV may be shared by individuals with a penis of diverse genders, such as transgender women or non-binary individuals. In the absence of sufficiently disaggregated studies by gender identity, the data presented here should be interpreted considering this possible population diversity. In addition, genotypes 16, 18, 31, 33, 45, 52, and 58 have been estimated to account for 89% of cancers attributable to HPV in both sexes.[13]

From a dermatological perspective, HPV is a frequent cause of consultation due to cutaneous and mucocutaneous lesions, particularly condyloma acuminata, which represent one of the main visible clinical manifestations of infection in men. These lesions, in addition to their clinical, functional, and psychosocial impact, may coexist with infections by high-risk genotypes, highlighting the role of the dermatologist in identifying patients at oncological risk. Dermatology plays an essential role not only in the topical or ablative treatment of these lesions but also in health education, vaccination indication, and early referral of cases suspicious for genital or anal malignancy. This multidisciplinary

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approach is key to optimizing prevention and clinical management of HPV in the male population.

This review aims to further explore these differences and evaluate effective prevention strategies in men to reduce the global burden of HPV-associated cancers.

Materials and methods

Study design

We conducted a structured clinical review to evaluate the available evidence on human papillomavirus (HPV) infection in men, including both heterosexual men and men who have sex with men (MSM). The review focused on four thematic axes: (1) sex-based differences in HPV transmission and clearance, (2) anatomical location and diagnostic methods, (3) associated diseases, both benign and neoplastic, and (4) preventive strategies, with emphasis on universal vaccination.

Sources and search strategy

We consulted the databases PubMed/MEDLINE, EMBASE, Scopus, and the Cochrane Library. As additional sources, updated clinical practice guidelines from international organizations such as the Centers for Disease Control and Prevention (CDC), the World Health Organization (WHO), and the European Centre for Disease Prevention and Control (ECDC) were reviewed. The search period included publications between 2005 and 2024 in both English and Spanish languages.

Study selection

A total of 453 health records were initially identified after searching the selected databases. The records were distributed into the following thematic categories: transmission and clearance ($n = 117$), infection location ($n = 84$), HPV-related cancer ($n = 198$), and preventive measures ($n = 54$).

Screening was performed in 2 phases:

- In phase #1, a total of 127 duplicate articles were removed.
- In phase #2, 2 investigators independently assessed titles and abstracts, excluding 211 records that did not meet the inclusion criteria.

Potentially eligible articles underwent full-text review. After this phase, 58 studies were included in the final review. Discrepancies between reviewers were resolved by consensus.

Inclusion and exclusion criteria

Studies conducted in humans were included, with results disaggregated by male sex and a sample size ≥ 50 participants. Clinical trials and observational studies (cohort, case–control, and cross-sectional studies) were accepted.

In vitro or animal studies, opinion articles, letters to the editor, and case series with fewer than 20 patients were excluded. Studies without full-text availability were also excluded.

Data extraction and analysis

Data extraction was performed independently by 2 investigators.

Key variables collected included: characteristics of the study population, diagnostic methodology used, anatomical location of HPV, clinical outcomes (infection rates, persistence, and complications), and effectiveness of the preventive strategies evaluated.

All discrepancies were resolved through discussion until consensus was reached.

Methodological quality assessment

Included studies were classified into 3 levels of quality:

- High, if they presented a robust design and adequate statistical analysis.
- Moderate, in case of minor methodological limitations.
- Low, if they presented significant biases or serious deficiencies; these were excluded from the final synthesis.

Results

Differences in HPV transmissibility and clearance according to sex

Well-documented differences exist. Although HPV transmission occurs in both sexes, some aspects of the natural history of infection differ significantly between men and women. For example, the prevalence of genital HPV is higher in heterosexual men than in women,[14] and while the incidence rate of infection decreases with age in women, in men it remains relatively stable throughout life.[15] This difference may be related to a more effective cell-mediated immune response in women,[16] which would provide some protection against new exposures.

However, literature on transmission rates stratified by sex is limited. Six relevant studies were identified,[17–22] allowing analysis of 877 heterosexual couples, showing a higher transmission rate from women to men (F–M) compared with men to women (M–F). In the HITCH cohort,[23,24] follow-up visits were conducted with a median of 5.5 months (range, 1.8–15.5), and >4 vaginal sexual encounters per week were documented. This study showed a modest difference between M–F transmission (3.5 per 100 person-months; 95%CI, 2.7–4.5) and F–M transmission (4.0 per 100 person-months; 95%CI, 3.0–5.5).

These findings contrast with those of the Hernández study,[18] in which the F–M transmission rate was 17.4 per 100 person-months (95%CI, 10.6–25.8), identifying the glans as the main transmission site in men, while the cervix and urine were the main sources of infection for women. In addition, the female anus also acted as a source of transmission, demonstrated by genotypic concordance between cervical and anal infections in women, suggesting that the same genotype may affect multiple anatomical sites and that some viral types may be selectively transmitted. In fact, oncogenic viruses were observed to be more frequently transmitted to men through urine than through the cervix.

These results are consistent with those of the prospective HIM study,[25] which showed higher incidence rates of oncogenic types such as HPV 16, 52, 51, and 39 in men, and HPV 51 and 16 in women. Furthermore, a higher incidence rate of infection was observed in men with stable partners, with F–M transmission being more common than M–F. Although it could be assumed that some men acquired the virus outside the relationship, the HIM study stratified analyses according to monogamy, confirming that even in monogamous relationships men had higher viral acquisition.

The study by Widdice[20] confirmed these findings, showing similar transmission patterns over short observation periods. Unlike other viral sexually transmitted infections (which are usually systemic), HPV infection primarily affects mucosal surfaces. Men tend to acquire more transient infections than women, probably due to a lower capacity to generate immune memory, making them more susceptible to reinfections.

On the other hand, in the cohort of Su,[22] a similar transmission rate between men and women was documented, probably related to more conservative sexual behaviors. In this study, the vagina, vulva, and perineal region behaved as viral reservoirs, consistent with what was observed by Hernández.

In addition to differences in transmission, differences in the rate of acquisition of new infections have also been identified. While in women the incidence of infection decreases with age, in men it remains constant.[15] This highlights men as a key target in prevention strategies, since

their risk of infection persists throughout life. One possible explanation is that the keratinized epithelium of the penis induces a weaker immune response than the mucosa of the cervix or anus. In fact, HPV seroprevalence is significantly lower in men than in women,[12] indicating that men may require a greater number of exposures to achieve effective seroconversion (Table 1).

Regarding viral clearance, data from the HIM study[25] show that younger men (18–30 years) have longer viral clearance times compared with older men. At 12 and 24 months, 66% and 90% of men, respectively, had cleared recently acquired infections. The only genotype whose clearance is not influenced by age is HPV-16, which is also observed in women (mean, 12 months), while other oncogenic types are cleared in approximately 6 months.

Data from the HIM study are comparable to those from the placebo group of the quadrivalent vaccine trial conducted by Duarte Moreira,[26] which reported mean clearance times of 6.1, 6.1, 7.7, and 6.3 months for HPV types 6, 11, 16, and 18, respectively. These discrepancies, especially for HPV-16, could be explained by population differences, such as the inclusion of men who have sex with men. Similarly, in young women (16–23 years), clearance times are 6.2, 6.5, 11.7, and 12.4 months, respectively,[15] suggesting that physiological differences between male and female epithelia also influence viral clearance kinetics.

Anatomical variations in localization and sampling

There are relevant differences in sample collection and anatomical localization of HPV infection between men and women.

In females, cervical sampling for viral DNA detection is well standardized, including established diagnostic and therapeutic pathways according to results. In contrast, in men there is no established clinical protocol nor any FDA-approved screening test for routine use.[23]

Scientific literature describes multiple anogenital sites susceptible to infection in men, which is explained by the “field infection” nature of HPV, with potential involvement of multiple epithelial surfaces. This anatomical variability complicates both sample collection and interpretation of results.

Nicolau et al. analyzed a total of 55 men with stable HPV-positive partners, using aqueous capture in six different anogenital areas and peniscopy with biopsy of suspicious lesions.[23,27] A total of 70% of men were HPV-positive: 32% for high-risk genotypes, 14% for low-risk, and 24% for both groups. The most frequently biopsied site was the inner foreskin (57 of 104 samples). Overall, HPV DNA was detected in 27 of 104 biopsies (26%), although only 14 samples (13.5%) showed histological evidence of infection.

A notable finding was the absence of genotypic correlation between partners, since in 50% of cases in which the woman had high-risk HPV infection, her male partner did not test positive for the same genotypic group. This phenomenon reinforces the complexity of viral transmission and immunological dissociation between sexes.

These data are consistent with those obtained by Anna Giuliano et al. in the HIM study,[25] whose objective was to identify optimal sites for HPV detection in heterosexual men. In a cohort of 463 men, HPV DNA presence was evaluated in seven different anatomical sites. In the overall cohort, 65.4% of men were positive in at least one location, a percentage similar to that observed in the subcohort of 186 men with complete anatomical analysis (67.7%).[28]

Detection was more frequent in the penile shaft (49.9% and 47.9%), followed by the glans and coronal sulcus (35.8% and 32.8%), and the scrotum (34.2% and 32.8%). Other areas such as the perianal region (20.0% and 22.6%), the anal canal (17.6% and 18.3%), the urethra (10.1% and 10.2%), and semen (5.3% and 4.8%) showed lower detection rates. These results support the need to use multiple sampling sites, considering the penile shaft as a priority region for HPV detection in men.

HPV-related cancers in men

A strong association has been established between HPV and the development of cancers in men, especially penile carcinoma, oropharyngeal carcinoma, and anal carcinoma. The relationship with other tumors such as bladder, urethral, or prostate cancer remains under investigation.

The association between HPV and tumor development was established during the 1970s and 1980s, with the pioneering discoveries of the German virologist Harald zur Hausen, who received the Nobel Prize in Medicine in 2008 for his work on HPV types 16 and 18.[29] Globally, HPV is responsible for 4.5% of all cancers in women and men, and in Europe approximately 2.5%, so it is important to highlight that the risk of progression to cancer is associated with the persistence of HPV infection.[8]

HPV infects epithelial cells, and its replication cycle is linked to epithelial differentiation. There are more than 200 genotypes with variable tissue specificity. While most cause benign lesions (such as genital warts or condyloma acuminata, mainly associated with HPV-6 and -11), a subgroup of high-risk HPV (HR-HPV) has oncogenic potential. It should be noted that some condylomas may contain coinfections with HR-HPV genotypes (e.g., HPV-16, -18, -31), which underscores the importance of viral typing in persistent or atypical lesions.[30]

The E6 and E7 oncoproteins block key cellular functions such as apoptosis and DNA repair, promoting malignant transformation.[31]

Penile cancer

HPV is estimated to be present in 38–40% of cases of penile squamous cell carcinoma,[32,33] and its incidence rate is expected to increase by 56% by 2040 according to data from the Global Cancer Observatory (GLOBOCAN).[9] HPV-16 is the most widely implicated genotype, responsible for up to 72% of cases.

Mechanistically, HPV contributes to penile carcinogenesis through integration of its viral DNA into the cellular genome, with overexpression of E6 and E7. The p16 protein, whose increase reflects inactivation of Rb by E7, has become a useful prognostic marker.[34] A recent meta-analysis demonstrated significantly higher disease-specific survival in HPV-positive men (HR, 0.61) and p16-positive men (HR, 0.45), supporting their clinical value.[35]

Despite this evidence, there is no established method for early screening of penile cancer, nor is routine HPV detection recommended at this site.[36] Treatment is based on conservative surgery when possible, radical lymphadenectomy if there is nodal involvement, and adjuvant chemotherapy in advanced stages.[37–39]

Oropharyngeal cancer

The association between oropharyngeal squamous cell carcinoma (OPSCC) and HPV, mainly HPV-16, has been confirmed over the last 2 decades; in some countries, these HPV-dependent tumors are already more frequent than HPV-independent ones, traditionally associated with tobacco and alcohol.[40] The incidence rate of HPV-positive OPSCC has increased in young men in high-income countries.[41] This subtype has a better prognosis and greater sensitivity to radiotherapy, which has been reflected in the eighth edition of the TNM system of the American Joint Committee on Cancer (AJCC).[42]

The diagnosis of HPV infection is based on immunohistochemistry for p16 combined with in situ hybridization for the detection of oncogenic genotypes (HPV-16 and HPV-18). Although there is no population-based screening method for head and neck cancers, treatment is based on chemoradiotherapy, generally with cisplatin and radiotherapy at 66–70 Gy.[43]

Anal cancer

HPV is responsible for >90% of anal carcinoma cases, with HPV-16 being the main implicated genotype.[44] The precursor lesion is high-grade squamous intraepithelial lesion (HSIL), equivalent to anal intraepithelial neoplasm (AIN). This progression is more common in high-risk populations, such as MSM and people living with HIV.

In 2022, the ANCHOR (Anal Cancer HSIL Outcomes Research) study was published in *The New England Journal of Medicine*, evaluating whether treatment of HSIL lesions could prevent anal cancer in people with HIV.[45] In a cohort of >4400 HIV-positive patients, treatment reduced the risk of progression to cancer by 57% vs observation. This is the first solid evidence that treating anal precancerous lesions can prevent cancer in high-risk patients. This finding supports the systematic use of anal cytology, high-resolution anoscopy (HRA), and targeted treatment of HSIL, replicating the cervical cancer prevention model.

In MSM with HIV, the prevalence of anal HPV approaches 100%,[46,47] and anal cancer rates are up to 80 times higher than in the general population[48] Therefore, the use of anal cytology and HRA is recommended in at-risk populations.

Currently, consensus guidelines published by the International Anal Neoplasia Society in 2024 include other high-risk groups in whom anal cancer screening is also indicated—not only MSM with HIV, but also women with a past medical history of high-grade genital intraepithelial neoplasm, immunocompromised individuals, and other patients with HIV.[49]

Although HPV DNA is not useful as a diagnostic test due to its high prevalence in this population, the combination of cytology and targeted examination has demonstrated high predictive value.

Treatment of anal cancer includes combined chemoradiotherapy, and in early cases, topical or ablative options. However, close follow-up is essential due to the risk of progression and recurrence.

Other tumors: urethra, bladder, and prostate

Some studies have identified HPV-16 infection in primary urethral carcinomas, although the evidence is still limited and considered to be in the investigational phase.[50,51]

Regarding the bladder, a recent meta-analysis including 14 studies showed a significant association between HPV and bladder cancer, with an OR of 4.18 (95%CI, 2.63–6.66), although the evidence is not conclusive due to methodological heterogeneity.[52]

The relationship between HPV and prostate cancer remains controversial. A meta-analysis including 27 studies ($n = 1607$ tumors; $n = 1515$ controls) found HPV in 25.8% of tumors vs 17.4% in benign prostatic hyperplasia and 9.2% in normal tissue. Although data suggest a possible association, more well-designed studies are required to establish causality.[53]

Impact of vaccination in men

Vaccination against human papillomavirus in men has proven to be an effective tool for preventing genital infections, warts, and several types of cancer, in addition to reducing community transmission. However, its implementation and coverage worldwide show notable inequalities.

In North America and Western Europe, countries such as the United States, Canada, the United Kingdom, Germany, and Australia have incorporated male vaccination into their national programs, recommending it from 9–13 years of age. In these contexts, significant reductions in the incidence of HPV infection and genital warts have been observed.[1,54]

In Latin America, nations such as Argentina, Brazil, Mexico, and Colombia have also introduced male vaccination schemes, although with variable coverage. Argentina, for example, has reported rates > 70% in some regions.[3]

In Asia and Africa, male vaccination is still limited and mainly focused on women. Japan and some Southeast Asian countries have begun to introduce vaccination in men through specific access strategies.[7]

In low-resource countries, economic, logistical, and awareness barriers continue to hinder implementation, with priority given to the prevention of cervical cancer in women.

In Spain, HPV vaccination is included in the common vaccination schedule throughout life, approved by the Public Health Commission of the Interterritorial Council of the Spanish National Health System. Current funded indications, according to official recommendations from the Spanish Ministry of Health, are:

Systematic vaccination of girls and boys at 12 years of age, with a single-dose schedule.[55]

Catch-up vaccination up to 18 years of age for unvaccinated adolescents, also with a single dose.[55,56]

Unvaccinated individuals with certain risk conditions up to 45 years of age, in whom vaccination is recommended if they belong to one of the following groups:

- MSM.
- Individuals engaged in prostitution.
- Immunocompromised individuals: HIV infection, solid organ transplant or hematopoietic stem cell transplant, WHIM syndrome.[55]

In these cases, the schedule varies according to age:

- From 9 to 25 years: one dose, except in immunocompromised individuals.
- From 26 to 45 years: two doses at least 6 months apart.
- In immunocompromised individuals: 3 doses with a 0, 2, and 6-month schedule, regardless of age.[57]
- For individuals > 45 years, evidence is limited; vaccination may be considered on an individual basis according to risk factors and clinical judgment.

In addition to national recommendations, autonomous communities have implemented their own strategies with differences in coverage, target groups, and catch-up age.[58] For example:

- Systematic vaccination at 12 years (common in Andalusia, Castile and León, Castile-La Mancha), with active catch-up up to 18 years in unvaccinated populations.
- Extension to adolescent males:
  - Catalonia (up to 18 years, cohorts 2007–2011, from 2025).
  - Madrid (13–18 years, from 2024).
- Extended coverage to young adults:
  - Galicia: men aged 18–21 years (cohorts 2004–2005), with vaccination starting on May 26, 2025.[59]
  - Murcia: men up to 25 years (cohorts 1999–2000 in phase #1), with progressive implementation from 2024 starting with older cohorts, with the aim of reaching all individuals born between 1999 and 2010 by 2027.[60]

These territorial differences affect catch-up age, type of vaccine administered, and populations included in funded programs, generating inequalities in access to prevention and highlighting the need to harmonize funding criteria across the national territory.

Male vaccination not only prevents infection in men but also significantly contributes to herd immunity, reduces disease burden, and supports HPV eradication programs. Its inclusion in systematic vaccination schedules should be a priority in global public health policies.

Table 2
Clinical recommendations for the management of HPV in the male population, stratified by clinical scenario. Actions include: (1) Preventive education (transmission, protective measures, and screening according to risk), (2) Therapeutic approach (according to the presence of warts, precancerous lesions, or cancer), and (3) Vaccination up to 45 years—recommended even post-exposure without lesions—for protection against non-acquired genotypes.

Clinical situation	Recommended action
Asymptomatic patient without history of STIs	• Preventive education: Transmission (skin-to-skin contact, even non-sexual or sexual), condom use, reduction in number of sexual partners. • Vaccination: Recommended up to 45 years if not previously vaccinated. • Screening: Routine HPV screening is not recommended.
Patient with a past medical history of partner with HPV	• Preventive education: Information on transmissibility and recommendation of genital self-examination. • Vaccination: Recommended, even after prior exposure (protection against non-acquired genotypes). • Clinical surveillance: Only if symptoms appear.
Presence of genital warts	• Treatment: Imiquimod, podofilox, or cryotherapy. Re-evaluation in 3–6 months if lesions persist. • Preventive education: Avoid sexual contact until complete resolution. • Vaccination: Recommended to prevent recurrence or new infections.
MSM or HIV +	• Screening: Anal cytology and high-resolution anoscopy in high-risk individuals*. • Follow-up: Annual evaluation. • Vaccination: Priority, even if prior exposure occurred. *High risk: history of condylomas, anal dysplasia, or advanced immunosuppression.
Patient with anal lesions or persistent symptoms	• Evaluation: Proctologic assessment, with biopsy if suspicious lesions are present. • Vaccination: Recommended (protection against other genotypes).
History of multiple sexual partners	• Preventive education: STI screening, reinforcement of condom use, and risk reduction. • Vaccination: Recommended up to 45 years; individualize in older patients.
Men with infertility without apparent cause	• Evaluation: HPV detection in semen only in specific infertility studies. • Vaccination: Consider in cases with persistent infection.
Prior HPV exposure without visible lesions	• Preventive education: Self-examination and clinical follow-up if symptoms appear. • Vaccination: Recommended, as it protects against other non-acquired genotypes. • Additional tests: Not indicated if no lesions are present.
Condom use	May reduce the risk of HPV transmission by 60–70%, although it does not completely eliminate the risk. Its effectiveness depends on correct and consistent use. Additionally, it promotes regression and viral clearance in already infected individuals, with demonstrated clinical benefit at least during the first 3 months.

MSM: Men who have sex with men; high risk: past medical history of condylomas, anal dysplasia, or advanced immunosuppression.

Proposed algorithm for HPV management in men

HPV infection in men includes a broad clinical spectrum ranging from asymptomatic carriers to benign signs (warts) or malignant conditions (penile, anal, or oropharyngeal cancers). A practical clinical management algorithm based on individual risk and clinical context is proposed below (Table 2).

Conclusions

HPV infection in men represents a clinical and public health challenge, with direct implications in dermatology, urology, oncology, and infectious diseases. Although research and prevention programs have historically focused on women, there is clear evidence that HPV causes a substantial burden of disease in men, including benign lesions such as condyloma acuminata and malignant anogenital and oropharyngeal neoplasms.

Dermatologists play a key role in the recognition, diagnosis, and treatment of cutaneomucosal signs of HPV, as well as in education, prevention, and timely referral of at-risk patients. Prophylactic vaccination in men, identification of precancerous lesions, and multidisciplinary management of advanced cases are essential pillars of the current strategy.

The approach to HPV in men must be comprehensive, evidence-based, and adapted to the individual risk profile, with special attention to MSM, immunocompromised patients, and individuals with a history of HPV-related lesions. Promoting equity in prevention and expanding access to male vaccination are fundamental steps toward the global control of this infection.

Conflict of interest

The authors declare that they have no conflict of interest.

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