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HPV AND ANAL CANCER: GLOBAL PATTERNS, KEY RISK FACTORS, AND ADVANCES IN PREVENTION

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ABSTRACT

Introduction and Purpose: This article comprehensively analyzes human papillomavirus (HPV) in anal cancer, covering its epidemiology, etiology, risk factors, and role in anal squamous cell carcinoma.
Methods: A literature search of PubMed and Google Scholar (2020–2025) included observational studies, clinical trials, meta-analyses, and systematic reviews.
State of Knowledge: HPV is the main cause of anal squamous cell carcinoma (ASCC), whose incidence is rising in high-income countries and is highest in men who have sex with men (MSM), people with HIV, and those with multiple partners, immunosuppression, receptive anal intercourse, smoking, or co-infections. Over 80% of ASCC cases are linked to HPV16, with HPV-positive cancers showing better survival, yet despite the vaccine being nearly 100% effective if given before sexual debut, global uptake remains low.
Conclusion: Persistent infection with high-risk HPV, especially HPV16, is the main driver of anal carcinogenesis, paralleling cervical disease, and HPV-driven anal cancer is an increasing public health concern. To reduce the incidence and improve outcomes, expanded vaccination, targeted screening, and public awareness are essential. Future efforts should focus on enhancing prevention uptake, clarifying the significance of HPV16 sublineages, and evaluating co-infections and immunity to improve prevention and management.

KEYWORDS

HPV, Anal Cancer, HPV‐Related Cancer, Human Papillomavirus, Anal HPV Infection

CITATION

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Introduction.

Human papillomavirus (HPV) has been identified as the most prevalent sexually transmitted infection on a global scale. While the majority of such infections are known to resolve naturally within a period of two years, those that persist can lead to various conditions, ranging from benign genital warts to malignant cancers. [1]

Human papillomavirus (HPV) is a double-stranded DNA virus that has been linked to cancers arising from squamous epithelial tissues, including those of the cervix, oropharynx, and anogenital regions. Approximately 200 different types of HPV have been identified, many of which have the potential to lead to hyperplastic lesions in mucosal or skin epithelia. These types are generally categorised into low-risk (LR-HPV) and high-risk (HR-HPV) groups based on their potential to cause cancer. Individuals with compromised immune systems are particularly susceptible to developing HPV-related lesions and malignancies, thus rendering HPV a valuable model for studying the virus-host dynamics involved in epithelial tumour formation. Cervical cancer is the most extensively studied HPV-related malignancy, with high-risk HPV types implicated in 99% of cases. However, HPV has also been identified as a significant causative agent in cancers affecting other mucosal sites, with detection rates ranging from 64 to 91% in vaginal cancers, 40 to 50% in vulvar cancers, 88 to 94% in anal cancers, and 40 to 50% in penile cancers. [2] HPV infections are frequently resolved by the immune system, with symptoms often abating within one to two years. However, persistent infections with high-risk HPV types have been shown to induce cellular changes that, if left unaddressed, may progress to precancerous lesions and ultimately cancer (Grant et al., 2019). Human papillomavirus (HPV) has been associated with six distinct types of cancer: cervical, vulvar, penile, vaginal, oropharyngeal, and anal cancers. It is estimated that chronic high-risk HPV infections account for approximately 90% of cervical and anal cancers, 70% of vulvar and vaginal cancers, 60% of penile cancers, and 70% of oropharyngeal cancers. [21]

Methods

A structured literature review was conducted using databases such as PubMed and Google Scholar. The search strategy involved the use of targeted keywords, including:'HPV', 'anal cancer', 'HPV-related cancer', 'human papillomavirus', 'anal HPV infection'. Studies published between and 2020 and 2025 were prioritized, as well as frequently cited publications from over a decade ago. The inclusion criteria were as follows: observational studies, clinical trials, meta-analyses and systematic reviews papers with a major focus on the epidemiology, etiology and risk factors in anal squamous cell carcinoma. Only articles published in English were included in the final analysis.

High-Risk HPV and the Rising Burden of Anal Cancer

Anal cancer is among six distinct cancerous conditions that have been identified as having a causal link with human papillomavirus (HPV), with approximately 90% of cases being attributed to HPV infection. Analogous to oropharyngeal cancer, a single HPV type tends to predominate in driving tumour development. HPV16 was identified in 80.7% of anal cancer cases. In accordance with the trends observed in other HPV-related malignancies, the global incidence of anal cancer is increasing, including in regions such as the United States and Europe. [3]

Individuals living with HIV are disproportionately impacted by HPV infection, experiencing reduced viral clearance, a higher risk of cancer development, and elevated mortality rates. [1]

With regard to the distribution of HPV genotypes in anal infections, HPV6 and HPV16 were the two most frequently detected overall and across the study groups. HPV16, in particular, has been identified as a primary contributing factor to both the global and local anal cancer burden. Furthermore, HPV16 has been found to be more prevalent in high-grade lesions when compared to cytology-negative or LSIL anal samples. The prevalence of HPV16 varies among men according to their HIV status and sexual orientation, ranging from 2–3% in HIV-negative men who have sex with women to around 30% in HIV-positive men who have sex with men. In HIV-negative women, concurrent cervical HPV16 infection is a significant risk factor for the

detection of HPV16 in the anus. A substantial pooled meta-analysis revealed a prevalence of 41% in women with cervical HPV16, in contrast to a mere 2% in those without. The prevalence of HPV6 is less frequently reported due to its exclusion from most commercial high-risk HPV DNA tests. However, HPV6 was also the most commonly identified genotype in our previous studies of anal infections among women, as well as in both HIV-negative and HIV-positive men.

[4][5] It is estimated that persistent infection with high-risk HPV types is responsible for approximately 90% of cervical and anal cancers, 70% of vulvar and vaginal cancers, 60% of penile cancers, and 70% of cancers in the oropharynx.[1] Anal cancer is one of six cancers associated with human papillomavirus (HPV). In approximately 90% of anal cancer cases, the virus is identified as the cause. As with oropharyngeal cancer, a single HPV type tends to predominate in driving disease progression. A meta-analysis by De Sanjosè et al. (2019) found that HPV16 was detected in 80.7% of anal cancer cases. [3] Human papillomavirus (HPV) infection is recognised as the primary risk factor for anal cancer. The virus encompasses a group of over 150 related types, several of which are known to cause cervical and other cancers. The International Agency for Research on Cancer has classified HPV as a carcinogen for a number of cancer types, including those affecting the cervix and anus. The presence of a history of cervical cancer or precancerous lesions in women is associated with an increased risk of developing anal cancer. In the United States, there has been an observed increase in the incidence of anal cancer among both men and women. There is a robust body of evidence indicating a link between high-risk HPV (human papillomavirus) strains, particularly HPV-16 and HPV-18, and the development of anal cancer. Globally, approximately 27, 000 new cases are reported each year, with females being affected more frequently than males, at a ratio of up to 5:1. These trends emphasise the pressing need for enhanced awareness and screening programmes to identify early indications of anal cancer. [6]

Contributing factors and Their Role in Anal Carcinogenesis

Oncogenic types of HPV infect mucosal or mucocutaneous epithelia, and certain anatomical areas — such as the cervix, anus, and tonsillar crypts in the oropharynx — are particularly susceptible to HPV-induced cancers. This susceptibility is attributable to two factors. Firstly, the anatomical features of the subjects in question facilitate the initial phase of infection. Secondly, the subjects' role in tumour development must be considered. The cervix and anus are distinguished by a squamo-columnar junction, otherwise known as a transformation zone (TZ), which is the point at which stratified and columnar epithelium meet. While HPV generally requires access to the basal layer of stratified epithelium through microscopic gaps to establish a persistent infection, the architecture of the squamo-columnar junction facilitates access to the basal cells of the cervix.

The anal transformation zone is the primary site of most HPV-related anal cancers. HPV-related malignancies are generally understood to develop from precancerous lesions. Anal lesions are classified as AIN, PIN, VAIN, and VIN, depending on their anatomical location. [7][8][19]

Mapping Genetic Variants and Cancer Risk

HPV16 variants have been identified through genomic sequencing and are categorised into four major phylogenetic lineages (A–D), which are further divided into 16 sublineages: A1–A4, B1–B4, C1–C4, and D1–D4. Whole-genome analysis has revealed distinct mutations that define these lineages. Research on cervical cancer has indicated that specific sublineages – such as D2, D3, A3, and A4 – are linked to an elevated risk of the disease, with A4, D2, and D3 particularly associated with the development of adenocarcinomas. Furthermore, lineage C and sublineages D2 and D3 have been associated with an elevated probability of developing premalignant cervical lesions.

A large international study conducted across Europe, Asia, and Central/South America found that HPV16 sublineages A1–A3 were the most commonly detected in cervical, vaginal, and penile cancers, regardless of geographic region. Conversely, the A4 sublineage was predominantly associated with anal cancer in Asian populations. Recent findings from the HPV in Men (HIM) study reported a high prevalence of the A1 sublineage in anal swabs from men diagnosed with anal cancer in Brazil, Mexico, and the United States, with no significant differences observed between variant lineages in terms of HPV16 persistence. [9]

Anal cancer, associated with HPV, has a development pattern analogous to that of cervical cancer, with the virus often targeting the anal transformation zone, a factor that can result in malignant changes. Conversely, the process of HPV-driven cancer formation in the oropharynx appears to follow a distinct biological pathway. [1]

Key Risk Factors

A number of risk factors have been identified as contributing to an increased incidence of anal cancer, including advanced age (particularly over 50 years), younger age at sexual debut, a greater number of sexual partners, a history of receptive anal intercourse, smoking, and compromised immune function. Furthermore, women with current or past high-grade cervical (CIN2/3+) or vulvar lesions are also at elevated risk. Furthermore, the incidence of anal cancer is found to be considerably elevated among individuals living with HIV (PLWH). [3]

It is noteworthy that individuals living with HIV are more susceptible to HPV infection and anal cancer. The presence of HIV-related immunosuppression has been demonstrated to increase the likelihood of anal squamous cell carcinoma (SCC), likely by accelerating the progression from anal intraepithelial neoplasia (AIN) to invasive cancer. [10]

A population-based study conducted in Granada has revealed a consistent upward trend in the incidence of anal cancer, particularly among the male demographic, over recent decades. Factors such as certain sexual behaviours, a higher number of sexual partners, frequent anal intercourse with a receptive partner, and HIV infection increase the risk of infection with high-risk HPV strains. It is anticipated that global incidence rates of anal cancer will continue to rise, particularly among high-risk demographics. These include HIV-infected men who have sex with men, with an annual incidence rate exceeding 131 per 100, 000, as well as women with a history of HPV-related cancers.

[9] Research has indicated that certain co-infections with pathogens may play a role in HPV-driven carcinogenesis, even in the absence of their ongoing presence – a concept known as the”hit-and-run" theory. As previously discussed, the co-infection of multiple oncoviruses could act synergistically in the development of cancer, potentially increasing the risk of HPV-positive individuals who also harbour other oncoviruses developing both HPV-related and unrelated cancers. It is important to note that co-infection does not necessitate the infection of the same cells by both viruses. Instead, virus-infected cells have been shown to influence surrounding cells and their microenvironment by releasing cytokines, chemokines, various signalling molecules, exosome, or microvesicles. [12] Smoking has been identified as a well-established risk factor for anal cancer in both sexes; however, its impact appears to be more pronounced in women over the age of 60. This association may be attributable not only to the carcinogens present in tobacco smoke, but also to smoking's role in the suppression of the clearance of anal HPV infections. Research studies have estimated that men who do not use condoms with casual partners face approximately double the risk of HPV infection compared to those who do. Early sexual debut, a history of multiple male partners, and specific sexual practices have been demonstrated to delay clearance of HPV. [10]

ASIL

Anal squamous intraepithelial lesions (ASIL) are considered to be precursors to anal cancer, in a manner analogous to the role of cervical squamous intraepithelial lesions (CSIL) in cervical cancer. The range of ASILs extends from low-grade squamous intraepithelial lesions (LSIL) to high-grade squamous intraepithelial lesions (HSIL). It is estimated that approximately 90% of cases of LSIL are associated with low-risk HPV types 6 and 11, which often manifest clinically as genital warts (condyloma). Conversely, high-risk HPV types 16 and 18 have been observed to be more frequently associated with HSIL. Nevertheless, persistent anal infections with either low-risk or high-risk HPV types, along with the corresponding ASILs, have been linked to a heightened risk of developing invasive anal cancer. [13]

Squamous cell carcinoma of the rectum and HPV

Anal cancer is a relatively uncommon malignancy that develops within the anal canal. It is primarily classified into two histological types: adenocarcinoma and squamous cell carcinoma (ASCC). ASCC is frequently observed to have its origins in the vicinity of the squamocolumnar junction, and there is a robust correlation between the presence of this condition and infection with human papillomavirus (HPV), particularly the high-risk HPV16 genotype. The majority of ASCC cases are found to be positive for HPV, with HPV16 being the most prevalent type, though a subset of cases involve other HPV types outside HPV16 and HPV18. [14]

It has been suggested that HPV may play a role in certain types of cancer, including squamous cell carcinomas that can occur in various parts of the body, such as the skin, oral cavity, vagina, penis, esophagus, and anal canal. In 2003, Kinjo et al. were the first to investigate the effects of introducing HPV into adenocarcinoma cells from the colon and lungs, demonstrating a clear association between HPV transfection and squamous metaplasia in colonic adenocarcinoma cells. Given the well-documented relationship between

HPV and squamous cell cancers, immunohistochemical staining for the high-risk HPV marker p16 was performed, which returned positive, suggesting a strong potential role of high-risk HPV in the development of rectal squamous cell carcinoma. [15] Evidence suggests that, in women, anal HPV infection may often originate from the cervix or the external genital area rather than through anal intercourse. This assertion is further substantiated by studies demonstrating that the number of sexual partners is more strongly associated with anal HPV than a history of anal sex. Moreover, findings indicate that the majority of women diagnosed with anal cancer report no history of anal intercourse. Research also links front-to-back wiping with anal HPV detection. In men, the presence of SCCA unrelated to HIV is hypothesised to be partly driven by HPV transmission through anal intercourse. This is due to the fact that men who have sex with men (MSM) without HIV are at higher risk than men who have sex with women (MSW) without HIV. Nevertheless, anal HPV has also been detected in HIV-negative MSW with no history of anal intercourse, indicating the existence of alternative transmission routes. It is important to note that HIV-negative MSW constitute a significant proportion of the male demographic, thus maintaining their representation in anal cancer cases among the male population.[16] [18]

Anal Cancer: HPV Profiles and Survival

In a study of 185 anal cancer specimens, HPV genotyping revealed that 164 cases (88.6%) tested positive for at least one HPV type. Furthermore, high-risk HPV types were identified in 87.03% of these samples. HPV16 monoinfection was identified in 145 cases (78.4%), while seven cases (3.8%) exhibited co-infection of HPV16 with other high-risk types. Two samples (1.1%) were found to be co-infected with HPV16 and a low-risk HPV type. HPV18 was the next most prevalent type, detected in three samples (1.6%). HPV types 33 and 68 were identified in two samples (1.1%), while types 35, 51, and 52 were each present in one sample (0.5%), and HPV39 was detected in three samples (1.6%). The presence of low-risk HPV types, devoid of high-risk types, was identified in three samples (1.6%). In the course of the study, 185 patients were analysed; 61 of these (33.0%) died during the follow-up period. Kaplan–Meier survival analysis demonstrated that patients positive for HPV and specifically HPV16 had significantly better survival outcomes (log-rank test $p = 0.0077$ and 0.006, respectively). [3]

Prevention- vaccines

The introduction of HPV vaccines has led to a substantial decline in HPV-associated diseases. When administered before the onset of sexual activity, the vaccine is almost 100% effective in preventing cervical cancer. However, global vaccination rates remain low, largely due to limited access and insufficient public awareness about HPV. Expanding education and improving vaccine availability are essential steps toward reducing HPV-related cancers and associated morbidity and mortality worldwide.[1]

The efficacy of HPV vaccines has been well-documented. The impact of the vaccine is twofold: it benefits vaccinated women directly, and it contributes to a reduction in the prevalence of genital warts among unvaccinated men through the principle of herd immunity. Research indicates that a three-dose course of the Gardasil-9 vaccine may diminish the infectivity of bodily fluids in women infected with HPV, thereby reducing the risk of transmission to sexual partners. A comparison of available HPV vaccines indicates that seven years after vaccination, the bivalent vaccine generates higher antibody levels than the quadrivalent vaccine, while the nonavalent vaccine shows the highest overall efficacy. The immunological consequences of these vaccines are twofold, engendering both innate and acquired immunity by means of altering responses to Toll-like receptor (TLR) ligands. Cervarix has been associated with stronger cytokine production, while Gardasil – despite lower cytokine expression – has been shown to enhance acquired immunity and increase TNF-α production after subsequent stimulation. [17]

The consistent utilisation of condoms during sexual activity has been demonstrated to offer protection against HPV infection. [9]

A cross-sectional study conducted in Italy examined the factors influencing HPV vaccination uptake among eligible adults. The primary motivation for vaccination was protection against HPV infection and related diseases. A noteworthy finding was that only 11.2% of participants had been vaccinated on the recommendation of a medical practitioner, underscoring the pivotal role of healthcare professionals in influencing vaccine acceptance. The enhancement of collaboration between general practitioners, public health specialists and gynaecologists is imperative for the provision of consistent and accurate information during routine visits. While the majority of participants were informed about the HPV vaccine, less than 20% relied on healthcare workers as their primary source of information. The Internet emerged as the most prevalent source of information, aligning with the findings of previous studies. [20]

Conclusions

Human papillomavirus (HPV) is the most common sexually transmitted infection globally. Persistent infection with high-risk types, especially HPV16 and HPV18, can cause precancerous lesions and cancers of the cervix, anus, penis, vulva, vagina, and oropharynx. Anal cancer is strongly linked to HPV16, with the anal transformation zone being particularly susceptible. Factors that increase risk include HIV infection, having numerous sexual partners, engaging in receptive anal intercourse, smoking, and a history of cervical or vulvar lesions.

Prevention relies on HPV vaccination, ideally before sexual debut, and safe sexual practices. Vaccines reduce infection, transmission, and HPV-related cancers, while condom use offers additional protection. Expanding vaccine coverage and public awareness is key to lowering global HPV-associated cancer rates.

Disclosure

Conceptualization, MK, and JK; methodology, MK; software, JK; check, KK, JC, PF, WK, AS; formal analysis, MK, WK; investigation, JC, AS; resources, PF; data curation, JC, MK; writing- rough preparation, PF, WK, AS; writing- review and editing, KK, PF, JC; visualization, KK, JK, MK, JC, PF; supervision, MK; project administration, JK; receiving funding, PF

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