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STUDY OF SOME MEDICINE USED IN HUMAN PAPILLOMA VIRUS (HPV)

Priyanka S. Gaikwad, Srushti C. Bhende, Vanshika P. Gondkar

Dr. J. J. Magdum Pharmacy College, Jaysingpur 416101-Maharashtra, India.

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For Correspondence:

Vanshika P. Gondkar

Dr. J. J. Magdum Pharmacy College, Jaysingpur 416101-Maharashtra, India.

E-mail:

vanshikagondkar@gmail.com

ABSTRACT

Human papillomavirus (HPV) is one of the most prevalent sexually transmitted infections worldwide and a major etiological factor in the development of cervical cancer and other anogenital and oropharyngeal malignancies. HPV comprises more than 200 related DNA viruses, broadly classified into low-risk and high-risk types based on their oncogenic potential. Persistent infection with high-risk types, particularly HPV 16 and 18, plays a critical role in carcinogenesis through the action of viral oncoproteins E6 and E7, which inactivate tumor suppressor proteins such as p53 and retinoblastoma (pRb). This review provides a comprehensive overview of HPV, including its epidemiology, transmission, pathogenesis, clinical features, diagnosis, management, and prevention strategies. Diagnostic approaches such as Pap smear cytology, HPV DNA and mRNA testing, and p16 immunostaining are essential for early detection of precancerous lesions. Although there is no definitive cure for HPV infection, various treatment modalities are available for managing associated lesions, including topical therapies, surgical procedures, and advanced cancer treatments. Furthermore, the review highlights the therapeutic potential of selected medicinal agents, including natural products such as turmeric (*Curcuma longa*), neem (*Azadirachta indica*), and betulinic acid, as well as synthetic drugs like tretinoin, tamoxifen, and toremifene. These agents exhibit promising antiviral, immunomodulatory, and anticancer properties, particularly in HPV-associated conditions. Preventive strategies, especially vaccination and regular screening, remain the most effective approaches to reduce the global burden of HPV-related diseases. Continued research into novel therapeutic agents and targeted interventions is essential for improving clinical outcomes and achieving long-term disease control.

INTRODUCTION

Human papillomavirus (HPV) comprises a diverse group of more than 200 related DNA viruses belonging to the Papillomaviridae family, of which over 40 types infect the anogenital tract [1]. These viruses are broadly categorized into low-risk and high-risk types based on their oncogenic potential. Low-risk HPV types, such as HPV 6 and 11, are primarily associated with benign lesions including genital warts (condylomaacuminata), whereas high-risk types, particularly HPV 16 and 18, are strongly implicated in the development of various malignancies [2]. Persistent infection with oncogenic HPV types is recognized as the principal etiological factor in cervical cancer and is also significantly associated with other anogenital cancers (anal, vulvar, vaginal, and penile) as well as oropharyngeal cancers [2].

HPV is the most prevalent sexually transmitted infection worldwide, with a majority of sexually active individuals acquiring the infection at some point in their lifetime. The virus is predominantly transmitted through direct skin-to-skin contact during vaginal, anal, or oral sexual activity. However, non-sexual modes of transmission, including contact with contaminated surfaces (fomites) and vertical transmission from mother to child during childbirth, have also been documented, although less commonly [3]. The natural history of HPV infection typically involves transient infection, with most cases being cleared spontaneously by the host immune system within 1–2 years. Nevertheless, persistent infection with high-risk HPV types can lead to progressive cellular abnormalities, culminating in precancerous lesions and invasive carcinoma.

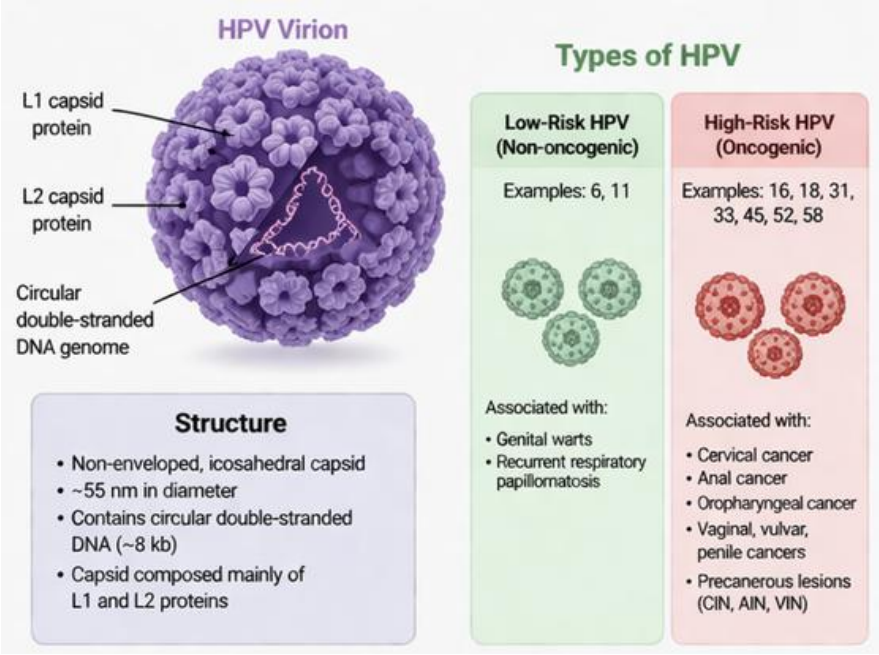

Figure 1: Structure and Types of Human Papillomavirus (HPV)

The introduction of prophylactic HPV vaccines has significantly transformed the landscape of HPV prevention. Currently available vaccines include bivalent (targeting HPV 16 and 18), quadrivalent (targeting HPV 6, 11, 16, and 18), and nonavalent vaccines (covering additional oncogenic types such as 31, 33, 45, 52, and 58) [4]. These vaccines have demonstrated high efficacy in preventing infections caused by high-risk HPV types and have been shown to reduce the incidence of cervical intraepithelial neoplasia and genital warts. Notably, vaccination programs have the potential to prevent approximately 70% of cervical cancer cases and up to 90% of genital warts, particularly when administered prior to exposure to the virus [4]. Consequently, widespread immunization, combined with regular screening programs, represents a cornerstone strategy in reducing the global burden of HPV-associated diseases.

Types of HPV

HPV types are broadly classified into low-risk and high-risk categories (Table 1) [5]

Table 1: Classification of HPV Types and Associated Diseases

HPV Type Category	Common Types	Associated Conditions	Oncogenic Potential
Low-risk HPV	6, 11	Genital warts, papillomas	Non-oncogenic
High-risk HPV	16, 18	Cervical cancer, anal cancer, oropharyngeal cancer	High
Additional high-risk	31, 33, 45, 52, 58	Cervical intraepithelial neoplasia (CIN), cancers	High

3. Epidemiology

Human papillomavirus (HPV) infection represents a major global public health concern due to its high prevalence and strong association with multiple cancers. It is estimated that approximately 80% of sexually active individuals will acquire HPV infection at some point during their lifetime, making it the most common sexually transmitted infection worldwide [5]. The burden of HPV infection varies significantly across regions, influenced by socioeconomic status, access to healthcare, screening programs, and vaccination coverage.

3.1 Global Prevalence and Incidence

The prevalence of HPV infection is particularly high among young adults shortly after the onset of sexual activity. Globally, cervical HPV infection affects a substantial proportion of women, with the highest prevalence reported in sub-Saharan Africa, Latin America, and parts of Asia [6]. In these regions, the incidence of HPV-associated cancers, especially cervical cancer, remains disproportionately high due to limited access to routine screening and vaccination programs.

In contrast, high-income countries have observed a significant decline in HPV prevalence and related diseases following the implementation of organized vaccination and screening initiatives. For instance, widespread use of HPV vaccines has led to a marked reduction in infections caused by HPV types 16 and 18, which are responsible for the majority of cervical cancer cases [7]. Despite these advances, disparities in vaccine uptake and healthcare access continue to pose challenges in achieving global control of HPV infection.

3.2 Risk Factors for HPV Infection

Several behavioral, biological, and socioeconomic factors contribute to the risk of HPV acquisition and persistence:

- **Multiple sexual partners:** Increased number of sexual partners elevates the likelihood of exposure to HPV.
- **Early age of sexual debut:** Early initiation of sexual activity is associated with a higher risk of infection.
- **Immunosuppression:** Individuals with compromised immune systems, such as those with HIV infection or undergoing immunosuppressive therapy, are more prone to persistent HPV infection and progression to malignancy [8].
- **Lack of screening and vaccination:** Limited access to preventive healthcare services significantly increases disease burden.

Persistent infection with high-risk HPV types is the most critical determinant for the development of precancerous lesions and invasive cancers.

4. Pathogenesis of HPV

HPV exhibits a unique life cycle that is closely linked to the differentiation of epithelial cells. The virus primarily infects the basal layer of stratified squamous epithelium through microabrasions in the skin or mucosal surfaces. Following entry, HPV establishes infection in basal keratinocytes and maintains its genome as an episome within the host cell nucleus [9].

As infected basal cells differentiate and migrate toward the epithelial surface, the virus undergoes replication and produces viral proteins necessary for its life cycle. In most cases, the infection is transient and cleared by the host immune response. However, in certain instances, particularly with high-risk HPV types, the viral DNA integrates into the host genome, leading to persistent infection and cellular transformation.

4.1 Mechanism of Oncogenesis

The oncogenic potential of high-risk HPV types is primarily mediated by the viral oncoproteins E6 and E7. These proteins disrupt normal cell

cycle regulation by targeting key tumor suppressor pathways:

- **E6 protein:** Binds to and promotes the degradation of the tumor suppressor protein p53, impairing DNA repair mechanisms and apoptosis.
- **E7 protein:** Inactivates the retinoblastoma protein (pRb), leading to uncontrolled progression of the cell cycle from the G1 to S phase [10].

The combined action of E6 and E7 results in genomic instability, accumulation of genetic mutations, and uncontrolled cellular proliferation. Over time, these changes can lead to the development of cervical intraepithelial neoplasia (CIN), which may progress to invasive cervical cancer if left untreated.

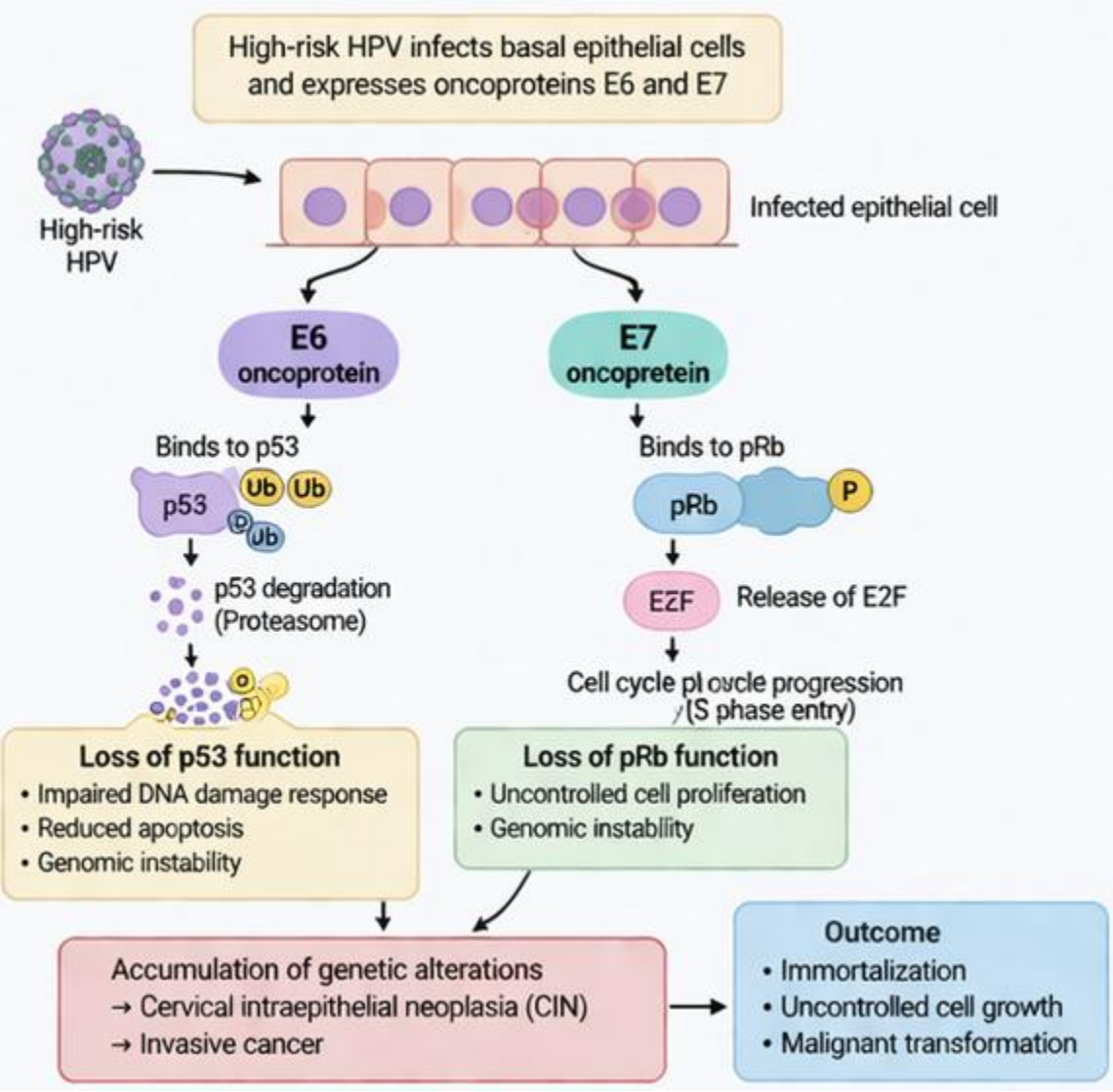

Figure 2: Mechanism of HPV-Induced Oncogenesis

4.2 HPV-Associated Malignancies

Persistent infection with high-risk HPV types is causally associated with several malignancies:

- **Cervical cancer:** Nearly all cases are linked to HPV infection, with types 16 and 18 accounting for approximately 70% of cases [11].
- **Anal cancer:** Strongly associated with HPV, particularly among immunocompromised individuals.
- **Oropharyngeal cancer:** Increasing incidence, especially in men, largely attributed to HPV type 16.

- **Other cancers:** Includes vulvar, vaginal, and penile cancers [12].

The progression from initial HPV infection to cancer is typically slow, often taking years to decades, which provides a critical window for early detection and intervention through screening programs.

5. Clinical Features

Most HPV infections are asymptomatic. When present:

- **Genital warts:** Cauliflower-like lesions caused by HPV 6 and 11
- **Cervical infection:** Often asymptomatic until advanced stages
- **Oropharyngeal infection:** Symptoms include sore throat, dysphagia, and neck mass [12]

6. Diagnosis of HPV

The diagnosis of human papillomavirus (HPV) infection depends on the anatomical site involved, clinical presentation, and the purpose of testing (screening vs. diagnostic confirmation). Since most HPV infections are asymptomatic, laboratory-based screening plays a crucial role in early detection, particularly for cervical cancer prevention.

6.1 Clinical Diagnosis

Clinical evaluation is primarily useful for identifying visible lesions such as genital warts. These lesions typically present as soft, flesh-colored, cauliflower-like growths in the anogenital region. Visual inspection by trained healthcare professionals remains a simple and effective initial diagnostic approach for low-risk HPV infections [13]. The various diagnostic approaches used for HPV detection are summarized in Table 2.

Table 2: Diagnostic Methods for HPV Infection

Method	Principle	Advantages	Limitations
Clinical examination	Visual inspection of lesions	Simple, cost-effective	Limited sensitivity
Pap smear	Cytological evaluation of cervical cells	Detects precancerous changes	Requires repeated screening
HPV DNA testing	Detection of viral DNA (PCR/Hybrid Capture)	High sensitivity	Lower specificity
HPV mRNA testing	Detection of E6/E7 oncogene expression	High specificity	Costly
p16 immunostaining	Biomarker for oncogenic HPV activity	Useful in cancer diagnosis	Requires biopsy

6.2 Cytological Screening (Pap Smear)

The Papanicolaou (Pap) smear is a widely used screening tool for detecting precancerous and cancerous changes in cervical epithelial cells. It identifies cytological abnormalities such as:

- Atypical squamous cells of undetermined significance (ASC-US)
- Low-grade squamous intraepithelial lesions (LSIL)
- High-grade squamous intraepithelial lesions (HSIL)

Regular Pap screening has significantly reduced cervical cancer incidence and mortality in populations with established screening programs [14].

6.3 HPV DNA Testing

HPV DNA testing detects the presence of high-risk HPV genotypes using molecular techniques such as polymerase chain reaction (PCR) and Hybrid Capture 2 assays. It is highly sensitive and is recommended for:

- Women aged ≥30 years as a primary screening tool
- Co-testing along with Pap smear
- Follow-up of abnormal cytology results

This method enables early identification of individuals at high risk for developing cervical cancer [15].

6.4 HPV mRNA Testing

HPV mRNA testing detects the expression of viral oncogenes E6 and E7, which are directly involved in malignant transformation. Compared to DNA testing, mRNA assays provide higher specificity for identifying clinically significant infections that are more likely to progress to cancer [16].

6.5 Immunohistochemistry (p16 Biomarker)

p16INK4aimmunostaining is widely used as a surrogate marker for high-risk HPV oncogenic activity. Overexpression of p16 protein indicates disruption of the pRb pathway by HPV E7 protein and is commonly used in the evaluation of cervical biopsies and oropharyngeal tumors [17].

6.6 Diagnosis of Oropharyngeal HPV

Diagnosis of HPV-related oropharyngeal cancer involves:

- Tumor biopsy
- HPV DNA or RNA detection
- p16 immunohistochemistry

These methods help confirm HPV involvement and guide prognosis and treatment decisions [18].

7. Management of HPV Infection

Currently, there is no definitive cure for HPV infection itself; however, various therapeutic strategies are available to manage HPV-associated lesions and prevent disease progression. Treatment approaches depend on the type, location, and severity of the lesions. Current treatment options for HPV-related conditions are summarized in Table 3

Table 3: Management Strategies for HPV Infection

Condition	Treatment Options	Mechanism/Action
Genital warts	Imiquimod, Podophyllotoxin, Cryotherapy	Immune stimulation / tissue destruction
Cervical precancer (CIN)	LEEP, Conization, Laser therapy	Removal of abnormal tissue
HPV-related cancers	Surgery, Chemotherapy, Radiotherapy	Tumor elimination
Emerging therapies	Therapeutic vaccines, Immunotherapy	Target viral oncogenes

7.1 Management of Genital Warts

Genital warts caused by low-risk HPV types (6 and 11) can be treated using patient-applied or clinician-administered therapies:

Topical Treatments

- **Imiquimod:** An immune response modifier that enhances local cytokine production and antiviral activity
- **Podophyllotoxin:** An antimitotic agent that causes necrosis of wart tissue
- **Trichloroacetic acid (TCA):** A chemical cauterizing agent that destroys wart tissue

Physical and Surgical Methods

- **Cryotherapy:** Freezing of lesions using liquid nitrogen
- **CO_2 laser ablation:** Precise removal of lesions
- **Electrosurgery:** Destruction using electrical current

These methods are effective but may require multiple sessions and are associated with recurrence [19].

7.2 Management of Cervical Precancerous Lesions

Early detection and treatment of cervical intraepithelial neoplasia (CIN) are critical in preventing progression to invasive cancer.

- **Loop Electrosurgical Excision Procedure (LEEP):** Removes abnormal cervical tissue using a wire loop
- **Cold knife conization:** Surgical excision of a cone-shaped portion of the cervix
- **Laser excision:** Precise removal of dysplastic tissue

These procedures are highly effective in treating high-grade lesions (CIN 2/3) [20].

7.3 Management of HPV-Related Cancers

Treatment of HPV-associated malignancies depends on cancer type and stage and may include:

- Surgery
- Radiation therapy
- Chemotherapy
- Targeted therapy and immunotherapy

Multimodal treatment approaches have improved survival outcomes, particularly in HPV-positive oropharyngeal cancers, which tend to have better prognosis compared to HPV-negative cancers [21].

7.4 Emerging Therapeutic Approaches

Recent research is focused on developing targeted therapies against HPV infection, including:

- Therapeutic vaccines targeting E6 and E7 oncoproteins
- Antiviral agents
- Immunotherapies aimed at enhancing host immune response

These strategies hold promise for future management of persistent HPV infections and associated malignancies [22].

8. Prevention

8.1 Vaccination

- Bivalent (HPV 16, 18)
- Quadrivalent (HPV 6, 11, 16, 18)
- Nonavalent (additional types)

8.2 Screening

- Pap smear
- HPV DNA testing

8.3 Safe Practices

- Barrier contraception
- Sexual health awareness [15]

9. Medicinal Agents in HPV Management

9.1 Turmeric (*Curcuma longa*)

Turmeric, derived from the rhizomes of ***Curcuma longa***, is widely recognized for its therapeutic properties, primarily attributed to its active polyphenolic compounds known as curcuminoids, with curcumin being the most prominent. Curcumin exhibits potent anti-inflammatory, antioxidant, antiviral, and anticancer activities. In the context of HPV-related diseases, curcumin has been shown to interfere with viral oncogenesis by downregulating the expression of HPV oncoproteins E6 and E7. This action leads to restoration of tumor suppressor proteins such as p53 and retinoblastoma (pRb), thereby promoting apoptosis and inhibiting uncontrolled cell proliferation. Additionally, curcumin modulates multiple cellular signaling pathways, including NF-κB, STAT3, and AP-1, which are involved in inflammation and cancer progression. Preclinical studies have demonstrated that curcumin suppresses tumor growth and induces cell cycle arrest in HPV-positive cervical cancer models. Its low toxicity and wide safety margin make it a promising adjunctive therapeutic agent in HPV-associated malignancies [16].

9.2 Neem (*Azadirachta indica*)

Azadirachta indica (neem) is a medicinal plant extensively used in traditional systems of medicine due to its broad spectrum of biological activities. Neem contains various bioactive compounds such as azadirachtin, nimbin, nimbidin, and quercetin, which contribute to its antiviral, antibacterial, antifungal, and immunomodulatory properties.

In HPV-related conditions, neem extracts have shown potential in enhancing host immune responses and reducing viral persistence. The immunomodulatory effects include stimulation of cell-mediated immunity, activation of macrophages, and increased production of cytokines, which collectively help in clearing viral infections. Neem also exhibits antioxidant properties that protect cells from oxidative stress-induced damage, which is a contributing factor in carcinogenesis. Furthermore, certain neem-derived compounds have demonstrated inhibitory effects on tumor cell proliferation and may play a supportive role in preventing the progression of HPV-associated lesions [17].

9.3 Betulinic Acid

Betulinic acid is a naturally occurring pentacyclictriterpenoid found in the bark of several plant species, particularly birch trees. It has gained significant attention due to its selective anticancer activity and minimal toxicity to normal cells.

In HPV-associated malignancies, betulinic acid exerts its therapeutic effects primarily through the induction of apoptosis via the mitochondrial (intrinsic) pathway. It enhances the expression of pro-apoptotic proteins (e.g., Bax) while downregulating anti-apoptotic proteins (e.g.,

Bcl-2), leading to cytochrome c release and caspase activation. Importantly, betulinic acid has been shown to counteract the effects of HPV E6 oncoprotein by restoring p53 function, thereby promoting apoptosis in HPV-infected cells. It also inhibits key signaling pathways such as NF-κB and reduces cellular proliferation. Studies using HPV-positive cervical cancer cell lines (e.g., HeLa and SiHa) have demonstrated significant growth inhibition and reduced tumor viability, highlighting its potential as a therapeutic agent in HPV-related cancers [18].

9.4 Tretinoin

Tretinoin, also known as all-trans retinoic acid, is a derivative of vitamin A widely used in dermatology. It exerts its effects by binding to nuclear retinoic acid receptors (RARs), which regulate gene expression involved in cell differentiation and proliferation.

In HPV-induced lesions, particularly flat warts (verruca plana), tretinoin promotes normalization of keratinocyte differentiation and accelerates epithelial cell turnover. This leads to exfoliation and gradual elimination of HPV-infected cells. Tretinoin also reduces hyperkeratinization and prevents the accumulation of abnormal epithelial cells. Additionally, it may enhance local immune responses by increasing antigen exposure, thereby facilitating immune-mediated clearance of the virus. Due to its non-invasive mechanism and minimal risk of scarring, tretinoin is especially useful in pediatric and cosmetically sensitive areas such as the face [19].

9.5 Tamoxifen

Tamoxifen is a selective estrogen receptor modulator (SERM) widely used in the treatment of hormone receptor-positive breast cancer. It acts by competitively binding to estrogen receptors, thereby inhibiting estrogen-mediated cellular proliferation.

Although not a primary treatment for HPV infection, tamoxifen has shown potential in the management of HPV-related malignancies due to its antiproliferative and pro-apoptotic effects. It has been reported to modulate growth factor signaling pathways, reduce insulin-like growth factor (IGF-1) activity, and increase sex hormone-binding globulin levels, ultimately limiting tumor growth. Additionally, tamoxifen may induce apoptosis through mechanisms involving inhibition of protein kinase C and alterations in intracellular calcium levels. These properties suggest a possible adjunctive role in controlling the progression of HPV-associated cancers, particularly in hormone-responsive tissues [20].

9.6 Toremifene

Toremifene is another selective estrogen receptor modulator structurally related to tamoxifen and used primarily in the treatment of metastatic breast cancer in postmenopausal women. It exhibits both estrogenic and antiestrogenic effects depending on the target tissue.

The anticancer activity of toremifene is largely attributed to its ability to block estrogen receptor signaling, thereby inhibiting tumor growth in hormone-dependent cancers. In addition to its antiestrogenic effects, toremifene has been shown to induce apoptosis, regulate oncogene expression, and interfere with growth factor-mediated signaling pathways. Although its direct role in HPV infection is limited, its ability to inhibit cell proliferation and promote apoptosis suggests potential utility as an adjunct therapy in HPV-related malignancies [21].

CONCLUSION

Human papillomavirus (HPV) continues to pose a significant global health challenge due to its high prevalence and strong association with various malignancies, particularly cervical cancer. The classification of HPV into low-risk and high-risk types is crucial in understanding its clinical implications, with persistent infection by high-risk types being the primary driver of carcinogenesis. The molecular mechanisms involving E6 and E7 oncoproteins highlight the complexity of HPV-induced tumor development and provide potential targets for therapeutic intervention.Advancements in diagnostic techniques, including HPV DNA testing, mRNA assays, and biomarker-based methods, have greatly improved early detection and risk stratification. Although there is no complete cure for HPV infection, current management strategies effectively control HPV-associated lesions and prevent disease progression. Preventive measures, especially vaccination and routine screening programs, have demonstrated substantial success in reducing HPV-related morbidity and mortality.

In addition, emerging evidence supports the potential role of natural and synthetic medicinal agents such as curcumin, neem, betulinic acid, tretinoin, and selective estrogen receptor modulators in the management of HPV-related conditions. These agents offer promising adjunctive therapeutic benefits through their antiviral, immunomodulatory, and anticancer mechanisms.

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