



REVIEW ARTICLE; AN OVERVIEW OF CERVICAL CANCER

Solomon Matthias Gamde¹, Ephraim Ephron Yerembede², Ikeh C Anthony³, Isaac Igbinosa⁴, Hauwa Kabiru⁵, Alheri Lawan Lailai⁶

¹Department of Medical Laboratory Science, Plateau State University, Bokokos, Nigeria

²Department of Medical Laboratory Science, University of Abuja, Nigeria

³Ahmadu Bello University, Zaria, Nigeria

⁴Department of Medical Laboratory Science, Federal University Wukari, Taraba State, Nigeria

⁵Department of Medical Laboratory Science, Baze University, Abuja, Nigeria

⁶Department of Nursing Science, Cosmopolitan University, Abuja, Nigeria

Correspondence: Solomon Matthias Gamde

Department of Medical Laboratory Science,

Plateau State University, Bokokos,

Plateau State, Nigeria.

solomongamde@plasu.edu.ng

Abstract

Cervical cancer is one of the leading causes of cancer-related morbidity and mortality in women globally. Despite advances in understanding the lesions, critical gaps remained unidentified. Most existing reports are merely observational and limit causal inferences. Therefore, studies assessing cervical pathology, including limited data on genotype-specific HPV responses to treatments, screening adherence, and the efficacy of novel immunotherapeutic outcomes, are needed. Inspired by the increased global optimism about the possibility of eliminating cervical cancer, the World Health Organization has recently launched a global initiative to accelerate the elimination of cervical cancer as a public health problem, its goal is to increase HPV vaccination coverage services and to ensure mid-adult women screening services and accessibility to appropriate treatment and care to those who need treatment. Prospective, multicenter studies are crucial to refining clinical guidelines.

Keywords: Cervical lesions, Human papillomavirus, Cervical cytology screening, Vaccination

Introduction

Cervical cancer is one of the leading causes of cancer-related morbidity and mortality in women globally. It is estimated that approximately 600,000 women are diagnosed with, and more than 300,000 women die from, cervical cancer worldwide annually (Sung et al., 2020). A disproportionate amount of its global burden is experienced by women living in low- and middle-income countries (LMICs), where it is often the first or second leading cause of cancer cases and deaths in women (Gamde et al., 2024a).

With the discovery of an obligate causative infectious agent, high-risk *Human papillomavirus* (HPV), elucidation of key steps in its natural history, and availability of highly effective primary and secondary prevention technologies, cervical cancer is an eminently preventable malignancy. Indeed, in countries with routine, effective cervical cancer screening and early detection programs, its incidence and mortality rates have declined precipitously over the past half century (Sung et al., 2020; Vaccarella et al., 2013). Inspired by the increased global optimism about the possibility of eliminating cervical cancer, the World Health Organization, with support from 194 countries, has recently launched a global initiative to accelerate the elimination of

cervical cancer as a public health problem, its goal is to increase the 90% of HPV vaccination coverage services and to ensure 70% of mid-adult women screening services and 90% of accessibility to appropriate treatment and care to those who need treatment (Das, 2021; Kuehn, 2021; Simelela, 2021).

There are 4 key stages in the development of cervical cancer: HPV acquisition, HPV persistence, progression to cervical premalignant lesions (low-grade squamous intraepithelial lesions to high-grade intraepithelial lesion), and development of invasive cancer (Schiffman et al., 2007). In addition to factors such as age, parity, smoking status, and hormonal contraceptive use, HIV co-infection, although not causal, has a profound impact on several steps in the natural history of HPV (HPV cofactor) that increases HPV-related cancer risk in women (Selji et al., 2025; Gamde et al., 2025).

Cervix

The cervix, a vital component of the female reproductive system, plays crucial roles in reproductive and gynaecological health. Its unique morphology and dynamic changes throughout a woman's life have significant implications for fertility, menstruation, and disease susceptibility (Kurmar, 1994). Anatomically, the cervix is cylindrical in shape and forms the lower part of the uterus, measuring approximately 2-4 cm in length. It connects the uterine cavity to the vaginal canal, comprising two regions: the endocervix, lined by glandular epithelium, and the ectocervix, covered by stratified squamous epithelium (Bosch et al., 2023). The external Os is the opening into the vagina, and the internal Os leads into the uterine cavity. Histologically, the cervix consists of three layers:

1. **Epithelium:** The ectocervix is covered by non-keratinised stratified squamous epithelium, while the endocervix is lined with columnar epithelium that produces mucus. Where the squamous and columnar epithelium meet is called the squamo-columnar junction (SCJ), which is dynamic and shifts with hormonal changes and ageing (Kurmar, 1994). The region where metaplasia occurs is termed the transformation zone (TZ), a critical site for the development of cervical intraepithelial neoplasia (CIN) and cervical cancer (Bhatla et al., 2021). The location of the TZ varies with reproductive age and menopause.
2. **Stroma:** A dense connective tissue layer containing collagen, elastin, and smooth muscle fibres provides structural integrity.
3. **Glands:** Mucus-secreting cervical glands play a critical role in modulating sperm transport and infection defence (Shaw et al., 2022).

The morphology of the cervix is integral to its diverse functions and disease susceptibility. Understanding the complex structure aids in advancing diagnostic and therapeutic approaches for cervical pathologies (Beckmann, 2013).

The transformation zone (TZ) is the primary site of cervical dysplasia development. This region undergoes dynamic changes during hormonal fluctuations, due to factors such as adolescence and pregnancy. These hormonal changes cause squamous metaplasia as columnar cells are replaced by immature squamous cells (Beckmann, 2013; Kumar et al., 2007). These Immature metaplastic squamous cells are more vulnerable due to their rapid division and incomplete maturation; hence lack fully developed protective mechanisms, thereby providing an entry point for some viral infections such as Human Papillomavirus through microabrasions (Kumar et al., 2007).

Pathogenesis

HPV infection and cellular transformation:

HPV, a double-stranded DNA virus targeting the basal layer of the squamous epithelium, has oncoproteins that disrupt normal cellular functions, hence making the basal cells the primary site of initial transformation. More than 200 HPV types exist, which are categorised into three risk groups: high-risk, intermediate-risk, and low-

risk, based on their association with premalignant and malignant lesions of the cervix (Gamde et al., 2024a). High-risk HPV types are strongly associated with cervical premalignant lesions, as they can cause persistent infections and lead to the development of cervical premalignant lesions, which may eventually progress to cancer. The high-risk types include HPV strains 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, and 59. Notably, HPV 16 and 18 are the most common high-risk strains, responsible for approximately 70% of cervical cancer cases worldwide (Bhatla et al., 2021; Gamde et al., 2025a). These two HPV species, $\alpha 9$ (also known as HPV16-like) and $\alpha 7$ (also known as HPV18-like), are of particular importance in the context of cervical premalignant lesion diagnosis (Evans et al., 2022). HPV-16 belongs to the Alpha-papillomavirus genus. It is responsible for nearly 50–60% of all cervical cancer cases, making it the most prevalent and oncogenic HPV type (Bhatla et al., 2021; Youn et al., 2020). The pathogenesis begins with micro abrasions in the cervical epithelium, allowing HPV to access the basal layer where it initiates infection. The earliest cellular changes occur in the basal and para-basal layers of the cells, then progressing to involve the full thickness of the epithelium, which correlates with increasing risk for invasive cervical cancer if left untreated.

Viral oncogene expression

The carcinogenic potential of HPV-16 is primarily attributed to the expression of three of its early region oncoproteins: two major oncoproteins, E6 and E7, and a minor oncoprotein, E5. These oncoproteins play a crucial role in HPV 16-mediated cervical carcinogenesis by disrupting the normal cellular regulatory pathways (Ren et al., 2020).

E5 Protein: The E5 protein of HPV plays a crucial role in augmenting the growth and survival signals within the host cell. By enhancing signalling pathways such as the epidermal growth factor receptor (EGFR) pathway, E5 promotes cellular proliferation and helps in the maintenance of malignant cells. In patients with compromised cellular immunity, the role of E5 in promoting oncogenesis becomes more pronounced, as the immune system is less capable of containing the unregulated state. In HIV infection, growth is induced by this protein. The interaction of E5 with host immunity suggests a potentiated oncogenic environment in the context of HIV co-infection (Ren et al., 2020).

E6 Protein: E6 is perhaps the most notorious of the HPV oncogenic proteins due to its ability to bind to and promote the degradation of the tumour suppressor protein p53. This interaction leads to the inhibition of apoptosis, allowing HPV-infected cells to survive and accumulate genetic damage. In the setting of HIV co-infection, the efficacy of the immune system to clear such damaged cells is diminished, which may lead to an increased risk of malignant transformation and cancer progression. Furthermore, studies have shown that HIV proteins like Vpr (Viral Protein R) can modulate the activity of molecules involved in the p53 pathways, thereby enhancing the oncogenic potential of E6 (Bosch et al., 2023; Peng et al., 2024; Singini et al., 2022; Zhang et al., 2022).

E7 Protein: The E7 protein contributes to oncogenesis by binding to the retinoblastoma (pRb) family of proteins, disrupting their function and thereby driving the cell cycle progression inappropriately. This disruption is crucial for viral replication and persistence but also leads to increased cellular turnover and potential malignant transformation. In HIV-positive individuals, the regulatory oversight over cell cycle progression may be further compromised due to systemic immune suppression, enhancing the impact of E7 on cervical epithelial cell transformation (Peng et al., 2024; Ren et al., 2020; Shaw et al., 2022; Singini et al., 2022).

Immune evasion

Also, E6 and E7 can cooperate to induce chromosomal aberrations, activate telomerase reverse transcriptase (hTERT) (Kovachev et al., 2023), and immortalise infected cervical epithelial cells, eventually resulting in malignant transformation (Liu et al., 2019; Nott et al., 2020).

The pathogenesis of cervical dysplasia is a multifaceted process driven by high-risk HPV infection, viral oncoprotein activity, and immune system evasion (Kurmar, 1994). Understanding these mechanisms is fundamental to effective prevention, early detection, and treatment strategies.

Risk Factors

Several studies have shown and proven that the primary cause of premalignant and malignant lesions of the cervix is ongoing infection with specific strains of the human papillomavirus (HPV) (Figueiredo et al., 2023; Gilham et al., 2023; Smith et al., 2023). Approximately 70% of cervical cancer cases worldwide are caused by HPV types 16 and 18. This finding has been reaffirmed by studies done recently by the National Cancer Institute and other similar organisations (National Cancer Institute [NCI], 2024; World Health Organization/International Agency for Research on Cancer. Global Cancer Observatory [WHO/IARC. GLOBOCAN], 2021). Various factors, such as age (Adeoye et al., 2018; Akhiwu et al., 2020; Basoya et al., 2022; Teame et al., 2018; Yuan et al., 2023), obesity (Bohn et al., 2022; Coffey et al., 2016; Frumovitz et al., 2014; Sassenou et al., 2021), marital status (Aboyegi et al., 2018; Ezechi et al., 2014; Global Report, 2013), hormonal imbalances (Iversen et al., 2021; Lasche et al., 2022; Zidi, Sghaier et al., 2020), genetic predisposition (Chandra et al., 2022; Liu et al., 2022b; Yadav et al., 2023a), and environmental exposures (Korsakov et al., 2022; Kyler et al., 2017), have been implicated in uterine cervical cancer development.

However, there are also numerous other risk factors, such as reproductive and sexual factors (Centres for Disease Control and Prevention [CDC], 2021; Nwobodo et al., 2017; Teame et al., 2018), and sexual intercourse at a young age (Ghebre et al., 2017; Roura et al., 2014). Behavioural factors such as smoking (Donkoh et al., 2019; Makuza et al., 2015; Manglaiviraj et al., 2008; NCI, 2024), use of alcohol (Bagnardi, 2015; Boffetta et al., 2006; Ho et al., 2017; Weiderpass et al., 2001).

The invasive cancer development process could prolong for up to 20 years from the precursor premalignant lesion caused by sexually transmitted HPV to the malignant stage (Yuan et al., 2021).

Grading Of Cervical Lesions

Numerous terms have been used to describe the premalignant lesion of the cervix. These changes are part of a continuum of slight to severe atypia, which can progress to invasive carcinoma. Cervical lesion grading depends on the degree of cytological atypia and the thickness of the epithelium involved (Kurman et al., 2014; Solomon et al., 2019). Three classification systems have been used in recent years. One relies on the term dysplasia (mild, moderate, or severe) to describe the early premalignant changes in the epithelium, and carcinoma in-situ to describe the most advanced premalignant changes. A second nomenclature that describes the same histological features utilises the terminology of cervical intraepithelial neoplasia (CIN): CIN I (mild dysplasia), CIN II (moderate dysplasia) and CIN III (severe dysplasia to carcinoma in-situ) (Kurman et al., 2014). A new terminology, the Bethesda system (TBS), for reporting cervical or vaginal cytological diagnosis was introduced in 1988 and revised in 1991 to establish uniform terminology and standardise diagnostic reports (Alrajjal et al., 2021). TBS was further reviewed and adopted by the National Cancer Institute (NCI) and the American Society for Colposcopy and Cervical Pathology Consensus Conference in May 2001 (Solomon et al., 2019). The 2001 revision in TBS reflects the improved understanding of the epidemiology and natural history of cervical epithelial abnormalities and cervical cancer. These revisions are designed to facilitate communication between the clinician and the laboratory and to improve the clinician's ability to accurately interpret the cytology report and plan initial management of any abnormality (Solomon et al., 2019).

The cervical cytology screening outcomes are mostly reported according to the Bethesda 2001 reporting system. The system classified cervical cytology results by initially grouping into specimen adequacy as satisfactory or unsatisfactory for evaluation. Specimens satisfactory for evaluation are further classified into negative for intraepithelial lesion or malignancy (NILM), for which no epithelial abnormality was identified. The epithelial cell abnormalities (cervical precancerous lesions) were classified as Atypical squamous cells,

which are qualified as “of undetermined significance” (ASC-US) or “cannot exclude HSIL” (ASC-H). The squamous intraepithelial lesion (SIL) was subdivided into low-grade SIL (LSIL) and high-grade SIL (HSIL) for reporting the non-invasive squamous cervical abnormalities. The NILM, atypical squamous cell and SIL groups were further classified into Normal for NILM, Mild dysplasia for ASC-US and LSIL, and Severe dysplasia for ASC-H and HSIL (Alrajjal et al., 2021; Kurman et al., 2014; Solomon et al., 2019).

Atypical squamous cells of undetermined significance (ASCUS) indicate inflammatory cells. Low-grade squamous intraepithelial lesion (LSIL) (same as mild dysplasia and CIN I) indicates human papillomavirus (HPV) infection, where only the deepest third of the epithelium is affected, from the basal layer upward with maturation more superficially. High-grade squamous intraepithelial lesion (HSIL) includes moderate dysplasia, severe dysplasia, and carcinoma in situ (CIN II & III), which affects two-thirds of the thickness of the epithelium, and severe dysplasia shows no maturation throughout the full thickness (Kurman et al., 2014; Solomon et al., 2019).

Co-infection of HIV and HPV

Both human immunodeficiency virus (HIV) and human papillomavirus (HPV) are sexually transmitted infections. HIV, a retrovirus that primarily targets the immune system, specifically CD4⁺ T cells, resulting in progressive immunosuppression (Deeks et al., 2015). Conversely, HPV is a DNA virus known for its tropism for epithelial cells and its potential to cause both benign and malignant lesions (Doorbar et al., 2012).

Human papillomavirus (HPV) and HIV infection are highly prevalent in Sub-Saharan Africa (CDC, 2021; WHO/IARC. GLOBOCAN, 2012). Increased risk of cervical cancer, decreased clearance of precancerous lesions and HPV, and higher rates of HPV acquisition are all linked to HIV (Abebe et al., 2018; American Cancer Society, 2015; Obstetrics and Gynaecology International, 2016). There is an increasing need for cervical cancer prevention, especially in low-resource settings, as HIV-positive women receive antiretroviral therapy (ART) (Bruni et al., 2017; WHO, 2016).

The Viral Oncogenesis Model elaborates on how HPV oncoproteins E6 and E7 disrupt p53 and retinoblastoma protein (pRb) pathways, leading to genomic instability and malignant transformation (Adeyemi et al., 2019). The model suggests that there is a direct correlation between immune status and the severity of HPV-related cervical disease. Studies indicate that immune reconstitution can lead to either regression or stabilization of cervical lesions (Okeke et al., 2020; Musa et al., 2022). However, the extent of immune recovery and its timing in relation to HPV infection are critical determinants of outcomes. Hence The progression from low-grade squamous intraepithelial lesions (LSIL) to high-grade squamous intraepithelial lesions (HSIL) and ultimately to invasive squamous cell carcinoma is governed by the host's immune status, primarily the CD4⁺ T-cell count and viral load suppression (Bello et al., 2021). The continuum of cervical lesion development explains the sequential progression from HPV infection to LSIL (CIN, mild dysplasia), HSIL (CIN II/III moderate-to-severe dysplasia), and invasive cervical cancer (Adeyemi et al., 2019). This conceptual framework informs the analysis of lesion patterns among women living with HIV.

HPV DNA Testing

HPV DNA testing provides direct detection of high-risk HPV strains. Adebayo, Adekunle, & Ajayi, in their study in 2022, advocate its integration into routine screening protocols, citing its superior sensitivity for identifying women at risk of developing cervical lesions. Another studies by Bello and Musa, both in 2021 and 2022 respectively, recommend HPV DNA testing inclusion in routine screening protocols for HIV-positive women due to its predictive value for CIN progression (Bello et al., 2021; Musa et al., 2022). However, its higher cost limits widespread adoption. HPV vaccines, such as Gardasil (Choi et al., 2023) and Cervarix (Roy et al., 2023), protect against the most common high-risk HPV types (particularly HPV 16 and 18). By preventing infection with these types, the vaccines can effectively reduce the likelihood of developing

precancerous cervical lesions and, ultimately, Uterine Cervical Cancer. Widespread vaccination has the potential to substantially decrease the overall incidence of Uterine Cervical Cancer.

Awareness

Awareness of cervical cancer screening and its utilization remain critical in reducing the burden of cervical cancer, especially in resource-limited settings. Studies have shown that a lack of knowledge about cervical cancer, its risk factors, and the importance of regular screening contributes significantly to low screening uptake (Kabir et al., 2005). Socioeconomic factors, cultural beliefs, and misconceptions about screening procedures often hinder women from accessing these services. Furthermore, limited access to healthcare facilities and trained personnel exacerbates the underutilization of screening programs. Addressing these barriers through targeted health education campaigns and integrating cervical screening services into routine health care can improve awareness and uptake, ultimately enhancing early detection and better outcomes for women (Adetule, 2016; Balogun et al., 2012; Ndikom & Ofi, 2012; Rabiou et al., 2011).

Cervical cancer remains a leading cause of morbidity and mortality among women in Nigeria, with most cases presenting at advanced stages. Despite being preventable through human papillomavirus (HPV) vaccination and early screening, the utilization of cervical cancer screening services remains low (Hyacinth et al., 2012). Various studies have highlighted poor awareness and significant barriers to accessing these services, especially in low-resource settings (Emanuel et al., 2015; Ndikom et al., 2012; Sherris et al., 2009).

Barriers to Awareness and Utilization

Several factors contribute to the underutilization of cervical cancer screening in Nigeria. Ignorance about cervical cancer, cultural beliefs, economic constraints, and limited spousal support has been identified as major obstacles. Mutyaba et al. (2019) emphasised that societal issues, such as domestic gender power relations and unfriendly healthcare services, further discourage women from seeking preventive care. In addition, illiteracy and misconceptions about the relevance of screening services to healthy individuals exacerbate the issue. Many women perceive screening as unnecessary unless symptomatic, while others fear testing positive and avoid it altogether (Bukar et al., 2012; Onyenwenyi et al., 2018; Owoeye et al., 2013).

Financial barriers also play a significant role. The cost of screening services, coupled with the high poverty levels in the country, limits accessibility. Furthermore, the absence of a robust cervical cancer control program leaves screening efforts largely dependent on tertiary health institutions and urban-based initiatives, neglecting rural populations (Modibbo et al., 2016; Wright et al., 2014).

Current Screening Statistics

Nigeria's cervical screening coverage is alarmingly low, at only 8.7%. Studies from rural areas report even lower uptake. For example, in a community-based study in Edo State, none of the participants had undergone screening (Igwilu et al., 2012). Similarly, only 4.3% of respondents in a recent study had ever had a cervical screening test, and studies in Lagos and Oyo reported uptake rates of 5% (Wright et al., 2014). These figures contrast starkly with developed countries, where national screening programs ensure higher coverage. In the United Kingdom, 91% of women have undergone cervical cancer screening at least once (Moser et al., 2009), while countries like Pakistan report a 73% screening rate (Hirani et al., 2021). Kenyan study, uptake was 6%, (Sudenga et al., 2013), 3.3% in Ethiopia, (Chaka et al., 2018), 5% in Zimbabwe, (Fitzpatrick et al., 2020), 7% in Cambodia (Touch & Oh, 2018), 9% in the Democratic Republic of Congo (Ali-Risasi, et al., 2014), 15.5% in Cameroon (Nkfusai et al., 2019), 17% in Zimbabwe (Barrow et al., 2020), 0.8% in a community-based study in Elmina, Ghana (Ebu et al., 2015).

Comparative Awareness

Awareness levels vary across Nigeria, often influenced by geographic and educational disparities (Abugu & Nwagu, 2012; Okunade et al., 2019). The Low level of awareness and knowledge of cervical cancer and screening has been identified as one of the causes of cervical screening underutilization in Nigeria (Omowhara et al., 2022). In rural areas, awareness is significantly lower compared to urban populations. For instance, only 9.4% of women in a recent study were aware of cervical cancer screening methods, a stark contrast to the 50.6% awareness rate among students and staff at Niger Delta University (Owoeye & Ibrahim, 2013). Similarly, regional differences exist, with awareness rates reported at 18.2% in Ekiti (Emanuel et al., 2015), 23.2% (Ifediora & Azuike, 2018) in Southeast Nigeria, 40.7% in Oyo (Akanbi et al., 2015) and 68.1% in Enugu (Abugu & Nwagu, 2021). These variations underscore the importance of tailored interventions to address specific community needs.

Management Of Cervical Lesions In Hiv-Positive Women

It is possible to prevent and cure cervical cancer. This is because precancerous lesions often advance slowly through distinguishable and recognised phases before changing into invasive illness, and cervical cancer has a rather long lead time. Therefore, the disease can be considered treatable if discovered before it advances to an advanced state (Beyazit et al., 2018).

Excisional and Ablative Procedures

Management strategies are tailored to the severity of cervical lesions. Low-grade lesions may resolve spontaneously, particularly in women on HAART, while high-grade lesions require surgical intervention. Okafor recommend Loop electrosurgical excision procedure (LEEP) and cold knife conization are preferred for HSIL due to their efficacy in excising dysplastic tissue (Okafor et al., 2022). However, recurrence rates remain higher in WLHIV range from 25% - 35%, necessitating rigorous follow-up (Ojo & Akinyemi, 2020).

Role of Therapeutic Vaccines

Investigational HPV therapeutic vaccines targeting E6 and E7 proteins are under evaluation. Preliminary data suggest immunogenicity improvements, although long-term efficacy remains unclear (WHO, 2023; Nwosu et al., 2023).

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