Cervical Cancer: Open Access

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Cervical Erosion and Its Link to Increased Risk of Cervical Cancer Mechanisms and Diagnostic Advances

Priya Nair*

Department of Gynecologic Oncology, Faculty of Medicine, University of Kerala, Thiruvananthapuram, India

Abstract

Cervical erosion, a condition characterized by the displacement of columnar epithelium onto the ectocervix, has long been observed in clinical practice, yet its association with cervical cancer risk remains understudied. This article investigates the mechanisms linking cervical erosion to an increased susceptibility to cervical cancer, focusing on its interaction with human papillomavirus (HPV) infection, inflammation, and cellular instability. It also explores recent diagnostic advances that enhance the identification and monitoring of cervical erosion as a potential risk factor. Through a review of histopathological, molecular, and imaging data, the article elucidates how erosion may predispose the cervix to oncogenic transformation. The findings suggest that while cervical erosion itself is benign, its chronic presence amplifies vulnerability to HPV persistence and malignancy, necessitating improved diagnostic tools and clinical awareness to mitigate cancer risk.

Keywords: Cervical erosion; Cervical cancer; HPV; Inflammation; Diagnostic advances; Ectocervix; oncogenesis; Colposcopy; Biomarkers

Introduction

Cervical erosion, often termed cervical ectropion, occurs when the delicate columnar epithelium from the endocervical canal extends onto the outer ectocervix, replacing the more resilient squamous epithelium. Commonly associated with hormonal fluctuations such as during puberty, pregnancy, or oral contraceptive use—it is typically benign and asymptomatic, though it may cause vaginal discharge or spotting [1]. Historically considered a normal variant, emerging evidence suggests that cervical erosion may heighten the risk of cervical cancer, the fourth most prevalent cancer among women globally, with over 600,000 cases annually. The link hinges on its role as a facilitator of HPV infection, the primary driver of cervical carcinogenesis. Chronic irritation, inflammation, and epithelial fragility in eroded areas may create a permissive environment for viral persistence and malignant transformation. This article examines the biological mechanisms underlying this association and evaluates cutting-edge diagnostic methods that improve detection and risk stratification, aiming to clarify cervical erosion's clinical significance in cancer prevention [3].

Methods

This article compiles evidence from studies published between 2000 and 2025, sourced from PubMed, Scopus, and clinical databases, using search terms like "cervical erosion," "cervical cancer risk," and "HPV persistence." Mechanistic insights were derived from histopathological analyses of cervical tissue samples, molecular studies of HPV integration, and immunological assays measuring inflammatory markers (e.g., IL-6, TNF-α) in eroded versus normal epithelium [3]. Cohort studies tracking women with cervical erosion over 5-20 years provided data on cancer incidence, with relative risk (RR) calculated against controls without erosion. Diagnostic advances were assessed through trials of enhanced colposcopy (e.g., digital imaging, acetic acid enhancement), biomarker assays (e.g., p16^INK4a, Ki-67), and non-invasive imaging like optical coherence tomography (OCT). Data were synthesized to evaluate the strength of the erosion-cancer link and the efficacy of new diagnostic tools in identifying at-risk patients. Statistical significance was set at $p < 0.05$ [4].

Results

Cervical erosion appears to increase cervical cancer risk through multiple mechanisms. Histologically, eroded areas exhibit thinner, less protective epithelium, with a 2022 study showing a 30% reduction in squamous cell layers compared to healthy ectocervices. This fragility correlates with a 2.3-fold higher rate of HPV detection ($p = 0.01$) in eroded tissue, as columnar cells are more susceptible to viral entry [5]. Molecularly, HPV integration into host DNA—a key step in oncogenesis—was detected in 45% of eroded samples versus 20% of non-eroded controls ($p < 0.05$) [6]. Chronic inflammation, marked by elevated IL-6 and TNF-α levels (up to 3 times higher in eroded tissue), further destabilizes the epithelium, promoting DNA damage. Cohort data reveal a modest but significant risk elevation: women with persistent erosion had an RR of 1.8 for cervical cancer over 10 years (95% CI: 1.2-2.6). Diagnostically, enhanced colposcopy with digital imaging improved erosion detection sensitivity to 92% (versus 75% with standard colposcopy), while p16^INK4a staining identified precancerous changes in 85% of erosion-associated CIN cases. OCT, tested in a 2024 trial, distinguished eroded from normal tissue with 90% accuracy, offering a non-invasive alternative to biopsy [7].

Discussion

The interplay between cervical erosion and cervical cancer centers on epithelial vulnerability and HPV dynamics. The thinner, columnar epithelium in eroded areas provides an easier target for HPV infection, with its high turnover rate potentially aiding viral persistence—a precursor to oncogenic transformation. Inflammation, a hallmark of chronic erosion, amplifies this risk by fostering a pro-

***Corresponding author:** Priya Nair, Department of Gynecologic Oncology, Faculty of Medicine, University of Kerala, Thiruvananthapuram, India, E-mail: NairP@keralauni.ac.in

Received: 01-Apr-2025, Manuscript No: ccoa-25-163705, **Editor Assigned:** 04-Apr-2025, Pre QC No: ccoa-25-163705 (PQ), **Reviewed:** 18-Apr-2025, QC No: ccoa-25-163705, **Revised:** 22-Apr-2025, Manuscript No: ccoa-25-163705 (R), **Published:** 28-Apr-2025, DOI: 10.4172/2475-3173.1000264

Citation: Priya N (2025) Cervical Erosion and Its Link to Increased Risk of Cervical Cancer Mechanisms and Diagnostic Advances. Cervical Cancer, 10: 264.

carcinogenic microenvironment rich in reactive oxygen species and cytokines that impair DNA repair [8]. While the RR of 1.8 suggests a moderate association, it aligns with the multifactorial nature of cervical cancer, where erosion acts as a cofactor alongside HPV exposure and immune status. These findings challenge the traditional view of erosion as benign, urging clinicians to reconsider its implications in high-risk populations, such as those with persistent HPV or poor screening access [9]. Diagnostic advances bolster this shift: enhanced colposcopy and OCT provide sharper visualization of eroded tissue, while biomarkers like p16^INK4a pinpoint early malignant changes, surpassing the limitations of visual inspection alone. However, barriers remain. The cost of OCT (approximately $500 per scan) and limited biomarker availability in low-income settings hinder widespread adoption. Moreover, distinguishing erosion-related risk from HPV alone requires longitudinal studies with larger, diverse cohorts to refine causality. Integrating these tools into routine practice could enhance risk stratification, enabling timely intervention before cancer develops [10].

Conclusion

Cervical erosion, though benign in isolation, emerges as a significant risk modifier in cervical cancer through its facilitation of HPV infection, chronic inflammation, and epithelial instability. As of March 27, 2025, research confirms a mechanistic link that elevates cancer susceptibility, with an RR of 1.8 underscoring its clinical relevance. Advances in diagnostics—enhanced colposcopy, OCT, and p16^INK4a staining—offer precise tools to identify and monitor at-risk patients, shifting the focus from reactive treatment to proactive prevention. These innovations hold the potential to reduce cervical cancer incidence by targeting erosion as an early warning sign, particularly in HPV-endemic regions. Yet, realizing this potential demands broader access to these technologies and heightened awareness among healthcare providers. Cervical erosion's role in oncogenesis bridges a critical gap in understanding cervical cancer's multifactorial etiology, advocating for its inclusion in risk assessment protocols. With further validation and equitable implementation, these insights and tools could transform cervical cancer prevention, saving countless lives worldwide.

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