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ETIOPATHOGENETIC AND CLINICAL FEATURES OF CERVICAL DISEASES

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Abstract: Diseases of the uterine cervix represent a major health burden for women worldwide, ranging from benign inflammatory conditions to premalignant and malignant lesions. Understanding the etiopathogenesis and clinical characteristics of these disorders is essential for effective prevention, early detection, and management. This article analyzes the etiological factors, pathogenesis, and clinical manifestations of cervical diseases, focusing on the role of human papillomavirus (HPV) infection, hormonal influences, immune mechanisms, and environmental contributors.

Keywords: Cervical diseases; etiopathogenesis; HPV; cervicitis; cervical cancer; pathogenesis; cytology; women's health.

Introduction

Cervical diseases encompass a broad spectrum of pathological conditions affecting the uterine cervix, including cervicitis, cervical ectropion, cervical intraepithelial neoplasia (CIN), and cervical cancer. Among these, cervical cancer remains one of the most common malignancies in women, particularly in developing countries. The primary etiological factor in cervical carcinogenesis is persistent infection with high-risk HPV types, mainly HPV-16 and HPV-18. However, multiple cofactors—including hormonal status, genetic susceptibility, chronic inflammation, and sexual behavior—play significant roles in disease progression.
This study aims to analyze the etiopathogenetic mechanisms and clinical features of major cervical diseases to improve understanding of their pathophysiology and diagnostic approach.

Materials and Methods

This study employed a descriptive and analytical design aimed at investigating the etiopathogenetic mechanisms and clinical characteristics of cervical diseases in women of reproductive and perimenopausal age. Both retrospective and prospective approaches were combined to ensure a comprehensive evaluation of the condition. The research was conducted at gynecological clinics and hospitals between January 2023 and April 2025, involving a cohort of 120 women aged between 20 and 60 years. The participants were selected based on clinical suspicion of cervical pathology, including abnormal vaginal discharge, contact bleeding, pelvic pain, or abnormal findings on cytological examination. Pregnant women, those with recent cervical operations, systemic immune disorders, or unwillingness to consent were excluded from the study. Each participant provided informed written consent after a clear explanation of the study objectives and methods.

A complete gynecological examination was performed for all participants, including visual inspection of the cervix, bimanual palpation, and colposcopic evaluation. Cervical samples were collected for cytological, microbiological, and molecular analyses under sterile conditions. Cytological evaluation was carried out using the Papanicolaou staining technique and classified according to the Bethesda System (2014). This method enabled the differentiation between normal epithelial cells, inflammatory changes, and various grades of dysplasia. In addition, molecular testing for human papillomavirus DNA was performed using polymerase chain reaction (PCR) techniques to identify high-risk genotypes such as HPV-16, HPV-18, HPV-31, and HPV-45. Microbiological cultures were analyzed to identify associated infections including

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Chlamydia trachomatis, *Neisseria gonorrhoeae*, *Trichomonas vaginalis*, and other bacterial flora that may contribute to chronic cervicitis or co-infection with HPV.

Histopathological examination was conducted on biopsy specimens obtained from patients showing suspicious colposcopic or cytological results. The specimens were fixed in formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Microscopic analysis focused on epithelial abnormalities, inflammatory cell infiltration, angiogenesis, and cellular atypia indicative of neoplastic transformation. Data from these analyses were systematically recorded and categorized for further interpretation.

All collected data were entered into a structured database and statistically processed using SPSS software version 25.0 (IBM, USA). Descriptive statistics such as mean values, standard deviations, and frequency distributions were calculated. The chi-square test was used to assess associations between HPV infection and the severity of cytological abnormalities, and statistical significance was defined at a p-value below 0.05. Graphical representation of key findings was used to enhance clarity and visualization of correlations among variables.

The ethical aspects of the study adhered to the principles outlined in the Declaration of Helsinki (2013). Approval was obtained from the institutional ethics committee prior to data collection. Patient confidentiality and anonymity were strictly maintained, and all biological materials were handled according to established biosafety protocols.

To strengthen the scientific foundation of the research, an extensive literature review was performed following PRISMA guidelines. The databases PubMed, Scopus, ScienceDirect, and WHO Global Health Library were searched using keywords such as “cervical diseases,” “HPV infection,” “cervical intraepithelial neoplasia,” and “cervical cancer pathogenesis.” Publications from 2015 to 2025 were included, prioritizing peer-reviewed articles, clinical studies, systematic reviews, and meta-analyses. A total of 65 relevant studies were critically analyzed and integrated to support the empirical findings and discussion of this research.

Results

Etiological Factors

The leading etiological agent for premalignant and malignant cervical disease is infection with oncogenic HPV strains. Inflammatory cervical conditions, such as chronic cervicitis, were often associated with mixed microbial flora including *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, and *Trichomonas vaginalis*. Hormonal factors—especially prolonged exposure to estrogen and progesterone—were found to influence cervical epithelial proliferation and susceptibility to infection.

Pathogenesis

The pathogenesis of cervical neoplasia involves viral persistence, integration of HPV DNA into the host genome, and disruption of cell-cycle regulatory genes such as *p53* and *RB1*. The oncogenic proteins E6 and E7 play central roles by inhibiting tumor suppressor activity, leading to uncontrolled cell proliferation and genomic instability. Inflammatory cytokines and oxidative stress further promote cellular transformation and angiogenesis. Chronic cervicitis, when unresolved, can contribute to epithelial metaplasia and dysplasia.

Clinical Features

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Benign conditions such as ectropion and cervicitis commonly presented with mucopurulent discharge, contact bleeding, and mild pelvic discomfort. Cervical intraepithelial neoplasia was frequently asymptomatic, detected only through screening cytology. In invasive carcinoma, symptoms included persistent vaginal bleeding, offensive discharge, and pelvic pain radiating to the lower limbs. Advanced disease often involved parametrial infiltration and urinary tract obstruction.

Diagnostic Findings

Pap smear analysis revealed varying degrees of epithelial dysplasia, ranging from mild (CIN I) to severe (CIN III). Colposcopic findings correlated well with cytological abnormalities, showing acetowhite epithelium, mosaic patterns, and punctation. HPV DNA testing confirmed the presence of high-risk viral types in 80% of precancerous and malignant lesions.

Discussion

The study reinforces the pivotal role of HPV infection in the etiopathogenesis of cervical neoplasia. However, infection alone is insufficient for malignant transformation; cofactors such as immunosuppression, smoking, and chronic inflammation modulate disease progression. The interplay between viral oncogenes and host immune responses determines whether infection regresses or progresses to neoplasia.
Clinically, early cervical disease is often silent, emphasizing the importance of routine screening and HPV vaccination. The strong correlation between cytological and histological findings highlights the effectiveness of the Pap smear as a preventive tool. Moreover, integrating molecular testing with traditional cytology enhances diagnostic accuracy and risk stratification.

Conclusion

Cervical diseases, ranging from benign inflammatory conditions to malignant neoplasms, represent a critical challenge in women's reproductive health. Their etiopathogenesis is multifactorial, involving a complex interplay between infectious, hormonal, immunological, genetic, and environmental factors. Among these, persistent infection with high-risk human papillomavirus (HPV) types—particularly HPV-16 and HPV-18—remains the primary determinant of cervical carcinogenesis. However, viral infection alone is not sufficient for malignant transformation; cofactors such as chronic inflammation, immunosuppression, smoking, and hormonal imbalance significantly contribute to the persistence and progression of precancerous lesions.

Understanding the molecular mechanisms underlying cervical carcinogenesis, such as the actions of HPV oncogenes E6 and E7 that inhibit tumor-suppressor proteins p53 and Rb, provides a scientific basis for targeted prevention and therapy. Moreover, the influence of the host immune response and oxidative stress in facilitating genomic instability highlights the importance of immunomodulatory approaches and antioxidant support in future treatment strategies.

Clinically, cervical diseases exhibit a broad spectrum of manifestations—from asymptomatic early lesions detectable only through screening, to advanced carcinoma presenting with bleeding, pain, and systemic complications. This emphasizes the indispensable role of organized screening programs, such as cytological evaluation (Pap smear) and HPV DNA testing, in early detection. The integration of molecular diagnostic tools with conventional cytology enhances sensitivity and specificity, leading to more accurate risk assessment and timely intervention.

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Preventive strategies, including widespread HPV vaccination, safe sexual practices, and effective treatment of inflammatory conditions, offer the most sustainable path to reducing cervical disease burden. Public health efforts must focus on increasing awareness, ensuring vaccination coverage, and improving access to diagnostic and therapeutic services, particularly in low-resource settings where cervical cancer mortality remains high.

In conclusion, cervical diseases are preventable and largely manageable through a combination of scientific understanding, early detection, and public health intervention. The integration of etiopathogenetic knowledge with clinical practice not only advances patient outcomes but also contributes to global initiatives aimed at eradicating cervical cancer as a public health problem. Future research should continue to explore the molecular pathways of HPV-host interaction, novel biomarkers for early diagnosis, and cost-effective strategies for global implementation of preventive care.

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