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Risk-Based Diagnosis and Modern Triage Strategies for Cervical Pathology in HPV-Positive Women

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Abstrak. Cervical pathology in HPV-positive women remains one of the most significant challenges in modern gynecology and gynecologic oncology. This is due not only to the high prevalence of human papillomavirus infection, but also to the fact that this group represents a major area of clinical uncertainty: some patients experience transient infection without significant morphological consequences, while others develop clinically significant lesions requiring timely diagnosis and active intervention. In recent years, universal screening and routing schemes have increasingly given way to risk-based and personalized management models. The aim of this review article was to analyze current literature data on the diagnosis of cervical pathology in HPV-positive women, focusing on the clinical heterogeneity of this group, the role of HPV DNA testing, cytology, colposcopy, targeted biopsy, and endocervical curettage, as well as the significance of new biomarkers and triage strategies. The article examines current understanding of the natural history of HPV infection, the differences between transient and persistent infection, the significance of individual highly oncogenic genotypes, and the role of age, immune status, the transformation zone, and reproductive cofactors. The article discusses in detail current approaches to HPV-based screening, the role of cytology as a triage method, the limitations of visual assessment in TZ3, and the importance of expanded genotyping, p16/Ki-67 dual stain, self-sampling, DNA methylation, E6/E7 mRNA, and oncoprotein tests as tools for more accurate risk stratification. It is concluded that modern diagnostics of cervical pathology in HPV-positive women should be built as a multi-level and personalized system, in which clinical decisions are made not based on a single test, but on a comprehensive assessment of viral, morphological, anatomical, and molecular parameters.

Keywords: human papillomavirus; HPV; cervical pathology; HPV DNA; risk stratification; cytology; colposcopy; endocervical curettage; p16/Ki-67; genotyping; self-sampling; DNA methylation.

1. Introduction

Cervical pathology in HPV-positive women currently represents one of the most significant challenges in modern gynecology, gynecologic oncology, and reproductive medicine. This is due not only to the widespread prevalence of human papillomavirus infection but also to its decisive role in the development of cervical intraepithelial neoplasia and invasive cervical cancer. According to the World Health Organization and GLOBOCAN, cervical cancer remains one of the most significant oncological diseases in women, with the main burden of the disease still occurring in low- and middle-income countries, where vaccination coverage, organized screening, and timely treatment of precancerous lesions remain limited [1–4, 7]. This problem is particularly relevant because cervical cancer is one of the few malignant tumors that is largely preventable with the right combination of vaccination, screening, triage, and treatment of precancerous lesions [1, 3, 4]. This is why the HPV-positive population is of not only clinical but also medical and social significance: it is in this group that the decision is made whether a viral infection will become a short-term blip or the beginning of a carcinogenic trajectory [1, 4, 7].

Modern understanding of the problem has long since moved beyond the simplified "HPV detected = high cancer risk" model. Almost all cases of cervical cancer are associated with oncogenic HPV types; however, the decisive condition for carcinogenesis is not the infection itself, but the persistence of a highly oncogenic infection followed by virus-induced epithelial transformation [1, 3, 7]. Most HPV infections in immunocompetent women are transient, while only a minority of cases progress to long-term carriage with the risk of developing CIN2+, CIN3+, and invasive cancer [3, 7, 14]. Therefore, a positive HPV test is not equivalent to severe pathology, and the clinical significance of the result is determined by the persistence time, viral genotype, cytological profile, colposcopic image, the state of the transformation zone, and individual patient characteristics [7, 11, 14].

Against this backdrop, a revision of screening approaches is also warranted. While cytology was recently considered the foundation of early detection programs, international recommendations are increasingly prioritizing HPV DNA testing as the primary screening method [1, 4, 7]. However, increasing the sensitivity of primary screening inevitably creates a new clinical challenge: how to identify, among the numerous HPV-positive women, those whose risk of CIN2+/CIN3+ is truly significant and requires immediate invasive measures, and how to separate them from patients for whom more gentle observation is warranted [4, 5, 7, 11].

This is precisely why triage occupies a central place in the modern diagnostic model. While a primary HPV test answers the question of the presence of viral risk, triage should answer a more important clinical question: how high is the probability of an existing or imminent clinically significant lesion [5, 8–11]. New tools have entered this field, significantly improving diagnostic accuracy: partial and extended genotyping, p16/Ki-67 dual stain, self-sampling, and a number of molecular biomarkers based on DNA methylation and viral activity assessment [5, 8–11, 25–32].

Thus, modern diagnostics of cervical pathology in HPV-positive women should be considered not as a sequence of individual tests, but as a multi-level system that combines virological sensitivity, morphological verification, anatomical completeness of examination, and molecular risk stratification. The aim of this review article is to analyze current literature data on the clinical heterogeneity of the HPV-positive population, modern approaches to diagnosing cervical pathology, and new triage strategies.

HPV-Positive Women as a Heterogeneous Clinical Risk Group

HPV-positive women cannot be considered a uniform clinical category, since the mere detection of DNA or RNA of a highly oncogenic virus does not necessarily determine the presence of a morphologically significant lesion or the likelihood of its progression. The current understanding of the natural history of HPV infection has become significantly more complex than the previous model, which interpreted viral detection as primary infection and the disappearance of the signal as complete elimination of the infection. It has now been shown that a new positive result can reflect both recent infection and autoinoculation, transient deposition of viral material after sexual contact, or the recurrence of a previously acquired latent infection.

Accordingly, the absence of viral detection does not always mean its complete disappearance; in many cases, it is more accurate to speak of immunological control of the infection than of absolute virological "sterility" [3, 14, 15]. Distinguishing between transient and persistent infection is crucial. According to the WHO, the immune system controls HPV infection in most infected individuals within 1–2 years, and only a minority of cases persist long enough to develop precancerous lesions [3]. The long-term persistence of highly oncogenic HPV types is considered a central factor in cervical carcinogenesis [3, 7, 14, 17]. In practice, this means that the same "hrHPV positive" result in two patients may conceal diametrically opposed scenarios: from a short-term, clinically insignificant infection to a biologically active process requiring in-depth triage and morphological verification [7, 11, 14].

Heterogeneity of risk is most clearly manifested at the level of genotype-specific oncogenicity. A global systematic analysis has shown that HPV16 makes the greatest contribution to the development of invasive cervical cancer, followed by HPV18, and then HPV45, HPV33, HPV58, HPV31, and HPV52 [6]. This has direct clinical significance. HPV16 remains the most aggressive genotype, closely associated with severe squamous cell lesions. HPV18 and HPV45 are of particular importance in glandular lesions, while HPV33, HPV31, HPV52, and HPV58 are among the genotypes whose carcinogenic potential is also clinically significant and should not be underestimated [6, 11, 16]. Therefore, already at the stage of interpreting the HPV test result, the physician should think not in terms of “there is/is not infection”, but in terms of “what exactly is the risk posed by a given genotype in a given clinical situation” [6, 11].

Age is a significant modifier of the natural course of infection. In young women, HPV infection is often transient, whereas the likelihood of long-term persistence increases with age, which is associated with immune senescence, hormonal changes, and changes in the cervical microenvironment [14, 17]. However, age is not an autonomous surrogate for progression; its influence is realized through associated mechanisms, including the type of transformation zone, immune changes, and the accumulation of associated risk factors [13, 14, 17].

Immune status occupies a special place. The WHO emphasizes that HPV prevalence is higher in individuals with immunodeficiency states and coinfections [3]. The most clinically significant example is HIV infection: women living with HIV have a significantly higher risk of developing cervical cancer compared to women without HIV [3]. This confirms the fundamental idea: the outcome of HPV positivity is determined not only by the properties of the virus but also by the body's ability to limit its persistence in the cervical epithelium.

Another critically important factor in risk heterogeneity is the state of the transformation zone. The transformation zone is the primary anatomical and biological substrate for the development of cervical neoplasia, but its availability for visual assessment changes with age. In some patients, especially postmenopausal women, TZ3 develops, in which the squamocolumnar junction shifts into the endocervical canal, significantly limiting the capabilities of colposcopy and increasing the risk of underestimating occult lesions [12, 13, 19–24]. This means that the same HPV positivity in a young woman with a fully visible transformation zone and in a patient with TZ3 carries fundamentally different diagnostic implications. Thus, HPV-positive women do not represent a homogeneous population, but a multi-level clinical group within which risk varies significantly depending on the nature of virus detection, its persistence, genotype, age, immune status, the state of the transformation zone, and associated reproductive factors [3, 6, 11, 13–18]. Recognition of this heterogeneity underlies the modern risk-stratified diagnostic model.

Modern approaches to the diagnosis of cervical pathology in HPV-positive women

Modern diagnostics of cervical pathology in HPV-positive women is undergoing a fundamental rethink. While cytology was previously considered the basis for detecting cervical precancerous changes, the HPV-focused screening model is now increasingly taking center stage. The primary goal is not simply to detect cellular atypia, but to identify women at real risk of persistent carcinogenic infection and subsequent neoplastic progression [1, 4, 7].

HPV DNA testing is currently considered the preferred primary screening method for women, as it best identifies those at risk for developing CIN2+, CIN3+, and cervical cancer [1, 4,

7]. According to WHO recommendations, highly effective HPV testing should be used for the general female population starting at age 30, and for women living with HIV starting at age 25, with more frequent monitoring [1, 4]. This shift is explained by the higher sensitivity of HPV testing compared to cytology alone in identifying risk groups for precancerous lesions and cancer [4, 7]. However, increased sensitivity of primary screening inevitably means an expansion of the group of HPV-positive women, within which it is necessary to more accurately identify those who truly require immediate colposcopy and morphological verification [4, 5, 7, 11].

This is where cytology remains important, but in a different role. In the modern diagnostic model, it increasingly acts not as a universal primary filter, but as a triage tool within the HPV-positive cohort [4, 5, 7, 11]. Its purpose is to clarify the probability of an existing high-grade lesion and help the physician determine the intensity of further testing. Thus, modern cytology is no longer the foundation of the entire screening program, but a contextual morphological risk modifier. Colposcopy occupies a special place in the diagnosis of HPV-positive women. It remains the primary method for visually localizing suspicious areas and selecting a site for targeted biopsy; however, today its role is understood more critically and accurately [19, 20]. Colposcopy is not a standalone "gold standard," but rather an operator-dependent step within a comprehensive algorithm, the quality of which is determined by the completeness of visualization, the type of transformation zone, and the adequacy of biopsy sampling. Therefore, a modern colposcopic approach requires not limiting itself to a single "most suspicious" point, but rather to a systemic assessment of the cervix and, in the presence of several abnormal areas, performing multiple targeted biopsies [19, 20].

Targeted biopsy remains the decisive step in confirming the nature and extent of cervical lesions. In the era of HPV testing and molecular biomarkers, histological verification has not lost its importance. On the contrary, it now increasingly influences the choice between observation, repeat testing, colposcopic follow-up, and active intervention. The practical value of targeted biopsy is particularly increased in HPV-positive patients with discrepancies between virological results and visualization, as morphology allows us to distinguish transient virus carriage and mild regressive changes from a true transforming process [12, 19–24].

The most challenging diagnostic area remains the group of patients with TZ3, in whom the squamocolumnar junction is partially or completely displaced into the endocervical canal and becomes inaccessible to full visualization [12, 13, 19–24]. This is where the risk of missing a significant lesion increases. In these clinical situations, endocervical curettage should not be considered an optional adjunct to colposcopy, but rather a targeted tool for addressing its weaknesses. Current data demonstrate that ECC is particularly useful in patients with incomplete visualization, TZ3, AGC, persistent hrHPV, and a discrepancy between high clinical suspicion and a "reasonable" colposcopic picture [12, 13, 19–24]. This is particularly important for addressing one of the most dangerous diagnostic deficiencies—the occult endocervical omission of HSIL, AIS, or an early invasive process.

Therefore, modern diagnostics of cervical pathology in HPV-positive women should not rely on a single "best" test, but on a combination of sensitivity, anatomical completeness of examination, and morphological accuracy. HPV DNA testing determines the presence of viral risk, cytology and biomarkers clarify its clinical significance, colposcopy localizes the suspicious lesion, biopsy confirms the nature of the lesion, and ECC fills the diagnostic gap in endocervical localization [4, 5, 7–13, 19–24].

Biomarkers and New Triage Strategies

The current stage of development of cervical screening increasingly demonstrates that the problem of HPV-positive women cannot be solved by simply repeating traditional cytology. The primary HPV test is highly sensitive, but for this very reason it leaves behind a broad and clinically heterogeneous group of patients, among whom it is necessary to distinguish transient infection from a biologically significant transforming process. This is where new triage strategies, based not only on morphology but also on molecular risk markers, become crucial [5, 8–11, 25–32]. Partial and Expanded Genotyping

One of the most significant advances in recent years has been the expanded role of HPV genotyping. While the classical model largely focused on identifying HPV16 and HPV18 as the highest-risk genotypes, current guidelines utilize expanded genotyping for more precise stratification of the probability of CIN2+/CIN3+ [11]. This represents a fundamental shift away from the crude "all non-16/18 are the same" model and a transition to a clinical model in which each set of genotypes is interpreted as a distinct risk profile. This approach not only allows for earlier referral of truly high-risk patients for colposcopy but also reduces the number of unnecessary invasive procedures in women with less aggressive genotypic combinations [11, 25].

The p16/Ki-67 dual stain has taken center stage among new triage biomarkers. Its value lies in the fact that it reflects not the mere fact of exposure to the virus, but rather the onset of cellular dysregulation: coexpression of p16 and Ki-67 in a single cell indicates a disruption in cell cycle control, characteristic of transforming HPV infection [8, 9, 25]. The key value of dual stain is that it helps bring triage closer to the biology of the process and reduces the dependence of clinical decisions on subjective cytological interpretation alone.

It is especially important that dual stain has already moved beyond the experimental stage and is considered a clinically acceptable tool for triaging HPV-positive women [5, 8, 9, 25]. Its importance lies not only in increasing sensitivity, but also in the fact that a negative result allows for a safer approach to avoiding immediate colposcopy in some patients, while a positive result strengthens the rationale for active follow-up testing.

Self-sampling

The next major trend is self-sampling, or self-collection of vaginal specimens for HPV testing. Its significance extends far beyond technical convenience: self-sampling has become an important tool for expanding the reach of women who, for various reasons, do not participate in traditional screening—due to shame, access difficulties, cultural barriers, or reluctance to undergo speculum examination [10]. This makes self-sampling not just an alternative method of collection, but an important organizational tool for population-based cancer prevention.

It is important to understand that self-sampling expands program participation but does not eliminate the need for subsequent high-quality triage. This is why its clinical value is greatest when combined with a clearly defined algorithm for further actions after a positive HPV result [10]. This is the modern meaning of self-sampling: not to replace the entire diagnostic pathway, but to increase the number of women who participate in this pathway.

DNA Methylation and Other Molecular Markers

DNA methylation remains the most intriguing and, perhaps, the most promising direction for the coming years. Unlike cytology, which evaluates the morphological result, and unlike HPV DNA, which reflects the presence of the virus, methylation allows us to detect epigenetic events associated with the transition from a regressing lesion to true neoplastic transformation [26–30]. This is why methylation is increasingly being viewed as a path to objective molecular triage that can be automated, standardized, and potentially applicable even to self-collected material. Along with methylation, promising molecular markers include tests for E6/E7 mRNA and E6/E7 oncoproteins. Their clinical logic is similar to dual stains: they seek to reflect not just the presence of the virus, but its biological activity [31, 32]. This is particularly important for distinguishing between transient DNA positivity and transcriptionally active infection, which is truly associated with the risk of neoplastic progression.

Thus, modern triage of HPV-positive women is moving from a morphologically oriented model to a multi-level, molecular, risk-oriented system. Partial and expanded genotyping allow for more precise control of the scope of subsequent steps, dual stains bring triage closer to the biology of transforming infection, self-sampling expands screening coverage, and methylation and viral molecular markers create the basis for the next stage of evolution—one that is more objective, automated, and less dependent on subjective interpretation [8–11, 25–32].

2. Discussion

The analysis shows that the diagnosis of cervical pathology in HPV-positive women can no longer be based on a simplified model where a positive HPV test is automatically interpreted as a single risk clinical scenario. Current data convincingly demonstrate that this population is heterogeneous, and the actual risk is determined not only by the fact of virus carriage but also by the HPV genotype, its persistence pattern, age, immune status, the state of the transformation zone, and cytological and colposcopic findings.

Modern diagnostics have also ceased to be a system based on a single leading test. The primary focus has shifted toward HPV-based screening, while cytology is increasingly used as a triage method within the HPV-positive cohort. Risk stratification capabilities have been significantly enhanced through expanded genotyping, p16/Ki-67 dual stain, self-sampling, and molecular biomarkers. At the same time, none of these methods should be considered in isolation: their greatest clinical value comes from a judicious combination with colposcopy, targeted biopsy, and, if necessary, endocervical curettage, especially in patients with TZ3 and limited lesion visualization.

This is why a modern diagnostic model for HPV-positive women must be multi-layered, risk-based, and personalized. Its goal is not simply to detect the virus, but to accurately identify clinically significant lesions while simultaneously reducing overdiagnosis and unnecessary invasive interventions.

3. Conclusions

1. HPV-positive women do not represent a homogeneous clinical group.

The actual risk is determined not only by the detection of hrHPV but also by the viral genotype, its persistence, the patient's age, immune status, the state of the transformation zone, and the morphological context.

2. Primary diagnosis of cervical pathology in HPV-positive women should be based on an HPV-based approach.

High-performance HPV DNA testing is currently considered the preferred primary screening tool, while cytology is increasingly used as a triage method within the HPV-positive cohort.

3. Colposcopy and morphological verification remain essential diagnostic steps.

Targeted biopsy and endocervical curettage are especially important in patients with TZ3, incomplete visualization, and suspected endocervical lesions, where there is a risk of occult clinically significant lesions.

4. The most promising triage strategy is a combination of genotyping and biomarkers of transforming infection.

Partial and expanded genotyping, p16/Ki-67 dual stain, self-sampling, and molecular markers allow for more accurate risk stratification and reduce the number of unnecessary colposcopies and invasive procedures.

5. The future of HPV-positive women's diagnosis is associated with a transition to a multi-level, personalized model.

Modern clinical decisions should be based not on a single test, but on a combination of viral, morphological, anatomical, and molecular data, which simultaneously improves the accuracy of detecting clinically significant lesions and limits overdiagnosis.

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