Egyptian Journal of Medical Microbiology Volume 34 / No.4 / October 2025 491-498 Online ISSN: 2537-0979

ORIGINAL ARTICLE

Diagnostic Accuracy of Rapid HPV Antigen Testing Versus PCR in Cervical Cancer Patients

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Key words:
Human papillomavirus (HPV); cervical cancer; high-risk HPV (hr-HPV); and Rapid HPV Antigen Test.

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ABSTRACT

Background: *Human papillomavirus (HPV) is the most common sexually transmitted infection. Cervical cancer is primarily caused by high-risk HPV (hr-HPV).* ***Objective****: This study aims to evaluate the diagnostic performance of a rapid HPV antigen screening test as a potential tool for improving early detection and reducing the burden of cervical cancer.* ***Methodology:*** *The case-control study; biological samples, including cancerous tissues, wart samples, and control specimens, were collected for molecular analysis. Genomic DNA was extracted from the collected samples. The extracted DNA was subjected to Polymerase Chain Reaction (PCR) using specific primers designed to amplify target DNA sequences of HPV.* ***Results:*** *Overall, HPV genotype-specific assays identified HPV-16 in* ***23%****, HPV-51 in* ***17%****, and HPV-35 in* ***12%*** *of samples whereas, 67% identified by the HPV rapid antigen test. The rapid HPV test showed* ***perfect sensitivity (100%)*** *across all PCR targets. However, specificity varied considerably, ranging from* ***37.7% (HPV-35)*** *to* ***51.3% (MY09/11).*** *Cohen's kappa (κ) values further quantified agreement between the rapid test and PCR results, revealing* ***moderate agreement*** *for broad-spectrum MY09/11 detection (κ = 0.42, 95% CI: 0.25–0.42) but only* ***slight-to-fair agreement*** *for genotype-specific targets (HPV-16: κ = 0.26; HPV-51: κ = 0.18; HPV-35: κ = 0.12). The test exhibited* ***perfect negative predictive value (NPV = 100%)*** *across all targets however, was suboptimal, with only* ***52.5% of rapid test-positive samples confirmed as MY09/11 PCR-positive****, decreasing to* ***17.5% for HPV-35. Conclusion:*** *Although the test excels as a screening tool for excluding HPV, its limited specificity and PPV necessitate confirmatory PCR in clinical decision-making.*

INTRODUCTION

Human papillomavirus (HPV) is the most common sexually transmitted infection (STI) globally and is classified as a carcinogenic infectious agent by the International Agency for Research on Cancer (IARC). Nearly all sexually active individuals will contract HPV at least once in their lifetime, with most infections remaining asymptomatic and resolving spontaneously without progressing to cancerous diseases. However, certain oncogenic HPV strains, particularly high-risk types, are strongly associated with the development of malignancies, including cervical cancer and some head and neck cancers. As such, HPV testing plays a critical role in clinical practice, enabling accurate diagnosis, patient-centered treatment, and prognostication [1].

Despite the availability of cervical cytologic testing (Pap test) for over five decades, cervical cancer remains the second most common cancer among women worldwide. In high-income countries, the introduction of Pap testing initially led to a significant decline in cervical cancer incidence. However, this decline has plateaued in recent years, partly due to the low sensitivity of the Pap test, which necessitates frequent retesting to achieve acceptable diagnostic accuracy[1]. Cervical cancer is the major cause of cancer-related mortality among women living with HIV and is the fourth most frequent malignancy in women worldwide. Alarmingly, over 85% of the 311,000 annual deaths from cervical cancer occur in low- and middle-income countries (LMICs), where limited access to screening, lack of awareness, and insufficient healthcare infrastructure contribute to low screening rates [2]. Cervical cancer is a preventable disease, and its rising incidence has prompted global health initiatives to target its reduction. The World Health Organization (WHO) has identified high-risk HPV (hr-HPV) as the primary causative agent of cervical cancer, with 14 oncogenic types (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, and 68) responsible for the majority of pre-cancerous and cancerous lesions. Achieving WHO's 2030 target for cervical cancer elimination will require the widespread adoption of innovative and accessible screening technologies, particularly in LMICs where the burden of disease is highest [2].

Around 569,000 new cases of cervical cancer were diagnosed globally in 2018, and the disease was responsible for about 311,000 deaths. Between 84 and 90 percent of these took place in LMICs, which include

China, Brazil, India, and South Africa.[3]. Every year in Iraq, there are roughly 159 cervical cancer deaths and 244 new instances of the disease. Additionally, most cervical cancer patients are diagnosed in advanced stages of the disease due to improper screening program implementation, which has a substantial impact on how they respond to therapy and how the disease progresses.[4]. Approximately 13.8 million Iraqi women aged 15 years and older are at risk of developing cervical cancer. Although data on HPV prevalence in Iraq are limited, studies in Western Asia estimate that about 2.5% of women in the general population harbor cervical HPV-16/18 infection at any given time, with 72.4% of invasive cervical cancers attributed to these strains. According to a study done on Iraqi women between the ages of 15 and 50 who visited health Centres, the prevalence of HPV was 17.96%, with the highest frequency seen in those between the ages of 30 and 34. Interestingly, 30% of HPV-positive women had cervical cytological abnormalities, and the majority of these women were housewives from lower socioeconomic backgrounds [5].

These results emphasise how urgently Iraq needs accessible and efficient cervical cancer screening services. The purpose of this study is to assess the diagnostic efficacy of a quick HPV screening test as a possible means of enhancing early diagnosis and lowering the incidence of cervical cancer, especially in environments with limited resources like Iraq. By assessing its sensitivity, specificity, and accuracy, we seek to contribute to the development of more effective screening strategies that align with WHO's global targets and address the disparities in cervical cancer prevention and care.

METHODOLOGY

Sample Collection

Biological samples, including cancerous tissues, wart samples, and control specimens, were collected for molecular analysis to evaluate the prevalence of Human Papillomavirus (HPV), and its correlation with oncogenesis. The samples included human blood, swabs, and biopsies obtained from patients diagnosed with cervical or urogenital cancer, along with control samples from individuals with no known HPV infection.

Ethical approval:

The study was performed in accordance with the ethical principles that have their origins in the Declaration of Helsinki. The committee of researchers at the Thi-Qar Health Directorate (No. 2024/188 on 20/8/2024) has viewed and approved this study. The person's informed consent was obtained.

DNA Extraction

Genomic DNA was extracted from the collected biological samples using a standard DNA extraction protocol. The process was performed using commercial DNA extraction kits, ensuring high-quality genomic DNA suitable for Polymerase Chain Reaction (PCR) amplification. The effectiveness of the extraction was confirmed by running the samples on a 1% agarose gel electrophoresis at 110V, followed by visualization under UV light after staining with ethidium bromide. Clear and well-defined bands were observed in the gel, confirming the successful extraction of DNA.

Polymerase Chain Reaction (PCR)

The extracted DNA was subjected to Polymerase Chain Reaction (PCR) using specific primers designed to amplify target DNA sequences of HPV. The primers used in this study included:

MY09/11 primers for general HPV detection targeting a 450 bp fragment.

HPV-16 specific primers for detecting the 467 bp fragment of the HPV-16 genome.

HPV-35 specific primers targeting a 230 bp sequence.

HPV-51 specific primers designed to amplify a 270 bp region.

Each PCR reaction was performed using a thermal cycler with an optimized protocol. The PCR protocol is as following:

- The primary temperature for denaturation is 95°C for 5 minutes.
- The primers for MY09/11 and HPV-16, HPV-35, and HPV-51 were denatured 35 times at 95°C for 30 seconds, annealed at 49°C and 59°C, respectively, and then extended at 72°C for 1 minute.
- Last extension: 10 minutes at 72°C.

Gel Electrophoresis

Following PCR amplification, 1.5% agarose gel electrophoresis was used to separate the PCR products. The electrophoresis was performed at 110V for 15 minutes, then the voltage was reduced to 75V and run for an additional 60 minutes. The gels were stained with ethidium bromide and visualized under a UV transilluminator. The gel images were analyzed to confirm the presence of the expected amplicons for each target HPV genotype.

Control and Negative Samples

To ensure the reliability and specificity of the PCR reactions, control samples were included in all PCR runs. Negative control samples, which lacked DNA, were included to rule out contamination, while positive control samples containing known HPV DNA were used to validate the PCR conditions. The negative control did not show any bands, confirming the absence of contamination or false-positive amplification.

Statistical analysis

All analyses were performed using R v4.4.2. Shapiro-Wilk tests was used to test for normal distribution. Descriptive statistics were computed for demographic and clinical variables, with continuous data reported as median (interquartile range) and categorical variables as frequencies (percentages). Non-

parametric analyses were employed for group comparisons: the Kruskal-Wallis test assessed age differences across patient groups. Categorical variables (e.g., family cancer history, HPV genotypes) They were analysed using either Pearson's chi-square test or Fisher's exact test.Diagnostic performance metrics—sensitivity, specificity, PPV, NPV—were derived from a confusion matrix comparing rapid test results to PCR outcomes. Cohen's kappa (κ) quantified agreement beyond chance. A Kappa value of 0 indicates no agreement and a value of 1 indicates perfect agreement. Values from 0.00–0.20 indicate poor agreement, 0.21–0.40 fair, 0.41–0.60 moderate, 0.61–0.80 good, and 0.81–0.99 very good agreement[6]. Statistical significance was set at $p < 0.05$.

Ethical approval:

The study was conducted in accordance with the ethical principles that have their origins in the Helsinki Declaration. This work has been reviewed and authorised by the Thi-Qar Health Directorate's research committee (No. 2024/188 on 20/8/2024). The individual gave their informed consent.

Limitation of the study

Any person who was doubted in his diagnosis regarding the cervical cancer or warts was excluded directly.

RESULTS

The demographic and clinical characteristics of the study participants are summarized in Table 1. Participants with cervical cancer were markedly older (median age 39 years, IQR 34–49) compared to those with genital warts (27 years, IQR 24–32) and controls (35 years, IQR 23–45; Kruskal-Wallis $p < 0.001$). Family cancer history differed significantly ($p = 0.016$), with 45% of cancer patients reporting ≥2 affected family members compared to 5% of controls.

All cancerous and wart samples tested positive for HPV via the rapid test, while controls were uniformly negative ($p < 0.001$). Subtype analysis revealed significant disparities: HPV-16 was detected in 60% of cancerous samples versus 10% of wart samples ($p < 0.001$). HPV-35 positivity was 30% in cancerous samples and 5% in wart samples ($p = 0.013$). and HPV-51 was identified in 40% of cancerous samples and 10% of wart samples ($p = 0.002$). No HPV subtypes were detected in controls (Figure1).

Table 1: Demographic and Clinical Characteristics by Study Groups

Variable	Total (N = 60)[1]	Patient Groups			p-value[2]
		Cancerous Samples (N = 20)[1]	Control (N = 20)[1]	Wart Samples (N = 20)[1]	
Age (years)					***<0.001***
Median (Q1, Q3)	34 (26, 40)	39 (34, 49)	35 (23, 45)	27 (24, 32)	
Age groups					**0.004**
20-29	22 (37%)	2 (10%)	8 (40%)	12 (60%)	
30-39	23 (38%)	9 (45%)	6 (30%)	8 (40%)	
40-49	6 (10%)	4 (20%)	2 (10%)	0 (0%)	
+50	9 (15%)	5 (25%)	4 (20%)	0 (0%)	
Family cancer history					**0.016**
None	34 (57%)	6 (30%)	16 (80%)	12 (60%)	
1 member	12 (20%)	5 (25%)	3 (15%)	4 (20%)	
2+ members	14 (23%)	9 (45%)	1 (5.0%)	4 (20%)	
Rapid test					***<0.001***
Positive	40 (67%)	20 (100%)	0 (0%)	20 (100%)	
Negative	20 (33%)	0 (0%)	20 (100%)	0 (0%)	
MY09/11_primers					***<0.001***
Positive	21 (35%)	17 (85%)	0 (0%)	4 (20%)	
Negative	39 (65%)	3 (15%)	20 (100%)	16 (80%)	
HPV_16					***<0.001***
Positive	14 (23%)	12 (60%)	0 (0%)	2 (10%)	
Negative	46 (77%)	8 (40%)	20 (100%)	18 (90%)	
HPV_35					**0.013**
Positive	7 (12%)	6 (30%)	0 (0%)	1 (5.0%)	
Negative	53 (88%)	14 (70%)	20 (100%)	19 (95%)	
HPV_51					**0.002**
Positive	10 (17%)	8 (40%)	0 (0%)	2 (10%)	
Negative	50 (83%)	12 (60%)	20 (100%)	18 (90%)	

[1]n (%)

[2]Kruskal-Wallis rank sum test; Fisher's exact test; Pearson's Chi-squared test

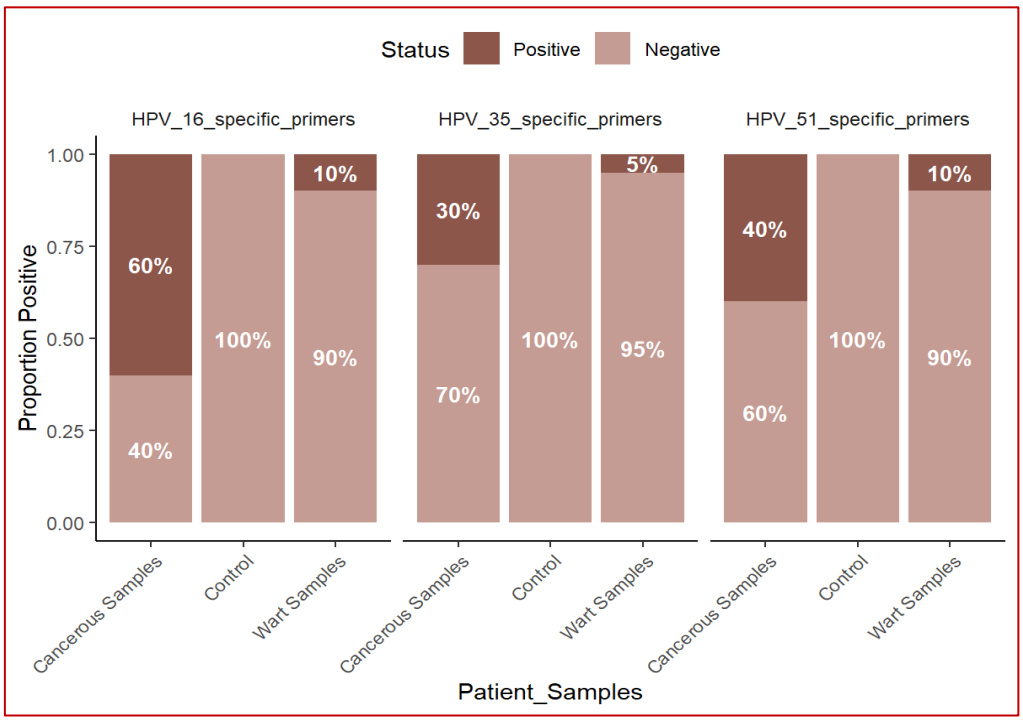

Fig. 1: HPV Genotype among study samples

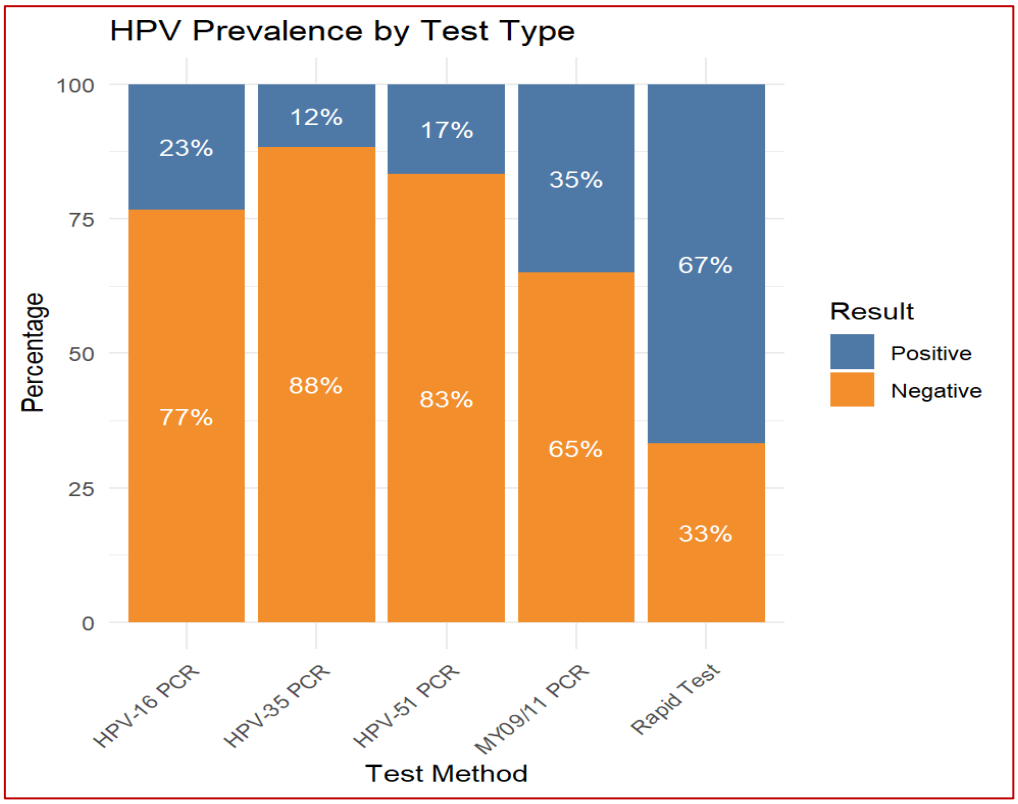

Fig. 2: HPV Genotype Prevalence. HPV genotype-specific assays identified HPV-16 in **23%**, HPV-51 in **17%**, and HPV-35 in **12%** of samples whereas, 67% identified by the HPV rapid test. The stacked bar visualization demonstrates HPV-16's predominance among genotype-specific detections.

Table 2. presents **comparison of PCR Results by Rapid Test Status.** The rapid HPV test demonstrated perfect specificity (100%) across all PCR targets, with none of the 20 rapid test-negative samples yielding positive results for MY09/11 broad-spectrum HPV detection or genotype-specific assays (HPV-16, HPV-35, HPV-51). However, concordance between rapid test positivity and PCR results varied significantly by target. Among the 40 rapid test-positive samples, MY09/11 PCR identified HPV in 53% (21/40) of cases ($p <$ 0.001), while genotype-specific detection rates were lower: 35% (14/40) for HPV-16 ($p = 0.002$), 25% (10/40) for HPV-51 ($p = 0.023$), and 18% (7/40) for HPV-35 ($p = 0.084$). A substantial proportion of rapid test-positive samples were PCR-negative, with discordance rates ranging from 48% (19/40) for MY09/11 to 83% (33/40) for HPV-35. Statistical significance (Fisher's exact test) was observed for MY09/11, HPV-16, and HPV-51, but not for HPV-35, likely due to its low prevalence.

Table 2: Comparison of PCR Results by Rapid Test Status

HPV Genotype	Total (N = 60)[1]	Rapid Test		p-value[2]
		Positive (N = 40)[1]	Negative (N = 20)[1]	
MY09/11_primers				***<0.001***
Positive	21 (35%)	21 (53%)	0 (0%)	
Negative	39 (65%)	19 (48%)	20 (100%)	
HPV_16				**0.002**
Positive	14 (23%)	14 (35%)	0 (0%)	
Negative	46 (77%)	26 (65%)	20 (100%)	
HPV_35				0.084
Positive	7 (12%)	7 (18%)	0 (0%)	
Negative	53 (88%)	33 (83%)	20 (100%)	
HPV_51				**0.023**
Positive	10 (17%)	10 (25%)	0 (0%)	
Negative	50 (83%)	30 (75%)	20 (100%)	

[1]n (%)
[2]Pearson's Chi-squared test; Fisher's exact test

Table 3. demonstrate that the rapid HPV test showed **perfect sensitivity (100%)** across all PCR targets, correctly identifying all HPV-positive samples in the rapid test-positive group. However, specificity varied considerably, ranging from **37.7% (HPV-35)** to **51.3% (MY09/11)**, indicating a high rate of false positives. Cohen's kappa (κ) values further quantified agreement between the rapid test and PCR results, revealing **moderate agreement** for broad-spectrum MY09/11 detection (κ = 0.42, 95% CI: 0.25–0.42) but only **slight-to-fair agreement** for genotype-specific targets (HPV-16: κ = 0.26; HPV-51: κ = 0.18; HPV-35: κ=0.12). The test exhibited **perfect negative predictive value (NPV = 100%)** across all targets, confirming its reliability in ruling out HPV infection when negative. Positive predictive value (PPV), however, was suboptimal, with only **52.5% of rapid test-positive samples confirmed as MY09/11 PCR-positive**, decreasing to **17.5% for HPV-35**. These results, visualized in the agreement plot**,** highlight the rapid test's utility as a screening tool for excluding HPV infection but underscore its limited accuracy for confirming positivity or identifying specific genotypes, particularly HPV-35. The rapid test exhibited substantial discordance with PCR across HPV genotypes, ranging from 43.3% (HPV-16) to 55% (HPV-35). HPV-35 showed the poorest agreement, with over half of cases yielding conflicting results.

Table 3: Comparative Diagnostic Performance of Rapid Human Papillomavirus (HPV) Test Versus PCR for MY09/11 and Genotype-Specific Detection

PCR Target	Sensitivity	Specificity	Cohen's κ (95% CI)	PPV	NPV
MY09/11 PCR	100.0%	51.3%	0.42 (0.25–0.42)	52.5%	100.0%
HPV-16	100.0%	43.5%	0.26 (0.12–0.26)	35.0%	100.0%
HPV-35	100.0%	37.7%	0.12 (0.03–0.12)	17.5%	100.0%
HPV-51	100.0%	40.0%	0.18 (0.06–0.18)	25.0%	100.0%

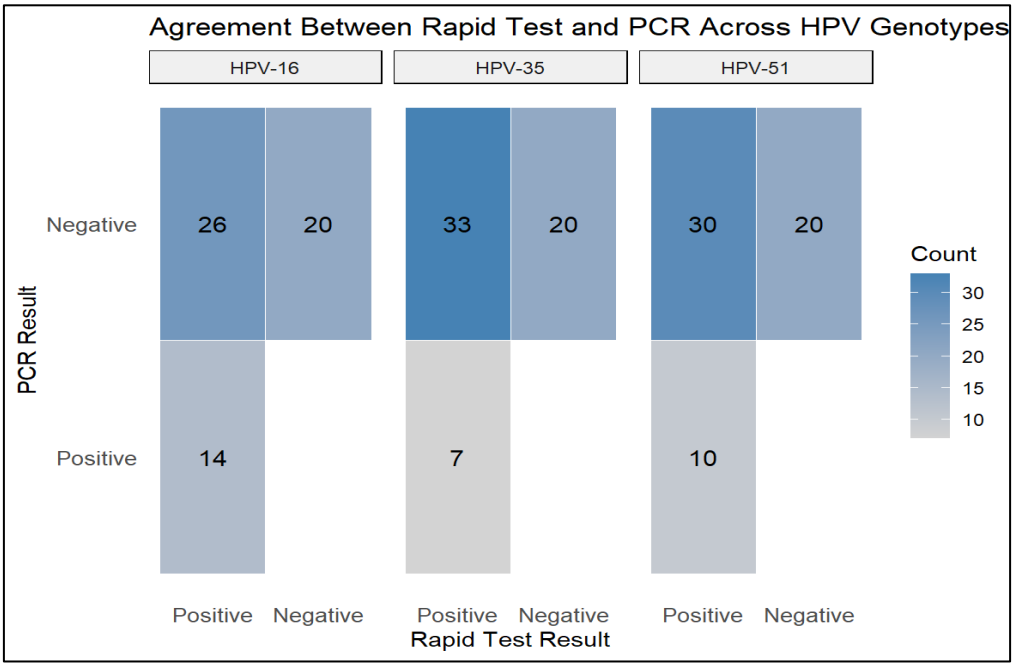

Fig. 3: Agreement plot of PCR results with rapid test results

The co-occurrence analysis of HPV genotypes revealed distinct patterns of infection and co-infection **(Table 4).** HPV-16 was the most prevalent genotype, detected in **14 samples**, followed by HPV-51 (**10 samples**) and HPV-35 **(7 samples).** Pairwise co-occurrence counts demonstrated frequent interactions between HPV-16 and other genotypes, with **7 samples** co-infected with HPV-16 and HPV-51, and **6 samples** co-infected with HPV-16 and HPV-35. In contrast, HPV-35 exhibited limited co-occurrence, sharing infections with HPV-51 in only **4 samples.**

Table 4: HPV Genotype Co-Occurrence

	Co-Occurrence Counts		
	HPV-16	**HPV-35**	**HPV-51**
HPV-16	14	6	7
HPV-35	6	7	4
HPV-51	7	4	10

DISCUSSION

This study highlights the rapid HPV test's role as a sensitive but nonspecific screening tool, emphasizing the irreplaceable role of PCR for diagnostic confirmation. The demographic and molecular distinctions between cancer, wart, and control groups reinforce the importance of tailored prevention strategies based on age, family history, and HPV subtype prevalence. Addressing the test's limitations through technical improvements and validation in diverse cohorts could enhance its clinical utility.

In this study, cervical cancer patients were significantly older (median 38.5 years) than those with genital warts (27.0 years) aligns with the natural history of HPV pathogenesis. High-risk HPV infections, such as HPV-16, often persist for decades before progressing to malignancy, as demonstrated in longitudinal cohort studies [7]. In contrast, low-risk HPV subtypes (e.g., HPV-6/11), which predominantly cause genital warts, typically resolve spontaneously or manifest clinically within 1–2 years post-infection, explaining the younger age of wart patients [8]. The absence of cancer patients under 25 years in our cohort mirrors global data, where cervical cancer incidence peaks in the fourth to fifth decades [9]. These findings reinforce the need for age-stratified screening protocols, as endorsed by WHO guidelines, which recommend initiating cervical cancer screening at 30 years in low-resource settings [10]. The strong association between cervical cancer and a family history of cancer (45% with ≥2 affected relatives) resonates with emerging evidence on genetic susceptibility to HPV persistence. Polymorphisms in HLA class II genes, which regulate immune response to viral antigens, have been linked to increased cervical cancer risk [11].

The predominance of HPV-16 (60% of cancer cases) reaffirms its role as the leading oncogenic subtype globally, responsible for ~60% of cervical cancers [12]. With cervical cancer ranking as the fourth leading cause of female cancer mortality worldwide (660,000 new cases and 350,000 deaths in 2022), our findings underscore the urgency of expanding nonvalent vaccine access to cover regionally prevalent subtypes [13]. HPV

genotype-specific assays identified HPV-16 in **23%**, HPV-51 in **17%**, and HPV-35 in **12%** of samples. This aligns with study by Zandnia *et al.* [14] in which reported that the highest rate of infection was for HPV 16. In Iraq, a study by Jihad *et al.* [4] reported that the High-risk HPV DNA was detected in 19% among women population with the oncogenic HPV-16, -18 and -58 were being the most prevalent high-risk genotypes among women at frequencies of 26.7%, 13.3% and 13.3%, respectively.

The rapid HPV test exhibited perfect sensitivity (100%) and negative predictive value (NPV, 100%), aligning with its intended role as a screening tool to reliably exclude HPV infection. However, its limited specificity (37.7–51.3%) and suboptimal positive predictive value (PPV, 17.5–52.5%) reflect broader challenges observed in rapid HPV assays, where high false-positive rates may compromise diagnostic precision. Notably, the moderate agreement for MY09/11 targets (κ = 0.42) compared to poor concordance for genotype-specific targets (κ = 0.12–0.26) underscores a critical trade-off: while rapid tests are effective for broad HPV detection, their utility diminishes significantly in genotyping accuracy, particularly for less prevalent genotypes such as HPV-35.

These findings mirror heterogeneity in genotype-specific performance reported by Golfetto L *et al.* [6],who observed perfect agreement (κ = 1) for HPV-33/58, very good agreement for HPV-51, and variable concordance for other high-risk types (e.g., HPV-16/18). Divergent detection rates between methods further highlight these limitations. For instance, PCR-RFLP identified HPV co-infections in only 25% (20/80) of cases, whereas the PapilloCheck® microarray detected co-infections in 62.5% (50/80), suggesting method-dependent variability in resolving complex infections.

Recent research support the clinical utility of rapid HPV test in screening setting, reporting 95% sensitivity and 99.2% specificity.[15] However these results contrast with earlier studies emphasizing the diagnostic superiority of molecular assays like PCR.[16] These disparities highlight the necessity of standardised validation procedures and may result from variations in study design, sample size, or population characteristics.

The observed frequent co- occurrence of HPV-16 with HPV-51 (7 cases) and HPV-35 (6 cases) raises compelling questions about potential synergistic interactions among high-risk genotypes. These co-infections may collectively amplify oncogenic risk through shared mechanisms, such as enhanced immune evasion or cumulative genomic instability, accelerating neoplastic progression. Notably, HPV-35 demonstrated reduced co-occurrence with HPV-51 (4/7 samples), suggesting either niche-specific biological behaviors or competitive exclusion between these genotypes. This finding underscores the complexity of HPV genotype interactions and warrants mechanistic studies to elucidate whether such patterns reflect biological competition, differential tropism, or host-pathogen adaptations.

The current research on HPV co-infections remains limited in scope and depth. As highlighted by Bi *et al.*[17], existing studies are predominantly qualitative, lacking granular data on infection dynamics and robust mechanistic discussions. For instance, Wu *et al.*, reported that 33.24% of HPV-16/18-positive individuals harbored concurrent high-risk HPV infections, implying a non-trivial overlap in co-infection risk. [18] Conversely, a research observed that Clade A10 (including HPV-6/11) was more prevalent in multi-infections than Clade A9 (including HPV-16), despite HPV-16's dominance as a single-type infection. [19]

Future research should prioritize quantitative analyses of co-infection patterns, mechanistic exploration of competitive or cooperative behaviors, and longitudinal studies to assess how these interactions influence disease progression.

CONCLUSION

This study reaffirms HPV-16's central role in cervical carcinogenesis while highlighting challenges in rapid test genotyping accuracy. Although the test excels as a screening tool for excluding HPV, its limited specificity and PPV necessitate confirmatory PCR in clinical decision-making. The observed co-occurrence patterns underscore the complexity of HPV interactions, advocating for integrated approaches combining vaccination, screening, and genotype-specific monitoring in high-risk populations.

Acknowledgements
No support or helpfulness was received for this study.
Funding: No funding.
Author contributions:
All authors had seen and approved the submission of the manuscript with full responsibility, and this research had not been published or under consideration by any other journal.
Conflicts of Interest:
The authors declare no competing interests.

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