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High-risk HPV genotype distribution and prevalence in cervical swabs from Western Turkey

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ABSTRACT

This retrospective study analyzed HPV-DNA genotyping, cytological, and histopathological findings from cervical swab samples of women aged 18 and older who attended the Gynecology and Obstetrics outpatient clinic at Balıkesir University Health Practice and Research Hospital between October 2022 and March 2024. A total of 1,447 samples from women aged 19–82 were evaluated. High-risk human papillomavirus (HPV) DNA was detected in 144 cases (9.9 %), including 67 single-type and 77 multiple-type infections. The most frequent types were multiple infections without HPV16/18 (22.92 %) and HPV 16 only(18.75 %). The highest positivity rate (18.7 %) was observed in the 35–39 age group. Cytological abnormalities were found in 52 cases (38.30 %), including infection (24.11 %), atypical squamous cells of undetermined significance (ASC-US, 23.07 %), low-grade squamous intraepithelial lesion (LSIL, 5.77 %), atypical squamous cells—cannot exclude high-grade squamous intraepithelial lesion (ASC—H), high-grade squamous intraepithelial lesion (HSIL), and atypical glandular cells (AGC), each at 1.92. Among HPV-positive patients who underwent biopsy, 6.25 % low-grade squamous intraepithelial neoplasia (LSIL/CIN-I), 4.17 % high-grade squamous intraepithelial neoplasia (HSIL/CIN-II) and 2.78 % HSIL/CIN-III. The results highlight the value of regional HPV genotype surveillance in guiding cervical cancer screening and vaccination strategies

1. Introduction

Cervical cancer is the fourth most common cancer among women worldwide in terms of both incidence and mortality [1]. According to the 2022 cancer statistics reported by the Global Cancer Observatory (GLOBOCAN), the global incidence rate is 14.1 per 100,000 women, and the mortality rate is 7.1 per 100,000. In contrast, the incidence rate in Türkiye is reported as 4.8 per 100,000, with a mortality rate of 2.0 per 100,000. Globally, the prevalence of human papillomavirus (HPV), the primary etiological agent of cervical cancer, is reported to be approximately 11.9 % [2].

Human papillomavirus (HPV) is considered the primary cause of nearly all cervical cancers and is also responsible for a significant proportion of anogenital and oropharyngeal cancers To date, more than 200 different HPV genotypes have been identified, and they are classified based on their oncogenic potential as either high-risk (HR-HPV) or low-risk (LR-HPV) types. Thirteen HPV genotypes—HPV 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59 and 66—are classified by the International Agency for Research on Cancer (IARC) as high-risk , based on sufficient evidence of carcinogenicity in humans. Additionally, HPV 26, 68, 73 and 82 are considered possibly carcinogenic to humans based on limited evidence. These genotypes have been occasionally associated with cervical cancer in case-control studies, but are rarely found in case series and lack consistent evidence from prospective studies. Several other genotypes, including HPV6, 11, 40, 42, 43, 44, 54, 61, 72, and 81, are classified as low-risk or not classifiable as to their carcinogenicity [3,4]. Persistent infection with HR-HPV genotypes has been shown to be the major cause of precancerous lesions of the cervix and cervical cancer [5]. Especially HPV-16 and HPV-18 are the most common genotypes with the highest cancer risk [4]. LR-HPV genotypes are considered non-oncogenic and are primarily associated with benign or low-grade cervical changes, genital warts and recurrent respiratory papillomatosis [6,7].

Cytological evaluation of cervical smears combined with simultaneous HPV testing is referred to as co-testing. Although HPV screening protocols differ across countries depending on their target populations,

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Table 1
The relationship between HPV subtypes and age groups.

HPV GENOTYPES	AGE GRUPS												p*
	<20 (n, %)	20-24 (n, %)	25-29 (n, %)	30-34 (n, %)	35-(n, %)	40-44 (n, %)	45-49 (n, %)	50-54 (n, %)	55-59 (n, %)	60-64 (n, %)	≥65 (n, %)	TOTAL	
Multiple infections without HPV16/18	0(0,00)	0(0,00)	3(9,09)	3(9,09)	6 (18,18)	6(18,18)	8(24,24)	4(12,12)	1(3,03)	1(3,03)	1 (3,03)	33	≤0.001
HPV16 with other types (excluding 18)	0(0,00)	4(14,81)	4(14,81)	3(11,11)	3 (11,11)	4(14,81)	3(11,11)	2(7,41)	1(3,70)	1(3,70)	2 (7,41)	27	0.001
HPV 16 and 18 co-infection only	0(0,00)	0(0,00)	1(100)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	1	-
HPV16 and HPV18 with other types	0(0,00)	1(20,00)	0(0,00)	1(20,00)	1 (20,00)	2(40,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	5	≤0.001
HPV18 with other types (excluding 16)	0(0,00)	2(18,18)	1(9,09)	2(18,18)	1(9,09)	2(18,18)	1(9,09)	1(9,09)	0(0,00)	1(9,09)	0 (0,00)	11	≤0.001
HPV16 HPV16 only	1(3,70)	3(11,11)	4(14,81)	1(3,70)	1(3,70)	6(22,22)	4(14,81)	2(7,41)	3(11,11)	0(0,00)	2 (7,41)	27	≤0.001
HPV18 only	0(0,00)	0(0,00)	1(16,67)	2(33,33)	2 (33,33)	0(0,00)	1(16,67)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	6	≤0.001
HPV31 only	0(0,00)	0(0,00)	1(14,28)	1(14,29)	0(0,00)	2(28,57)	0(0,00)	1(14,29)	0(0,00)	2(28,57)	0(0)	7	≤0.001
HPV33 only	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	1 (100)	1	-
HPV35 only	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	1(100)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	1	-
HPV39 only	0(0,00)	1(20,00)	1(20,00)	1(20,00)	2 (40,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	5	≤0.001
HPV45 only	0(0,00)	0(0,00)	0(0,00)	1(100)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	1	-
HPV51 only	0(0,00)	0(0,00)	1(33,33)	0(0,00)	2 (66,67)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	3	-
HPV52 only	0(0,00)	0(0,00)	0(0,00)	1(20,00)	1 (20,00)	2(40,00)	0(0,00)	1(20,00)	0(0,00)	0(0,00)	0 (0,00)	5	≤0.001
HPV56 only	0(0,00)	0(0,00)	0(0,00)	0(0,00)	3 (75,00)	0(0,00)	0(0,00)	1(25,00)	0(0,00)	0(0,00)	0 (0,00)	4	-
HPV58 only	0(0,00)	0(0,00)	0(0,00)	0(0,00)	2(100)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	2	-
HPV59 only	0(0,00)	1(50,00)	0(0,00)	1(50,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	2	-
HPV66 only	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	0	-
HPV68 only	0(0,00)	0(0,00)	0(0,00)	0(0,00)	3(100)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0(0,00)	0 (0,00)	3	-
TOTAL	1 (% 0,69)	12 (%8,33)	17 (% 11,80)	17 (% 11,80)	27 (% 18,75)	24 (% 16,67)	18 (% 12,50)	12 (%8,33)	5 (%3,47)	5 (%3,47)	6 (% 4,17)	144	≤0.001

*Row percentage.

in Türkiye, the National Standards of the Cervical Cancer Screening Program by the Ministry of Health recommend that asymptomatic women aged 30–65 undergo both smear and HPV-DNA testing every five years [8]. Since cervical cancer typically develops as a result of persistent infection with high-risk HPV (HR-HPV) genotypes, incorporating HR-HPV testing into screening programs significantly enhances their effectiveness by enabling early detection of these infections. Research has shown that the prevalence and distribution of HPV genotypes vary considerably by region [9]. In this study, we aimed to retrospectively evaluate the HPV genotypes detected in cervical swab samples collected over a two-year period from patients who presented to the gynecology and obstetrics outpatient clinic of our hospital. Our objectives were to determine the regional prevalence of HPV, assess the genotype distribution, and correlate these findings with cytological results from cervical smears and histopathological findings from biopsies.

2. Materials and methods

This study was approved by the Balıkesir University, Non-Interventional Research Ethics Committee with decision no. 2024/221 dated 3.12.2024.

This retrospective study included 1,447 cervical swab samples collected from women aged 19–82 years who visited the Gynecology and Obstetrics outpatient clinic of Balıkesir University Hospital between October 2022 and March 2024. HPV-DNA results were evaluated alongside cervical cytology and, when available, cervical biopsy findings. Cytology smears were prepared using the liquid-based ThinPrep method, stained with Papanicolaou (PAP), and reported according to the 2017 Bethesda Classification system.

DNA extraction from the cervical swab samples was performed using the EZ1 Advanced XL automated extraction system (Qiagen, Germany) with the EZ1 Virus Mini Kit v2.0 (Qiagen, Germany). The extracted viral DNA was genotyped using the HPV Genotypes 14 Real-TM Quant kit (Nuclear Laser Medicine, Italy). This test kit is based on real-time PCR (RT-PCR) amplification and is designed for the qualitative and quantitative detection of 14 high-risk HPV genotypes (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, and 68) in urogenital swab and biopsy samples. Amplification and genotyping of the isolated HPV DNA were performed using the Rotor-Gene Q real-time thermal cycler (Qiagen, Germany). The results were qualitatively assessed using dedicated software. For each run, positive and negative controls were included, and internal control evaluation was based on the detection of beta-

Table 2
Genotype distribution in HPV positive cases.

HPV GENOTYPES	POZİTİF ($n = 144$)	
	n	%*
Multiple infections without HPV16/18	33	22,92
HPV16 with other types (excluding 18)	27	18,75
HPV 16 and 18 co-infection only	1	0,69
HPV16 and HPV18 with other types	5	3,47
HPV18 with other types (excluding 16)	11	7,64
HPV16 only	27	18,75
HPV18 only	6	4,17
HPV31 only	7	4,86
HPV33 only	1	0,69
HPV35 only	1	0,69
HPV39 only	5	3,47
HPV45 only	1	0,69
HPV51 only	3	2,08
HPV52 only	5	3,47
HPV56 only	4	2,78
HPV58 only	2	1,39
HPV59 only	2	1,39
HPV66 only	0	0,00
HPV68 only	3	2,08
TOTAL	144	100

*Row percentage.

globin, which indicates the adequacy of cellular material in the sample. The distribution of detected HPV genotypes was analyzed by age groups as follows: <20, 20–24, 25–29, 30–34, 35–39, 40–44, 45–49, 50–54, 55–59, 60–64, and ≥65 years.

Cervical smears obtained from cervical specimens by liquid-based cytology (Thin Prep 2000 liquid system Hologic Quality System) were stained by PAP method and examined by a pathologist and reported according to the Bethesda system. The samples were classified as follows: infection, atypical squamous cells of undetermined significance (ASC-US), atypical squamous cells—cannot exclude high-grade squamous intraepithelial lesion (ASC—H), atypical glandular cells (AGC), low-grade squamous intraepithelial lesion (LSIL), and high-grade squamous intraepithelial lesion (HSIL). Under the category of infection, inflammation with organisms such as *Candida* spp. and *Trichomonas vaginalis*, bacterial vaginosis-like flora, and reactive cellular changes due to radiation or intrauterine device use were evaluated.

Cervical biopsy specimens were stained with hematoxylin and eosin (H&E) and evaluated under light microscope by a pathologist. Immunohistochemical staining with p16 and Ki-67 was also applied. The biopsy findings were reported as reactive cellular changes such as chronic cervicitis, low-grade squamous intraepithelial lesion (LSIL/CIN I), high-grade squamous intraepithelial lesion (HSIL/CIN II–III), and adenocarcinoma.

2.1. Statistical analysis

The data obtained in the study were recorded and statistically analyzed using the SPSS 22.0 software (SPSS Inc., Chicago, IL, USA). Numerical data were presented as percentages and mean ± standard deviation, while categorical data were presented as percentages. The Chi-square test was used to compare independent groups containing categorical variables. Only data with a total count of five or more were included in the evaluation. A p-value of less than 0.05 was considered statistically significant.

3. Results

Of the 1,447 patients, 144 (9.95 %) were HPV-DNA positive. Among them, 46.53 % had single-type and 53.47 % had multiple-type infections. The age range of HPV-positive patients was between 19 and 82 years, with a mean age of 40.3 ± 12.3 years. The highest HPV positivity rate was observed in the 35–39 age group (18.7 %), which was found to be statistically significant ($p < 0.001$). The second highest rate was found in the 40–44 age group (16.67 %), followed by the 45–49 age group (12.50 %) (Table 1).

Table 3
Distribution of cervical smear results (insufficient /normal/abnormal) by HPV genotypes.

HPV GENOTYPES	SMEAR SONUÇLARI*					
	Insufficient		Normal		Abnormal	
	n	%*	n	%	n	%*
Multiple infections without HPV16/18	0	0,00	21	24,14	12	23,08
HPV16 with other types (excluding 18)	1	50,00	16	18,39	9	17,31
HPV 16 and 18 co-infection only	0	0,00	0	0,00	1	1,92
HPV16 and HPV18 with other types	0	0,00	3	3,45	2	3,85
HPV18 with other types (excluding 16)	0	0,00	8	9,19	3	5,77
HPV16 only	1	50,00	19	21,84	6	11,54
HPV18 only	0	0,00	4	4,60	1	1,92
HPV31 only	0	0,00	1	1,15	6	11,54
HPV33 only	0	0,00	1	1,15	0	0,00
HPV35 only	0	0,00	1	1,15	0	0,00
HPV39 only	0	0,00	4	4,60	1	1,92
HPV45 only	0	0,00	0	0,00	1	1,92
HPV51 only	0	0,00	1	1,15	2	3,85
HPV52 only	0	0,00	3	3,45	2	3,85
HPV56 only	0	0,00	2	2,30	2	3,85
HPV58 only	0	0,00	1	1,15	1	1,92
HPV59 only	0	0,00	1	1,15	1	1,92
HPV66 only	0	0,00	0	0,00	0	0,00
HPV68 only	0	0,00	1	1,15	2	3,85
TOTAL	2	1,42	87	61,70	52	36,88

*Column percentage.

When the genotype distribution of HPV-positive cases was examined, the most frequently detected types were as follows: Multiple infections without HPV16/18(22.92 %), HPV16 only (18.75 %), HPV16 with other high-risk types (excluding 18) (18.75 %), HPV18 with other high-risk types (excluding 16) (7.64 %), HPV31 only (4.86 %), HPV18 only (4.17 %), HPV39 only (3.47 %), HPV52 only (3.47 %), HPV16 and HPV18 with other types (3.47 %), HPV56 only(2.78 %), HPV51 only (2.08 %), HPV68 only(2.08 %), HPV58 only(1.39 %), HPV59 only (1.39 %), HPV33 only (0.69 %), HPV35 only (0.69 %), HPV45 only (0.69 %), and co-infection with only HPV16 and HPV18 (0.69 %) (Table 2).

Cytology results were available for 141 HPV-positive patients. Normal cytology was reported in 61.7 %, infection in 24.11 %, insufficient material in 1.42 % and abnormal findings in 36.88 %. The most commonly detected epithelial cell abnormality other than infection was ASC-US (atypical squamous cells of undetermined significance), observed in 23.07 % of the cases, and this was found to be statistically significant ($p < 0.001$). The smear cytology results of the patients—categorized as normal or abnormal (including infection, ASC-US, ASC—H, LSIL, HSIL, and AGC)—along with their corresponding numbers and percentages, are presented in Tables 3 and Table 4.

Among the 52 patients with abnormal cytology, 51.92 % had single-type HPV infections, while 48.08 % had multiple HPV infections. The most frequently detected HPV genotype , multiple infections without HPV16/18 observed in 23.08 % of the patients. This was followed by HPV16 with other high-risk types(excluding 18) (17.31 %), HPV16 only (11.54 %), and HPV31 only (11.54 %). Among six patients who were HPV16 positive and had abnormal smear results, ASC-US was detected in three (50.00 %), infection in two (33.33 %), and HSIL in one (16.66 %). In the single patient who was HPV18 positive and had an abnormal smear, infection (100 %) was reported. ASC-US was the most commonly observed epithelial cell abnormality other than infection in the following genotypes: Multiple infections without HPV16/18 (16.66 %) HPV16 with other high-risk types(excluding 18) (22.22 %), HPV18 with other high-risk types(excluding 16) (33.33 %), HPV31 only (50 %) and

Table 4
Abnormal cervical smear results by HPV genotypes.

HPV GENOTYPES	ABNORMAL CERVICAL SMEAR RESULTS												P*
	Infection		ASC-US		ASC—H		LSIL		HSIL		AGC		Total	
	n	%	n	%	n	%	n	%	n	%	n	%		
Multiple infections without HPV16/18	10	83,33	2	16,66	0	0,00	0	0,00	0	0,00	0	0,00	12	≤0.001
HPV16 with other types (excluding 18)	5	55,55	2	22,22	0	0,00	1	11,11	0	0,00	1	11,11	9	≤0.001
HPV 16 and 18 co-infection only	1	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	1	-
HPV16 and HPV18 with other types	2	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	2	-
HPV18 with other types (excluding 16)	2	66,66	1	33,33	0	0,00	0	0,00	0	0,00	0	0,00	3	-
HPV16 only	2	33,33	3	50,00	0	0,00	0	0,00	1	16,66	0	0,00	6	≤0.001
HPV18 only	1	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	1	-
HPV31 only	3	50,00	3	50,00	0	0,00	0	0,00	0	0,00	0	0,00	6	≤0.001
HPV33 only	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	-
HPV35 only	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	-
HPV39 only	0	0,00	0	0,00	0	0,00	1	100	0	0,000	0	0,00	1	-
HPV45 only	0	0,00	0	0,00	1	100	0	0,00	0	0,00	0	0,00	1	-
HPV51 only	1	50,00	0	0,00	0	0,00	1	50,00	0	0,00	0	0,00	2	-
HPV52 only	2	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	2	-
HPV56 only	2	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	2	-
HPV58 only	1	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	1	-
HPV59 only	1	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	1	-
HPV66 only	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	-
HPV68 only	1	50,00	1	50,00	0	0,00	0	0,00	0	0,00	0	0,00	2	-
TOTAL	34	65,38	12	23,07	1	1,92	3	5,77	1	1,92	1	1,92	52	≤0.001

*Row percentage.

Table 5
Cervical biopsy results by HPV genotypes.

HPV GENOTYPES	**BIOPSY STATUS**														
	None		Adenocarcinoma/ Malignant		LSIL/ CINI		HSIL/ CINII		HSIL/ CINIII		Cervicitis		Negative		Total
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	
Multiple infections without HPV16/18	25	75,76	0	0,00	3	9,09	1	3,03	1	3,03	2	6,06	1	3,03	33
HPV16 with other types (excluding 18)	20	74,07	0	0,00	1	3,70	3	11,11	1	3,70	1	3,70	1	3,70	27
HPV 16 and 18 co-infection only	1	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0	0	0,00	1
HPV16 and HPV18 with other types	3	60	0	0,00	1	20	0	0,00	0	0,00	1	20	0	0,00	5
HPV18 with other types (excluding 16)	7	63,64	0	0,00	1	9,09	0	0,00	1	9,09	1	9,09	1	9,09	11
HPV16 only	20	74,07	1	3,70	2	7,41	1	3,70	0	0,00	3	11,11	0	0,00	27
HPV18 only	6	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	6
HPV31 only	6	85,71	0	0,00	0	0,00	1	14,28	0	0,00	0	0,00	0	0,00	7
HPV33 only	1	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	1
HPV35 only	1	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	1
HPV39 only	4	80	0	0,00	0	0,00	0	0,00	0	0,00	1	20	0	0,00	5
HPV45 only	0	0,00	0	0,00	0	0,00	0	0,00	1	100	0	0,00	0	0,00	1
HPV51 only	2	66,66	0	0,00	1	33,33	0	0,00	0	0,00	0	0,00	0	0,00	3
HPV52 only	5	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	5
HPV56 only	4	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	4
HPV58 only	2	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	2
HPV59 only	2	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	2
HPV66 only	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0
HPV68 only	3	100	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	0	0,00	3
TOTAL	109	75,69	1	0,69	9	6,25	6	4,17	4	2,78	9	6,25	3	2,08	144

*Row percentage.

HPV68 only (50 %). LSIL was found HPV39 only (100 %) and HPV51 only (50 %). A single case of ASC—H was associated with HPV45 (Table 4).

Biopsy results, when available, showed chronic cervicitis as the most frequent diagnosis in HPV16-positive cases (11.11 %), followed by LSIL (7.41 %), HSIL (3.70 %), and adenocarcinoma (3.70 %). Among all HPV-positive patients, LSIL/CIN I was seen in 6.25 %, HSIL/CIN II in 4.17 %, and HSIL/CIN III in 2.78 %. These lesions were associated with various genotypes, including, multiple infections without HPV16/18, HPV16 with other high-risk types(excluding 18), HPV16 and HPV18 with other types, HPV18 with other high-risk types(excluding 16), as well as HPV31 only, HPV45 only, and HPV51 only (Table 5).

4. Discussion

HPV is one of the most common sexually transmitted infections and can lead to a wide range of clinical outcomes, from asymptomatic infections to cervical cancer [7]. Cervical cancer ranks fourth among cancers affecting women globally. According to GLOBOCAN 2022 data, around 662,301 new cases and 348,874 deaths occur annually [10]. While the global prevalence is reported at 11.9 %, regional differences are significant [11]. Determining regional HPV genotype prevalence is crucial for effective vaccination and screening strategies.

HPV prevalence often shows a bimodal age distribution, peaking in women under 25 and again in those aged 45–54 [12–14]. This late-age peak may be due to reactivation of latent infections and hormonal or

immune changes [13]. In Türkiye, some studies report higher positivity in women under 30 [15,16], while others find higher rates in women in their 30 s and 40 s [17,18]. In our study, the highest positivity was in the 35–39 age group (18.75 %), followed by 40–44 (16.67 %) and 45–49 (12.50 %) This age distribution may reflect specific demographic and behavioral characteristics of the study population. For instance, women in their mid to late 30 s may have a higher likelihood of persistent HPV infections acquired earlier in life or may be more likely to attend routine gynecological examinations and cervical cancer screening. Conversely, the relatively lower HPV positivity observed in women under 30 could be influenced by factors such as uneven age distribution in the sample, increased awareness regarding HPV transmission, and the potential impact of recently implemented vaccination programs targeting younger cohorts.These findings highlight the importance of considering local epidemiological factors, healthcare utilization behaviors, and screening practices when interpreting HPV prevalence data.

In Türkiye, HPV prevalence has been reported between 4.39 % and 45.4 % [15,17,19–21]. Our study found a rate of 9.95 %. Variability in prevalence likely stems from differences in geography, methodology, and sample characteristics. Social and cultural factors may also influence HPV rates. Since most studies, including ours, are based on outpatient populations, generalization to the wider population is limited.

Globally, HPV16 is reported as the most commonly detected genotype. It is followed in prevalence by HPV18 and other oncogenic types [12,13,22]. Studies conducted in Türkiye to identify HPV genotypes have generally reported HPV16 as the most frequently detected type, while the prevalence of other genotypes varies [14,15,17,18,20]. However, genotype distributions vary. For example, some studies report HPV51 or HPV59 as the most frequent [21,23,24]. In our study multiple infections without HPV16/18 were most common (22.92 %), followed by HPV16 only(18.75 %) and HPV18 only(4.17 %). Differences may relate to regional, immunologic, or methodological factors. Broader studies with standardized protocols are needed to better define genotype targets for vaccines.

Co-infection with multiple HPV genotypes is frequently observed in the literature [14,15,19,21,25,26]. In our study, single HPV infection was detected in 46.53 % of the HPV-positive cases while multiple high-risk HPV genotypes were identified in 53.47 %. These findings are consistent with some previously published studies [25–27]. While their carcinogenicity remains unclear, some studies associate multiple infections with more severe lesions [26]. This may suggest a higher oncogenic potential, particularly when high-risk types co-occur.

We found that 61.70 % of HPV-positive patients had negative cytology. This is consistent with prior studies [17], supporting the importance of HPV genotyping alongside cytology in screening. High-risk HPV in women with normal cytology still warrants close follow-up [28]. Therefore, it is of great importance to effectively implement screening programs in our country and to re-evaluate the screening intervals in these cases

ASC-US is the most commonly reported cytological abnormality in literature [15–18,21,29], and similarly, it was the most frequent finding in our study (23.07 %). Other abnormalities included LSIL (5.77 %), ASC—H, HSIL, and AGC (1.92 %).

In our study, when HPV positivity was evaluated based on cytological findings, the subtypes most frequently associated with cytological abnormalities were multiple infections without HPV16/18 ; HPV16 with other high-risk types(excluding 18); HPV16 only; and HPV31 only. In addition, HPV18 with other high-risk types (excluding 16); HPV18 only, HPV39 only, HPV45 only, HPV51 only, HPV52 only, HPV56 only, HPV58 only, HPV59 only, HPV68 only were the other types with cytologic abnormalities. Notably, HPV16 was linked to ASC-US (50 %), HSIL (16.66 %), LSIL (7.41 %) and CIN II (3.70 %). It also accounted for one case of adenocarcinoma. Biopsy-confirmed LSIL, HSIL-II and HSIL-III were observed in association with multiple high-risk types, suggesting a greater oncogenic effect of co-infections. . Given the well-established strong association between cervical cancer and HPV infection, the detection of oncogenic high-risk HPV genotypes in abnormal cytological biopsy samples is to be expected.

Determining the prevalence and genotypes of HPV is a crucial step in developing strategies for the prevention of cervical cancer. Currently, highly protective vaccines have been developed against the most oncogenic types of HPV. The World Health Organization recommends vaccination of girls aged 9–14 years against cervical cancer. The HPV vaccine has been included in routine immunization programs in more than 100 countries worldwide. Although the HPV vaccine was first introduced in our country in 2007, it has not yet been incorporated into the national immunization schedule. In addition to screening programs, regional prevalence studies are of great importance for planning HPV vaccination strategies according to local characteristics.

Our study has limitations. It was based on a hospital population, so may not reflect the general community. In addition, limited biopsy data and unclear effects of multiple infections restrict broader interpretation. Nevertheless, by analyzing 1447 patients and correlating age, cytology, and genotype, we provide valuable regional insights that can inform prevention and early detection efforts.

During the preparation of this work the authors used ChatGPT, an AI language model developed by OpenAI, in order to assist with translation. After using this tool, the authors reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

CRediT authorship contribution statement

Nurefşan Erdiren: Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Tuğba Kula Atik:** Writing – review & editing, Project administration, Data curation. **Gülay Turan:** Writing – review & editing, Data curation. **Aslı Gamze Şener:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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