Zeynep Kamil Med J 2025;56(2):84–89
DOI: 10.14744/zkmj.2025.82957

ORIGINAL ARTICLE

Effect of COVID-19 infection on HPV clearance

[1]Celal AKDEMİR
[2]Mücahit Furkan BALCI
[3]Halil İbrahim YILDIZ
[2]Mehmet Bora BOZGEYİK
[1]Özgür ERDOĞAN
[4]Denizhan BAYRAMOĞLU
[1]Muzaffer SANCI

[1]Department of Gynecologic Oncology, Health Sciences University Tepecik Training and Research Hospital, Izmir, Turkey

[2]Department of Obstetrics and Gynecology, Health Sciences University Tepecik Training and Research Hospital, Izmir, Turkey

[3]Department of Medical Oncology, Health Sciences University Tepecik Training and Research Hospital, Izmir, Turkey

[4]Department of Gynecologic Oncology, Izmir City Hospital, Izmir, Turkey

ORCID ID
CA : 0000-0002-4070-7583
MFB : 0000-0002-2821-3273
HİY : 0009-0007-3713-2700
MBB : 0000-0003-2169-0576
ÖE : 0000-0002-0319-3560
DB : 0000-0002-6183-8398
MS : 0000-0002-8494-4302

ABSTRACT

Objective: The objective of the present study was to ascertain the impact of the dysregulated immune response caused by the novel coronavirus (SARS-CoV-2) on the clearance of human papillomavirus (HPV) in infected patients.

Material and Methods: In this retrospective study, patients who were followed up at our centre between 2020 and 2022 due to positive test results for HPV 16 or 18 and who had a documented history of infection with the SARS-CoV-2 virus were included. Patients without a history of SARS-CoV-2 infection constituted the control group. To reduce the number of variables, patients who had received HPV or COVID-19 vaccination were excluded from the study.

Results: The study included 105 patients with single HPV (16 or 18) positivity, of whom 55 (52.4%) were COVID-19 positive and 50 (47.6%) were COVID-19 negative. It was found that HPV clearance was statistically significantly lower in HPV-positive patients infected with COVID-19 compared to those who were not infected ($p<0.001$). Additionally, HPV clearance was found to be statistically significantly lower in the group with a history of smoking ($p<0.001$).

Conclusion: This study demonstrated that the immune response to SARS-CoV-2, commonly referred to as “coronavirus”, may be associated with a reduction in the clearance of HPV types 16 and 18. Despite the modest number of cases included, the findings are significant in establishing a benchmark for future research with larger sample sizes.

Keywords: COVID-19, dysregulated immune response, HPV clearance, lymphopenia, smoking.

Cite this article as: Akdemir C, Furkan Balcı M, Yıldız Hİ, Bozgeyik MB, Erdoğan Ö, Bayramoğlu D, Sancı M. Effect of COVID-19 infection on HPV clearance. Zeynep Kamil Med J 2025;56(2):84–89.

Received: December 28, 2024 **Revised:** January 27, 2025 **Accepted:** February 06, 2026 **Online:** May 23, 2025
Correspondence: Celal AKDEMİR, MD. Sağlık Bilimleri Üniversitesi, Tepecik Eğitim ve Araştırma Hastanesi, Jinekolojik Onkoloji Kliniği, İzmir, Türkiye.
Tel: +90 543 497 13 05 **e-mail:** akdemircelal@gmail.com
Zeynep Kamil Medical Journal published by Kare Publishing. Zeynep Kamil Tıp Dergisi, Kare Yayıncılık tarafından basılmıştır.
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INTRODUCTION

At the end of December 2019, the local health authorities reported an increase in the number of cases of pneumonia of unknown origin. The epidemiological investigation revealed a correlation between the cases under investigation and a seafood market located in Wuhan, Hubei Province, China.[1] The global health crisis caused by Coronavirus Disease 19 (COVID-19) has had a huge impact on so many people all over the world. According to data provided by the World Health Organization (WHO), as of January 2024, the number of confirmed cases had surpassed 774 million, resulting in over seven million deaths on a global scale.[2]

Despite the fact that a considerable number of studies have been conducted since the first case was identified, the pathophysiology of the novel human coronavirus and its dysregulated immune response, characterised by lymphopenia and a cytokine storm, remains to be fully elucidated. The relationship with secondary bacterial and viral infections has not been clearly established to date.[3]

HPV is a virus associated with precancerous lesions and cancers of the genital system in women, infecting mucosal or cutaneous stratified epithelium. Despite prophylactic HPV vaccination programs and PAP smear screening tests, cervical cancer is the fourth most common type of cancer among women who are sexually active.[4] HPV is the most prevalent sexually transmitted infection and the primary causative agent of cervical cancer and genital warts.[5,6]

Approximately 200 HPV strains have been identified in humans, of which around fifteen are classified as high-risk HPV (HR-HPV) strains, which are associated with cancer. It is an established fact that when HPV infections become persistent, there is an increased risk of patients developing precancerous lesions and cancer. This risk is particularly higher in patients who smoke, have immune dysfunction, are malnourished, and live under physiological stress.[7]

To design effective therapeutic interventions for secondary bacterial and viral infections associated with COVID-19, it is crucial to better understand the dysregulated immune response. In our study, we aimed to reveal the impact of the dysregulated immunoregulation caused by COVID-19 on HPV clearance in infected patients.

MATERIAL AND METHODS

Our retrospective study was conducted by examining the records of patients admitted to the Gynaecological Oncology Clinic of Tepecik Training and Research Hospital between 2020 and 2022 who were found to be positive for HPV 16 or 18. In order to reduce variables in our study, only patients who had not received the HPV or COVID-19 vaccines were included.

Patients who underwent colposcopy due to HPV 16 or 18 positivity according to the ASCCP 2019 guidelines and were recommended for a co-test control after one year without the need for biopsy or diagnostic excisional procedure following the colposcopic examination were included in the study. Patients who had COVID-19 between the two tests constituted the main group of the study, while those who did not have COVID-19 between the two tests formed the control group. Patients with uncomplicated COVID-19 and those who were followed up on an outpatient basis with mild to moderate pneumonia were included in the study. Patients who were admitted to hospital or required intensive care due to the infection were not included in the study.

All colposcopic examinations were performed by gynecologic oncology surgery specialists experienced in colposcopy. The research was conducted in accordance with the principles of the Declaration of Helsinki and approved by the Ethics Committee. (Tepecik Ethics Committee, Date: 03/05/2023, Decision No: 2023/04-26).

Statistical Analysis

The data were analyzed using the IBM SPSS Statistics 26.0 (IBM Corp., Armonk, New York, USA) statistical package program. Continuous variables were presented as Mean±Standard Deviation. Categorical variables were presented as percentages (%). The Shapiro-Wilk test was used to investigate the normality of data distribution. For comparisons of normally distributed groups, the independent samples t-test was used when the number of groups was two. For comparisons of non-normally distributed groups, the Mann-Whitney U test was used when the number of groups was two. Chi-Square Test analyses were used in the analysis of the cross-tabulations created. The calculation of the cut-off value for measurements that were statistically significant was performed using ROC (Receiver Operating Characteristic) analysis, with a p-value of <0.05 accepted as the criterion for statistical significance.

RESULTS

The study comprised 105 patients with single HPV (16 or 18) positivity, of which 55 (52.4%) were found to be positive for SARS-CoV-2 and 50 (47.6%) were negative. The average age of the patients was found to be 35.49 years (min: 23, max: 57, std: 6.967). It was observed that 53.3% (n=56) of the patients did not smoke. The average interval between HPV control tests was 12.7 months (min: 11, max: 14, std: 0.763). It was determined that 45 patients (42.9%) were infected with HPV18, and 60 patients (57.1%) were infected with HPV16. Upon examining the control HPV results, clearance was observed in 42 patients (40%), while HPV infection persisted in 63 patients (60%). Among those without clearance, 31 patients (29.5%) continued to be HPV16 positive, and 32 patients (30.5%) continued to be HPV18 positive. When examining the COVID-19 clinical data, it was found that 43 patients (41%) had a mild clinical course, and 12 patients (11.4%) had a moderate clinical course of COVID-19 (Table 1).

When examining HPV clearance in patients who had COVID-19, it was found that HPV clearance was statistically significantly lower in HPV-positive patients infected with COVID-19 compared to those who were not infected with COVID-19 ($p<0.001$) (Table 2). When examining the effect of smoking on clearance in patients who had COVID-19, clearance was statistically significantly lower in patients who smoked ($p=0.031$). The analysis revealed no statistically significant association between the clinical condition and HPV clearance among patients diagnosed with COVID-19 ($p=0.392$). When comparing smoking and HPV clearance, it was found that HPV clearance was statistically significantly lower in the group that smoked ($p<0.001$) (Table 3).

Table 1: Patients overall averages

Parameters	COVID positivity		Total	Parameters	COVID positivity		Total
	Neg. (%)	Pos. (%)			Neg. (%)	Pos. (%)	
HPV DNA (18)				Control HPV DNA 16			
Negative				Negative			
Count	27	33	60	Count	46	28	74
% within HPV DNA (18)	45.0	55.0	100.0	% within control HPV DNA 16	62.20	37.80	100.00
% within COVID infection	54.0	60.0	57.1	% within COVID infection	92.00	50.90	70.50
% of total	25.7	31.4	57.1	% of total	43.80	26.70	70.50
Positive				Positive			
Count	23	22	45	Count	4	27	31
% within HPV DNA (18)	51.1	48.9	100.0	% within control HPV DNA 16	12.90	87.10	100.00
% within COVID infection	46.0	40.0	42.9	% within COVID infection	8.00	49.10	29.50
% of total	21.9	21.0	42.9	% of total	3.80	25.70	29.50
HPV DNA (16)				HPV clearance			
Negative				Yes			
Count	23	22	45	Count	33	9	42
% within HPV DNA (16)	51.10	48.90	100.00	% within HPV clearance	78.60	21.40	100.00
% within COVID infection	46.00	40.00	42.90	% within COVID infection	66.00	16.40	40.00
% of total	21.90	21.00	42.90	% of total	31.40	8.60	40.00
Positive				Negative			
Count	27	33	60	Count	17	46	63
% within HPV DNA (16)	45.00	55.00	100.00	% within HPV clearance	27.00	73.00	100.00
% within COVID infection	54.00	60.00	57.10	% within infection	34.00	83.60	60.00
% of total	25.70	31.40	57.10	% of total	16.20	43.80	60.00
Control HPV DNA 18				Smoking			
Negative				Negative			
Count	37	36	73	Count	31	25	56
% within control HPV DNA 18	50.70	49.30	100.00	% within smoking	55.40	44.60	100.00
% within COVID infection	74.00	65.50	69.50	% within infection	62.00	45.50	53.30
% of total	35.20	34.30	69.50	% of total	29.50	23.80	53.30
Positive				Positive			
Count	13	19	32	Count	19	30	49
% within control HPV DNA 18	40.60	59.40	100.00	% within smoking	38.80	61.20	100.00
% within COVID infection	26.00	34.50	30.50	% within COVID infection	38.00	54.50	46.70
% of total	12.40	18.10	30.50	% of total	18.10	28.60	46.70

HPV: Human papillomavirus DNA: Deoxyribonucleic acid; Neg: Negative; Pos: Positive.

Our investigation revealed no statistically significant correlation between the clearance of HPV and the age of patients (p=0.296). No statistically significant relationship was found between HPV clearance and the time until the control HPV test (p=0.719). Similarly, no statistically significant relationship was identified between clearance and lymphopenia (p=0.141) (Table 4).

Table 2: Relationship between COVID-19 infection and HPV clearance

	Clearance		Total	p
	Yes	No		
COVID infection				<0.001
Negative	33	17	50	
Positive	9	46	55	
Total	42	63	105	

HPV: Human papillomavirus.

Table 3: Relationship between smoking and HPV clearance

	HPV clearance		Total
	Positive	Negative	
Smoking			
Negative	34	22	56
Positive	9	46	55
Total	42	63	105

HPV: Human papillomavirus.

Table 4: Factors affecting HPV clearance

Parameters	p
Covid infection	**0.001**
Smoking	**0.001**
COVID clinical condition	0.392
Age	0.296
Time between control HPV test	0.719

HPV: Human papillomavirus.

DISCUSSION

We investigated the impact of changes in the immune system caused by COVID-19 on the clearance of HPV, especially related to HPV16 and HPV18. It is understood that the present study is the first of its kind in the relevant literature. The findings of this study indicated that patients infected with HPV exhibited a statistically significant decrease in clearance rates in comparison to those who were not infected with HPV.

The majority of women (~90%) clear HPV infections spontaneously within a period of 6–18 months.[8] Research in the field has demonstrated that host defence mechanisms, the genital microbiome, and other factors present in the female genital tract play pivotal roles in the clearance or persistence of HPV, including the risk of developing cervical cancer.[9,10]

A number of studies have demonstrated an association between innate immune responses and host susceptibility to infection, as well as the development of cervical cancer and preinvasive lesions. Nevertheless, the causality of this relationship remains to be substantiated.[11]

The serum levels of interleukin-6 and interleukin-10 have been observed to increase in cases of SARS-CoV-2 infection, with the gene expression of these two interleukins being identified as a contributing factor to this increase. It is evident that heightened secretion of the anti-inflammatory cytokine IL-10 in the Th2 response is associated with compromised innate and adaptive immune defence and cervical lesion progression during high-risk HPV infection. This association bears similarity to the association observed in the context of SARS-CoV-2 infection.[12]

It has been established that macrophages play a pivotal role in the eradication of HPV infection through the production of two key mediators: nitric oxide-dependent mechanisms and tumour necrosis factor-alpha.[13] Natural killer cells have been shown to help clear viruses, get rid of cells infected with HPV, and stop cancer from developing in cases where HPV causes cancer.[14] Cytotoxic T lymphocytes, CD4+ T cells, and other Th1 responses have been demonstrated to play a pivotal role in the effective clearance of HPV16 and HPV18.[15]

However, further research is required to fully characterise the factors that render women more vulnerable to HPV infection. Nevertheless, further studies are required to definitively establish the factors that render HPV-infected patients susceptible to HPV infection. The identification of cellular and cytokine markers of inflammation as biomarkers associated with neoplastic disease in cervical carcinogenesis has been well validated.[16]

Tian et al.[17] conducted an interrupted time series analysis of HPV prevalence, incidence, and clearance among men who have sex with men in Xinjiang, China. The analysis compared the periods before and during implementation of non-pharmaceutical interventions to control the SARS-CoV-2 pandemic. In contrast to our study on clearance, they found an increase in clearance in their study. The application of binomial segmented regression analysis revealed the clearance of various HPV types, including low-risk HPV and HPV16. However, the same binomial segmented regression analysis demonstrated a decline in clearance of HPV6 and HPV18. The investigation revealed a decline in the prevalence and incidence of numerous HPV genotypes, concomitant with an increase in clearance, in the period following the implementation of NPIs. However, the study utilised a less rigorous definition of clearance (this is to be understood as follows: two negative visits in succession following a positive visit [1-0-0]), which may have resulted in an underestimation of HPV incidence and clearance. The study revealed that trends in clearance of HPV remained relatively unchanged between the periods preceding and coinciding with the implementation of NPIs. The NPIs implemented in response to the pandemic of 2020 may have contributed to the prevention of HPV infections through the enforcement of stringent social distancing measures, which resulted in a significant reduction in high-risk sexual behaviours in the period of the pandemic and subsequent blocking periods.

The persistence of HPV infection is a prerequisite for the initiation of the oncogenic process. A substantial body of evidence exists that attests to the fact that clearance of infection is a common occurrence in young adults. The load and type of virus are the primary cofactors in the progression of infection to cervical intraepithelial lesions and cervical cancer. Additional risk factors for progression to cancer include smoking, hormonal exposure, and HIV.[18] In addition, HPV infection in patients with a weak immune system, like those who have had a transplant, can be more common and harder to treat.[19]

In the case of cervical carcinoma and HPV-positive dysplastic lesions, the reason for the inadequate level of nitric oxide is the insufficient expression of inducible nitric oxide synthase (iNOS) that appears to be progressively reduced with the histological severity of lesions.[20] Nitric oxide is a gasotransmitter that helps the body fight infection. It also affects blood vessel function. The body makes NO when it is fighting off infection, and it stops viruses from spreading.[21] In conclusion, NO has been identified as a pivotal component of the immune system. The role of activated macrophages in producing NO has been demonstrated to elicit antimicrobial, antitumour, and antiviral effects.[22] Banerjee et al.[23] discovered that a novel nitric oxide-releasing compound has the capacity to inhibit HPV18 virus production by interfering with the functions of the E6 and E7 oncoproteins. Furthermore, Tyring et al.[24] discovered evidence that the investigational topical drug SB206 releases NO and has the potential to treat genital and perianal warts, which are primarily caused by HPV types 6 and 11. Concurrently, the reduced NO concentration has been demonstrated to encourage mutagenesis, thereby expediting neoplastic progression and fostering angiogenesis, which is mediated by VEGF. This process ensures a sufficient supply of metabolites for the sustained growth of the tumour. Theoretical considerations indicate that oxidative stress and high-risk HPV, two potent initiating and promoting carcinogens, may act synergistically. Indirect clinical epidemiological evidence and preliminary biochemical data support the conclusion that oxidative stress potentiates viral infection, the establishment of persistent chronic infection, and viral integration.[25]

Like HPV, coronavirus infection can also activate both innate and adaptive immune responses in infected patients. The irregular immune response to coronavirus infection has not yet been fully explained. Initially, COVID-19 was characterized by a hyperinflammatory pathophysiology. However, new data emerging in the later stages of the pandemic have shown that some patients infected with the virus can also exhibit an immunosuppressive profile. Immune activation from complements and neutrophils creates inflammatory damage in the lungs, while decreased antiviral responses, irregular dendritic cells and macrophages, severe lymphopenia, and an increased tendency for secondary infections occur. Lymphopenia is a common feature of coronavirus infection; a significant decrease is observed in T cells (CD4+ and CD8+) and CD19+ B cells. Emerging new data have shown that reductions in T and B cells occur not only in severe infections but also in non-severe infections, suggesting that there may be immune dysfunction in the pathogenesis of COVID-19. It is thought that the irregular immune responses in the pathogenesis of COVID-19 increase the susceptibility to secondary infections and reduce the clearance of these secondary infections. A substantial number of patients with confirmed cases of the novel coronavirus have exhibited signs of leukocytopenia, with a particular prevalence of lymphocytopenia, as indicated by laboratory findings.[26]

In contrast to COVID-19, we did not find a statistically significant relationship between HPV clearance and lymphopenia in our study (p=0.141). Rather than lymphopenia, the increased concentrations of IL-10 and TGF-β in tissues may indirectly compromise T cell function by restricting the capacity of antigen-presenting cells to encourage CD4+ T cell differentiation and proliferation. This, in turn, affects the adaptive immune responses associated with HPV clearance.[27]

However, the precise results demonstrating the specific impact of smoking on HPV persistence and the corresponding mechanism have yet to be fully established. A study of Romanian women demonstrated a link between smoking and persistent HPV infection, with an odds ratio of 2.320 and a p-value of 0.033.[28] A further study conducted in Britain discovered no substantial correlation between smoking and the likelihood of HR-HPV persistence following a period of six months (risk ratio, RR=1.12; p=0.516).[29] In accordance with the findings of the aforementioned studies, it was also determined that there was a statistically significant decrease in HPV clearance in patients who smoked (p=0.031).

There are several limitations to our study. Firstly, the study included a retrospective, single-centre, small sample group of patients; standardized data for a larger cohort would be better for evaluating HPV clearance after COVID-19 infection. Secondly, our interrupted time series cannot determine whether a causal relationship exists between previous COVID infection and HPV clearance. Finally, it is important to note that our study did not utilise a stricter definition of clearance (such as two consecutive negative visits following a positive visit [1-0-0]), which could potentially lead to an underestimation of HPV clearance. Despite these, only patients who had not received the HPV or COVID-19 vaccines were included to reduce variables in our study.

CONCLUSION

In conclusion, the present study revealed that the dysregulated immune response to SARS-CoV-2 may reduce the clearance of human papillomavirus (HPV) types 16 and 18. Despite the limited number of cases in the present study, its findings are important as a benchmark for future research with larger sample sizes. Further studies with larger sample sizes and evaluation of changes in immune cell ratios in cervical tissue may elucidate the relationship between HPV and SARS-CoV-2.

Statement

Ethics Committee Approval: The Health Sciences University Tepecik Training and Research Hospital Hospital Ethics Committee granted approval for this study (date: 03.05.2023, number: 2023/04-26).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Use of AI for Writing Assistance: Not declared.

Author Contributions: Concept – CA, MFB; Design – CA; Supervision – MS, DB; Resources – ÖE; Materials – MBB; Data collection and/or processing – CA; Analysis and/or interpretation – CA, HİY; Literature search – CA, MFB; Writing – MBB, CA; Critical review – MS, DB.

Peer-review: Externally peer-reviewed.

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