

The Impact of The COVID-19 Pandemic on Smoking Cessation

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Abstract

Objective: In our study, we aimed to reveal the number of applications made to the smoking cessation clinic in our hospital, smoking cessation behavior, and the relationship between this situation and the status of being diagnosed with COVID-19 in the nine months before and after March 11, 2020, when the first case with a diagnosis of COVID-19 was detected in our country.

Methods: Individuals over 18 who applied to the Ministry of Health's Ordu University Training and Research Hospital smoking cessation outpatient clinic within nine months before (Group A) and after the COVID-19 pandemic (Group B) were included in our study. Age, gender, chronic diseases, Fagerström addiction scores, and treatment they received for smoking cessation were noted through the tobacco addiction treatment monitoring system (TÜBATİS), and their smoking status was questioned by reaching them three months after their application to us. In the first year of the pandemic, the patients in Group A were reached again and questioned about whether they had been diagnosed with COVID-19 (PCR positivity).

Results: It was determined that there were 320 patients in Group A and 60 patients in Group B, and there was a statistically significant difference in age and smoking cessation behavior between the two groups ($p < 0.05$). While 20.6% of Group A was 55 years old and over and 8.1% was under 25 years old, these rates were 13.3% and 21.7% in Group B, respectively ($p = 0.041$). The percentage of those who quit smoking was 48.9% in Group A and 30.9% in Group B ($p = 0.029$). When the patients in Group A were re-evaluated in the first year of the pandemic, the rate of having COVID-19 was 6.6% in those who quit smoking, 6.3% in those who did not quit, and 31.2% in those who quit and started again ($p = 0.001$).

Conclusion: Health services have had to give up their workforce to fight the epidemic during the pandemic process, and therefore, there has been a decrease in patient admissions in smoking cessation polyclinics. During the restriction periods, there was an increase in the tendency to smoke due to reasons such as social isolation, increased mental and physical slowdown, psychological effects, and economic concerns, and a decrease in the application to health centers due to the risk of transmission, especially in elderly patients with chronic diseases. We think the decrease in our smoking cessation rates and especially in the applications of patients over 55 years old compared to the pre-pandemic period may be due to this reason. Smoking cessation studies should be carried out more decisively, and information should be provided about the combined risks associated with smoking, even in regular outpatient clinic meetings.

Keywords: COVID-19 infection, smoking addiction, smoking cessation clinic

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INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a disease that mainly affects the respiratory system and can progress to acute respiratory failure. The disease picture occurs when the causative Severe Acute Respiratory Syndrome-Coronavirus-2 (SARS-CoV-2) rarely enters the body from the upper respiratory tract, rarely from the conjunctival mucosa, and mainly from the nose and mouth.

There are transmembrane spike (S) glycoproteins on the cell surface of coronaviruses. This glycosylated cell surface protein contains two distinct functional domains (S1 and S2) that are thought to mediate virus entry into the host cell. The S1 domain contains the angiotensin-converting enzyme-2 (ACE2) receptor binding domain and is responsible for host cell entry (1).

The increase in the expression of this gene, which is responsible for cell entry, increases the affinity and allows the virus to spread more easily from person to person. Studies have shown that ACE2 expression increases in those who consume cigarettes and tobacco products (2, 3).

In addition to increased ACE2 gene expression in smokers, COVID-19 infection and complications arising from accompanying comorbidities play an active role in the severe course of the infection. Comorbidities, especially cardiovascular and chronic respiratory diseases, can cause COVID-19 to

progress into a severe clinical picture and result in mortality (4).

Smoking or passive exposure to tobacco products, especially cigarettes, deteriorates mucociliary activity in the respiratory tract, increases permeability in the epithelium, and causes an inflammatory response (5). COVID-19 causes unexplained and abnormal blood clotting by predisposing to thrombosis, and smoking addiction increases this complication (6).

Identifying and preventing potential host risk factors reduces virus transmission and the severity of COVID-19 infection. In our study, we aimed to reveal the number of applications made to our hospital's smoking cessation outpatient clinic during the nine months before and after the pandemic, smoking cessation behavior, and the relationship between this situation and the status of being diagnosed with COVID-19.

METHODS

Permission for our study was received from the Ordu University Ethics Committee with KAEK application number 58 and decision number 51. Individuals over 18 who applied to the Ministry of Health's Ordu University Training and Research Hospital smoking cessation outpatient clinic within nine months before (Group A) and after the COVID-19 pandemic (Group B) were included in our study. Age, gender, chronic disease, Fagerström addiction score, and treatment they

received for smoking cessation were noted through the tobacco addiction treatment monitoring system (TÜBATİS). The patients were contacted three months after their application to us, and their smoking status was questioned. In the first year of the pandemic, the patients in Group A were reached again and questioned about whether they had been diagnosed with COVID-19 (PCR positivity).

Statistical Analysis

Analyses were performed with IBM SPSS Package Program version 22.0 (IBM Corporation, Armonk, NY, USA). Discrete and categorical data were expressed as numbers and percentages. The chi-squared test was used to compare these data. $P < 0.05$ was considered the statistical significance level.

RESULTS

While the number of patients who applied to the smoking cessation outpatient clinic in the nine months before the pandemic (Group A) was 320, this number was only 60 in the nine months after the pandemic (Group B). It was determined that there was a statistically significant difference between the age of the patients and their success in smoking cessation treatment before and after the pandemic ($p < 0.05$). While 20.6% of the patients who applied to the outpatient clinic before the pandemic were aged 55 and over, 8.1% were under 25; these rates were 13.3% and 21.7%, respectively, in the post-pandemic period ($p = 0.041$). There was no significant difference

between the genders in the smoking cessation outpatient clinic admission. The patients were given nicotine replacement therapy, varenicline, varenicline+nicotine replacement therapy, bupropion+nicotine replacement therapy, or psychosocial support therapy, and there was no significant difference between the two groups in terms of the treatments given (Table 1).

A statistically significant result was obtained when the success of the given treatment was questioned. While the percentage of those who applied to the outpatient clinic and quit smoking was 48.9% before the pandemic, this rate was only 30.9% in the post-pandemic period ($p = 0.029$) (Table 2).

The smoking status of addicts according to the Fagerström addiction score in the pre-pandemic and post-pandemic periods is presented in Table 3. There were no statistically significant differences in smoking cessation status according to addiction level both before and after the pandemic ($p = 0.990$ and $p = 0.794$, respectively) (Table 3).

Among the patients in Group A who were reached in the first year of the pandemic, the status of being diagnosed with COVID-19 (PCR positivity) was 6.6% for those who quit smoking, while this rate was 6.3% for those who did not quit smoking and 31.2% for those who quit and started again ($p = 0.001$) (Table 4).

Table 1. Application to the smoking cessation outpatient clinic in the nine months before and after the pandemic

	Group A		Group B		Total		p-value
	N	%	N	%	N	%	
Age							
18-25	26	8.1	13	21.7	39	10.5	0.041
26-35	77	24.1	11	18.3	88	23.1	
36-45	87	27.2	16	26.7	103	27.0	
46-55	64	20.0	12	20.0	76	19.9	
56-65	50	15.6	5	8.3	55	14.4	
Over 65	16	5.0	3	5.0	19	5.0	
Total	320	100.0	60	100.0	380	100.0	
Gender							
Female	119	37.2	18	30	137	36.1	0.287
Male	201	62.8	42	70	243	63.9	
Total	320	100.0	60	100.0	380	100.0	
Treatment Received							
Nicotine replacement	25	7.8	3	5	28	7.3	0.768
Varenicline (Champix)	268	83.8	51	85	319	83.7	
Varenicline (Champix)+ nicotine replacement	1	0.3	-	-	1	0.3	
Bupropion+nicotine replacement	3	0.9	-	-	3	0.8	
Psychosocial support	23	7.2	6	10	30	7.9	
Total	320	100.0	60	100.0	380	100.0	
Chronic Disease Status							
None	217	67.8	40	66.7	258	67.7	0.693*
Hypertension	29	9.1	5	8.3	34	8.9	
Diabetes mellitus	10	3.1	3	5	13	3.4	
COPD	7	2.2	2	3.3	9	2.4	
Asthma	7	2.2	-	-	7	1.8	
Heart failure/atherosclerotic heart disease	1	0.3	-	-	1	0.3	
Depression	8	2.5	2	3.3	10	2.6	
Liver failure	1	0.3	-	-	1	0.3	
Multiple systemic diseases	40	12.5	8	13.4	48	12.6	
Total	320	100.0	60	100.0	380	100.0	

* Chi-square test

Table 2. Smoking cessation status with the given treatment

	Group A		Group B		Total		p-value
	N	%	N	%	N	%	
Smoking status							
Quit	152	48.9	17	30.9	169	46	0.029*
Quit and start again	16	5.1	2	3.6	18	4.9	
Did not quit	143	46.0	36	65.5	179	49	
Total	311	100.0	55	100.0	366	100.0	

* Chi-square test

Table 3. Comparison of smoking cessation status according to addiction level

Fagerström addiction score	Smoking status (Pre-pandemic)			Total	p-value
	Quit	Quit and start again	Did not quit		
Very low dependence	2 (1.3)	0 (0.0)	3 (2.1)	5 (1.6)	0.990*
Low dependence	7 (4.6)	1 (6.3)	5 (3.5)	13 (4.2)	
Moderate dependence	17 (11.2)	2 (12.5)	19 (13.3)	38 (12.2)	
High dependence	47 (30.9)	5 (31.5)	39 (27.3)	91 (29.3)	
Very high dependence	79 (52.0)	8 (50.0)	77 (53.8)	164 (52.7)	
Total	152 (100.0)	16 (100.0)	143 (100.0)	311 (100.0)	
Fagerström addiction score	Smoking status (Post-pandemic)			Total	p-value
	Quit	Quit and start again	Did not quit		
Moderate dependence	1 (5.9)	0 (0.0)	5 (13.9)	6 (10.9)	0.794*
High dependence	6 (35.3)	1 (50.0)	9 (25.0)	16 (29.1)	
Very high dependence	10 (58.8)	1 (50.0)	22 (61.1)	33 (60.0)	
Total	17 (100.0)	2 (100.0)	36 (100.0)	55 (100.0)	

* Chi-square test

Table 4. Group A smoking status and COVID-19 PCR positivity

Smoking status (Group A)	COVID-19			Total	p-value
	With positivity	PCR	Without positivity		
Quit	10 (6.6)		142 (93.4)	152 (100.0)	0.001
Quit and start again	5 (31.2)		11 (68.8)	16 (100.0)	
Did not quit	9 (6.3)		134 (93.7)	143 (100.0)	
Total	24 (7.7)		287 (92.3)	311 (100.0)	

DISCUSSION

Active or passive exposure to tobacco products increases the risk of respiratory tract infections. It is known that all tobacco products, especially cigarettes, cause lung damage through the activation of inflammatory cytokines, programmed cell death in the pulmonary tissue, and circulating immune cells such as T cells (7).

In addition to this immunological mechanism, which causes susceptibility to infection in smokers, structural damage is also

observed. These structural changes are the main ones that include inflammation and fibrosis around the bronchi and alveoli, increased permeability of the respiratory tract mucosa, and inadequate mucociliary clearance (8).

It has been found that the angiotensin-converting enzyme-2 (ACE2) gene, which is thought to mediate the entry of the coronavirus into the host cell, is more expressed in respiratory tract samples of smokers (2, 3). One study found that ACE2 gene expression did not differ significantly between genders and age

groups, but it was higher in male smokers than in non-smokers. For this reason, it has been stated that the higher number of cases in men and the more severe clinical course in China may be related to the fact that men smoke more (3, 9).

In a study by Zhang et al., in which 140 patients were included and 58 patients with severe clinical conditions were examined, it was found that 3.4% were still smoking and 6.9% had smoked in the past (10). Guan et al. included 1099 patients with COVID-19, 173 of whom were severe and 926 of whom were in a mild clinic; 16.9% of the severe ones were smokers, 5.2% of them were past smokers, and 11.8% of the mild ones were smokers, and 1.3% of them had smoked in the past and quit. In the patients included in the study, clinical worsening developed that required follow-up in the intensive care unit in 25.5% of smokers and 7.6% of former smokers (11).

In a meta-analysis evaluating the relationship between smoking and COVID-19, when the test positivity and hospitalization rates of smokers, quitters, and never-smokers were evaluated, it was found that smokers had lower test positivity than never-smokers. However, hospitalization rates were higher in smokers than in non-smokers (12).

In another study evaluating those who have never smoked, those who smoke, and those who are still smokers in patients infected with COVID-19, values close to those of who have

never smoked were found in those who quit (13).

In a study investigating the relationship between clinical worsening and smoking, 78 patients had 27.3% cigarette smoking in the severe clinical group and 3.0% in the mild clinical group (14).

In a study examining the risk factors of cases diagnosed with COVID-19 who developed fibrotic lung disease during follow-up, it was found that the severity of the disease, length of stay in the intensive care unit and mechanical ventilator, advanced age, smoking, and alcohol use facilitate the progression to fibrosis (15).

When comorbidities and smoking status were examined, the presence of comorbid diseases was higher in smokers among 1590 patients diagnosed with COVID-19. One comorbid disease (1.79 times), and two or more comorbid diseases (2.59 times) cause a more severe course in the COVID-19 clinic (16).

Considering that there may also be asymptomatic cases infected with SARS-CoV-2, it is not clear whether smoking is associated with SARS-CoV-2 infection or with this infection becoming symptomatic.

Some studies also state that smoking is not associated with severe COVID-19 infection. In a meta-analysis study, it was stated that COVID-19 does not cause a severe clinical picture in patients who are smokers and that smoking cannot be shown as a cause of exacerbation of the disease (17).

It has also been hypothesized that nicotinic acetylcholine receptors (nAChR) play a critical role in the pathophysiology of SARS-CoV-2 infection, and as a result, nicotine and nicotinic orthosteric and/or allosteric agents can be recommended as a possible treatment for SARS (18).

Ivermectin, which has been shown to inhibit the replication of SARS-CoV-2 in *in vitro* cells, is a positive allosteric modulator of $\alpha 7$ nAChR (19, 20). The nicotinic hypothesis should be further explored with experimental observations, electrophysiological testing, and animal experiments determining whether SARS-CoV-2 physically interacts with nAChR *in vitro*. These opposing viewpoint studies are hypothetical.

There has been an increase in the effort to quit smoking due to fear and anxiety in societies during the pandemic period. In a study conducted in our country, it was stated that while the success rate of smoking cessation was 23.7% in the pre-pandemic period in patients who applied to the smoking cessation outpatient clinic, this rate increased to 31.1% with the pandemic period (21).

A meta-analysis observed that COVID-19 infection causes a 30-50% more severe clinical picture in current and former smokers than in never-smokers (22). Although current studies are insufficient to draw firm conclusions about the relationship between the risk of contracting COVID-19 infection and the severity of

infection and smoking status, preventing serious consequences of COVID-19 infection, including death, is the most convincing reason against smoking.

The limitations of our study include some important restrictions on smoking cessation clinic services due to the effects of the COVID-19 pandemic on healthcare services. These restrictions have manpower limitations, patient reductions, and psychological effects. During the pandemic period, manpower in health services was used intensively, especially to combat the pandemic. Therefore, there were not enough personnel in smoking cessation clinics to provide further services to patients. Older individuals with chronic diseases have reduced their visits to health centers in order to avoid COVID-19 infection. This situation has also led to a decrease in applications to smoking cessation clinics. The pandemic has come with factors such as social isolation, mental and physical slowdown, and economic concerns. These factors have negatively affected the psychological health of individuals participating in smoking cessation treatment. Therefore, it may have reduced the success of smoking cessation treatment.

These limitations may affect the effectiveness of smoking cessation clinics during the pandemic period. Future studies may focus on overcoming these challenges and investigating how we can make smoking

cessation treatment more effective under pandemic conditions.

CONCLUSION

Health services had to use their workforce to fight the epidemic during the pandemic process. As in our center, there has been a decrease in patient acceptance in smoking cessation outpatient clinics. The number of patients aged 55 and older who applied to our smoking cessation outpatient clinic decreased after the pandemic, while the number of patients aged 25 and under increased. We think this may have been caused by elderly people with chronic diseases avoiding applying to the health institution due to the fear of being severely affected by the epidemic.

Social isolation, mental and physical slowdown, psychological effects, and economic reasons lead to increased cigarette smoking during restriction periods. We think the decrease in our smoking cessation rates compared to the pre-pandemic period may be due to this.

Considering that smokers may be more affected by COVID-19 infection during this period and the clinical course may be worse, smoking cessation studies should be carried out more decisively, and information should be provided about the combined risks of smoking even in regular outpatient clinic meetings.

Ethics Committee Approval: Approval for the study was obtained from the Ministry of Health (<https://bilimselarastirma.saglik.gov.tr/>) and Ordu University Clinical Research Ethics Committee with the decision number KAEK 58 2021/51.

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REFERENCES

1. Li F, Li W, Farzan M, Harrison SC. Structure of SARS coronavirus spike receptor-binding domain complexed with receptor. *Science*. 16 Eylül 2005;309(5742):1864-8.
2. Brake SJ, Barnsley K, Lu W, McAlinden KD, Eapen MS, Sohal SS. Smoking Upregulates Angiotensin-Converting Enzyme-2 Receptor: A Potential Adhesion Site for Novel Coronavirus SARS-CoV-2 (Covid-19). *J Clin Med*. 20 Mart 2020;9(3):841.

3. Cai G, Bossé Y, Xiao F, Kheradmand F, Amos CI. Tobacco Smoking Increases the Lung Gene Expression of ACE2, the Receptor of SARS-CoV-2. *Am J Respir Crit Care Med*. 15 Haziran 2020;201(12):1557-9.
4. Zheng YY, Ma YT, Zhang JY, Xie X. COVID-19 and the cardiovascular system. *Nat Rev Cardiol*. Mayıs 2020;17(5):259-60.
5. Samet JM. Tobacco Products and the Risks of SARS-CoV-2 Infection and COVID-19. *Nicotine Tob Res*. 01 Aralık 2020;22(Supplement_1):S93-5.
6. Sifat AE, Nozohouri S, Villalba H, Vaidya B, Abbruscato TJ. The Role of Smoking and Nicotine in the Transmission and Pathogenesis of COVID-19. *J Pharmacol Exp Ther*. 01 Aralık 2020;375(3):498-509.
7. Nordman JC, Muldoon P, Clark S, Damaj MI, Kabbani N. The $\alpha 4$ nicotinic receptor promotes CD4⁺ T-cell proliferation and a helper T-cell immune response. *Mol Pharmacol*. Ocak 2014;85(1):50-61.
8. Dye JA, Adler KB. Effects of cigarette smoke on epithelial cells of the respiratory tract. *Thorax*. Ağustos 1994;49(8):825-34.
9. Cai H. Sex difference and smoking predisposition in patients with COVID-19. *Lancet Respir Med*. Nisan 2020;8(4):e20.
10. Zhang JJ, Dong X, Cao YY, Yuan YD, Yang YB, Yan YQ, vd. Clinical characteristics of 140 patients infected with SARS-CoV-2 in Wuhan, China. *Allergy*. Temmuz 2020;75(7):1730-41.
11. Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, vd. Clinical Characteristics of Coronavirus Disease 2019 in China. *N Engl J Med*. 30 Nisan 2020;382(18):1708-20.
12. Simons D, Shahab L, Brown J, Perski O. The association of smoking status with SARS-CoV-2 infection, hospitalization and mortality from COVID-19: a living rapid evidence review with Bayesian meta-analyses (version 7). *Addiction*. 2021;116(6):1319-68.
13. Leung JM, Yang CX, Tam A, Shaipanich T, Hackett TL, Singhera GK, vd. ACE-2 expression in the small airway epithelia of smokers and COPD patients: implications for COVID-19. *Eur Respir J*. Mayıs 2020;55(5):2000688.
14. Liu W, Tao ZW, Wang L, Yuan ML, Liu K, Zhou L, vd. Analysis of factors associated with disease outcomes in hospitalized patients with 2019 novel coronavirus disease. *Chin Med J (Engl)*. 05 Mayıs 2020;133(9):1032-8.
15. Ojo AS, Balogun SA, Williams OT, Ojo OS. Pulmonary Fibrosis in COVID-19 Survivors: Predictive Factors and Risk Reduction Strategies. *Pulm Med*. 2020;2020:6175964.
16. Guan WJ, Liang WH, Zhao Y, Liang HR, Chen ZS, Li YM, vd. Comorbidity and its impact on 1590 patients with COVID-19 in China: a nationwide analysis. *Eur Respir J*. Mayıs 2020;55(5):2000547.

17. Lippi G, Henry BM. Active smoking is not associated with severity of coronavirus disease 2019 (COVID-19). *Eur J Intern Med.* Mayıs 2020;75:107-8.
18. Changeux JP, Amoura Z, Rey FA, Miyara M. A nicotinic hypothesis for Covid-19 with preventive and therapeutic implications. *C R Biol.* 05 Haziran 2020;343(1):33-9.
19. Caly L, Druce JD, Catton MG, Jans DA, Wagstaff KM. The FDA-approved drug ivermectin inhibits the replication of SARS-CoV-2 in vitro. *Antiviral Res.* Haziran 2020;178:104787.
20. Krause RM, Buisson B, Bertrand S, Corringer PJ, Galzi JL, Changeux JP, vd. Ivermectin: a positive allosteric effector of the alpha7 neuronal nicotinic acetylcholine receptor. *Mol Pharmacol.* Şubat 1998;53(2):283-94.
21. Kayhan Tetik B, Gedik Tekinemre I, Taş S. The Effect of the COVID-19 Pandemic on Smoking Cessation Success. *J Community Health.* Haziran 2021;46(3):471-5.
22. Gallus S, Scala M, Possenti I, Jarach CM, Clancy L, Fernandez E, vd. The role of smoking in COVID-19 progression: a comprehensive meta-analysis. *Eur Respir Rev Off J Eur Respir Soc.* 31 Mart 2023;32(167):220191.