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Association of smoking type, status, number times of smoking and their effect on infected people by COVID-19

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Abstract

Background: Smoking has a significant role in weakening the pulmonary immune system function. Infection with infectious agents can cause more serious outcomes in the pulmonary system among people who smoke than those who do not.

Objective: To show the relationship between smoking and the progression of the infectious COVID-19.

Patients and Methods: A questionnaire the following data was gathered from both sexes using a Google form: age, smoking status, smoking habits, blood type, and medical conditions.

Results: Results: Statistical analysis indicated that there are highly significant differences between males and females, and older patients included in the study who are infected and non-infected with COVID-19 ($P < 0.01$), and there are significant differences among blood groups ($P < 0.05$). In regard to smoking status, there are highly significant differences in the disease severity between the never-smoking group compared to current smokers ($P \leq 0.01$, $P \leq 0.05$), respectively. This differences increase with the number of times of smoking, and there is no significant difference between types of smoking between infected and non-infected groups with COVID-19. Statistical analysis indicated that there are highly significant differences in the disease severity in the male and female groups who have not smoked compared to former smokers ($P < 0.01$).

Conclusion: Recent studies have reported the real role of the virus in the health status of smokers. However, this study showed no strong relationship between the virus and smokers.

Keywords: COVID-19, Smoker, Shisha, (e-cigarettes), Cigar

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Introduction

The coronavirus pandemic began in December 2019 and has rapidly spread globally [1]. The causative agent of this disease is SARS-CoV- 2, which belongs to the subgenus Sarbecovirus (Beta-CoV lineage B), which has a significant role in the development of the respiratory tract symptoms by attachment to the ACE2 receptors which are found in the alveolar epithelial cells, the symptoms varying from an infection without symptoms to a serious case of acute respiratory distress syndrome (ARDS) [2]. Corona viruses have spike

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proteins which facilitate entry into the alveolar epithelial cells [3].

Smoking and vaping increase the risk and severity of pulmonary infections because of damage to upper airways and a decrease in pulmonary immune function by causing cytokine release, which leads to increased mucoid secretion, reduced mucociliary clearance, and finally causes damage to the epithelial wall [4]. Shisha is a method of smoking tobacco invented in the 16th century by a physician named Hakim Abul-Fath Gilani. The purpose of the device was to pass smoke through water in an attempt to 'purify' the smoke, an unproven concept that has been repeatedly questioned by the medical community [5].

As a result, the smoke contains components from both tobacco and coal. These include polycyclic aromatic hydrocarbons (PAH), volatile aldehydes, CO, nitric oxide (NO), nicotine, furans, and nanoparticles [6]. Nicotine levels increase significantly from 2 to 6 ng/ml after smoking shisha for five minutes. Increasing nicotine levels have been shown to induce increases in heart rate and may contribute to various cardiovascular diseases [7]. The purpose of this study is to summarize the key points that distinguish the various types of smoking that may be a risk factor for COVID-19, as well as the data from observational studies and reviews on the connections between smoking history and COVID-19.

Patients and Methods

A Google forma questionnaire was designed to collect information from different groups of Iraqi people on an online way during the period ranging between February and March of 2022. This study included 232 individuals (males and females) (133) confirmed COVID-19 cases by PCR technique, and (99) not infected with COVID-19, whose participation in full the questionnaire. Some information was gathered from each person, including the sex, age, smoking status, type of smoking, and comorbidities of the people contributing to this study according to a questionnaire online prepared previously.

Statistical Analysis

In this study, the Statistical Analysis System-SAS (2012) program was used to detect the effect of different factors on study parameters. A chi-square test was used to compare percentages (0.05 and 0.01 probability) in this study. This study was performed to determine the association between smoking type, status, number of times smoked and their effect on infected people by COVID-19.

Results

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Table (1): Characteristics of cohorts included the study

Cohorts included the study	All participant-no. (%)	Confirmed COVID-19 cases (PCR) - no. (%)	No COVID-19-no. (%)	P-value
Participants no. (%)	232 (100%)	133 (57%)	99 (43%)	0.0001 **
Male no. (%)	137 (59%)	79 (34%)	58 (25%)	0.0084 **
Female no. (%)	95 (41%)	54 (23%)	41 (18%)	0.0097 **
Age	All participant	Confirmed COVID-19 cases (PCR) - no. (%)	No COVID-19	
20-29	76 (32%)	45 (19%)	31 (13%)	0.0091 **
30-39	77 (33%)	46 (20%)	31 (13%)	0.0098 **
40-49	79 (34%)	43 (18%)	36 (16%)	0.0261 *
Blood group—no. (%)	All participant	Confirmed COVID-19 cases (PCR) - no. (%)	No COVID-19	
A- no. (%)	53 (22%)	24 (10%)	29 (12%)	0.0498 *
B- no. (%)	58 (25%)	38 (16%)	20 (9%)	0.0355 *
AB- no. (%)	55 (23%)	33 (14%)	22 (9%)	0.0361 *
O- no. (%)	66 (28%)	39 (16%)	27 (12%)	0.0244 *
Comorbidities-no (%)	All participant	Confirmed COVID-19 cases (PCR)- no. (%)	No COVID-19	
Hypertension	36 (100%)	22 (61.1%)	14 (38.8%)	0.0001 **
Diabetes mellitus	24 (100%)	12 (50%)	12 (50%)	0.0001 **
Congestive heart failure	21 (100%)	13 (62%)	8 (38%)	0.0001 **
Recurrent infection with COVID-19	133 (57%)	32 (24%)	101 (76%)	0.0001 **
* ($P \leq 0.05$), ** ($P \leq 0.01$), NS: Non-Significant.				

Statistical analysis indicated that men were slightly more affected by COVID-19 than women, and there were highly significant differences between males and females included in this study who were infected and non-infected with COVID-19 ($P < 0.01$). Also, there are highly significant differences between age groups (20-29, 30-39) compared with age groups (40-49) ($P \leq 0.01, P \leq 0.05$) respectively. According to the ABO group system, there were significant differences among all groups that were infected and non-infected with COVID-19 ($P < 0.05$).

In regard to comorbidities, there were highly significant differences among all groups that were infected and non-infected with COVID-19 ($P < 0.01$).

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Table (2): Comparison of the studying groups in the confirmed COVID-19 cases (PCR) and no COVID-19

Studying groups / Smoking status	All participant no. (%)	Confirmed COVID-19 cases (PCR) no. (%)	No COVID-19	P-value
Never smoking	155 (66%)	90 (38%)	65 (28%)	0.0018 **
Former smokers	60 (25%)	36 (15%)	25 (10%)	0.0261 *
Current smokers	16 (7%)	8 (4%)	8 (3%)	0.419 NS
Type of smoking	All participant—no. (%)	Confirmed COVID-19 cases (PCR)—no. (%)	No COVID-19	P-value
Shisha	21 (9%)	11 (5%)	10 (4%)	0.172 NS
Vape (e-cigarettes)	21 (9%)	10 (4%)	11 (5%)	0.172 NS
Cigar	35 (15%)	23 (10%)	12 (5%)	0.071 NS
No. of times of smoking	All participant—no. (%)	Confirmed COVID-19 cases (PCR)—no. (%)	No COVID-19	P-value
Never smoking	155 (66%)	90 (38%)	65 (28%)	0.0018 **
One time	76 (100%)	65 (86%)	11 (14%)	0.0001 **
3 times	76 (100%)	73 (96%)	3 (4%)	0.0001 **
More than 3 times	76 (100%)	60 (79%)	16 (21%)	0.0001 **
* (P≤0.05), ** (P≤0.01), NS: Non-Significant.				

In regard to smoking status, there are highly significant differences in the disease severity between never-smoking individuals compared to current smokers (P≤0.01, P≤0.05) respectively. There is no significant difference between the types of smoking between infected and non-infected groups with COVID-19. According to the times of smoking, there are highly significant differences between the never-smoking group and other smoking individuals, who are infected and non-infected groups with COVID-19.

Table (3): Comparison the clinical picture of COVID -19 between male smokers and female smokers

Clinical Picture of COVID -19 at Onset	Never /Current smokers			Former smokers		
	Male	Female	P-value	Male	Female	P-value
Fever	42 (100%)	35 (92.11%)	0.079 NS	48 (94.12%)	3 (100%)	0.438 NS
Headache	39 (92.86%)	38 (100%)	0.078 NS	51 (100%)	3 (100%)	1.00 NS
Diarrhea	13 (30.95%)	17 (44.74%)	0.0392 *	34 (66.67%)	3 (100%)	0.0036 **
Cough	22 (52.38%)	28 (73.68%)	0.0095 **	45 (88.24%)	2 (66.67%)	0.0097 **
Vomiting	21 (50.00%)	30 (78.95%)	0.0072 **	20 (39.22%)	1 (33.33%)	0.207 NS
Myalgias/ Joint pain	40 (95.24%)	33 (86.84%)	0.071 NS	40 (78.43%)	3 (100%)	0.0089**
Dyspnea	16 (38.10%)	29 (86.84%)	0.0001 **	29 (56.86%)	2 (66.67%)	0.072 NS
Rhinitis	13 (30.95%)	20 (52.63%)	0.0074 **	38 (74.51%)	2 (66.67%)	0.0819 NS
* (P≤0.05), ** (P≤0.01), NS: Non-Significant.						

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According to the clinical picture of COVID-19, statistical analysis indicated that there were highly significant differences in the disease severity in the male and female groups who had never smoked compared to former smokers ($P < 0.01$).

Discussion

The immune pulmonary system is suppressed by smoking, and this increases the risk factor for other infectious diseases and leads to serious outcomes among people who become infected. Smoking and cigarettes cause an increased risk and severity of pulmonary tract infections because of damage and a decrease in pulmonary immune function of the upper airways [8].

This study showed that infection with COVID-19 is slightly more prevalent in males than in females; that may explain why men are more susceptible to infection because they spend more time in public areas.

Other studies focus on the immune system (innate and adaptive) and show some differences in the X human chromosome and sex hormones between males and females (9), specifically in men who have a high mortality rate and are more infected than females. In general, females have a stronger, innate and adaptive immune system than males, and they are also have resistant to virus infections [10].

In regard to the age group, this study showed highly significant differences between age groups and those whose comorbidities ranged between (20–29, 30-39) compared with age groups 40–49 ($P \leq 0.01$, $P \leq 0.05$) respectively. According to the age groups, previous studies showed that about fifty percent of the infected patients ranged in age from 20–50 years. Those over the age of 80 years noted that the disease gradually decreases until it reaches about 1 percent. This may explain why adult males spend a long time exposing themselves to the threat of infection [11].

According to previous research, severe COVID-19 specifically targets the older population over 65 years with comorbidities, in whom smoking rates are approximately 3–5-fold lower than in the general population. Thus, background smoking rates in the severe COVID-19 susceptible subgroups may be much lower than the general smoking rates of the population [1]. According to the blood groups, this study concluded significant differences among all groups who were infected and non-infected with COVID-19 ($P < 0.05$). Earlier studies showed that it needed additional results to improve understanding of the relationship between COVID19 and ABO blood group [12].

Previous clinical studies have found a correlation between hypertension and an increased risk of mortality in SARS and Middle East respiratory disease patients. These findings concur with this study's findings that showed highly significant differences between all groups with and without COVID-19 infection ($P < 0.01$). The association between hypertension and severe COVID-19 may be the underlying cause of the imbalance in cytokine levels [13].Shenoy has demonstrated that diabetic patients who are infected with COVID-19 have higher levels of pro-inflammatory cytokines released than non-diabetics [14], making them more susceptible to severe COVID-19, ARDS, and chronic diseases with COVID-19.

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COVID-19 primarily targets lung epithelial cells, causing viral pneumonia and acute respiratory distress syndrome (ARDS), especially in elderly patients [15]; these results matched the results of this study. Previous reports showed that smoking increased the progression of COVID-19. According to other studies, people who have smoked previously or currently are substantially more likely to have severe COVID-19. Similar findings from earlier studies have been reported [16].

The present study's findings differ from earlier research, which found that illness severity increased when a person had a history of smoking. According to findings from five earlier studies, there is no discernible difference in COVID-19 severity between patients with and without a history of smoking. This contradicts the findings of the earlier studies that were presented. Additionally, a Lippi meta-analysis [17] on Chinese patients found no association between smoking and the severity of COVID-19, and a different meta-analysis showed that active smoking is not a risk factor for hospitalization [18].

According to a recent meta-analysis [19], active smokers also had a higher risk of death and serious complications. This appears to be as a result of former smokers having longer exposure times or associated diseases like COPD brought on by smoking. Although not formally reported, hookah smoking (like e-cigarettes) could disproportionately increase COVID-19 in youth. Furthermore, Middle East respiratory syndrome coronavirus was also thought to be transmitted by waterpipe smoking [20]. Guan [21] demonstrated in a recent study that smokers were more infected with COVID-19 than non-smokers . While, Liu [22] showed that smoking history was associated with the progression of COVID-19. Contrary to these findings, a study by Huang [23] showed that current smoking history was not associated with the need for intensive care unit care. Similar findings were observed in subsequent larger observational studies [24-26].

Nicotine has an important role in the regulation of ACE 2 receptors' immunomodulation, and this is shown in the fact that ex-smokers have a 25% increase in pulmonary ACE2 receptor expression over non-smokers. These findings revealed that former smokers were more susceptible to getting viruses and allowed them to enter their lungs [27]. Smoking has emerged as an independent risk not only for transmission but also for the severity of coronavirus disease 2019 (COVID-19).

Similarly, hookahs are ideal for transmission and may exacerbate the risk of severe COVID-19 through shared use. They have long, difficult-to-clean pipes and a cold water reservoir, ideal for the transmission of SARS-CoV-2 [28]. Furthermore, hookah tobacco smoke contains several hazardous chemicals that injure the respiratory lining and predispose the smoker to viral infections, tuberculosis and other infectious diseases, and the coronavirus is no exception. Recent studies conclude that COV-MERS have a high risk of infection and mortality rate in smokers [29]. This study focuses on the types of smoking, including hookah, vape, and cigars, and there is no significant difference between types of smoking between infected and non-infected groups with COVID-19.

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Smoke from shisha and cigarettes harms pulmonary cells and alveoli, affects the surrounding tissue, and reduces the lungs' ability to fight off infections. In fact, users of shisha have been found to maximize lung inflammation and cell damage after just one session [30]. Thus, smoking decreases the lung's capacity to absorb O_2 and release CO_2, causing the mucus to build up and cause persistent coughing and breathing problems [31]. Smoking tobacco is known to make people more susceptible to infections in a variety of ways, including by changing the hosts' mechanical (mucociliary) and immune responses and perhaps increasing the virulence of viruses and bacteria [8].

Interestingly, tobacco smoking has a truly significant role on the immune system and activation of inflammation; the structural and Ig alterations in chronic bronchitis, COPD and emphysema patients represent ideal attachment areas for bacterial and viral infections [32]. In England, current smokers and long-term ex-smokers had significantly greater odds of self-reported COVID-19 compared with never smokers after controlling for a number of potential confounding variables. There was sufficient evidence to rule out a large association of self-reported COVID-19 with NRT use and a medium association with e-cigarette use [33]. Compared with never smokers, recent ex-smokers had significantly lower odds of washing their hands before touching their faces. Recent ex-smokers also had greater odds of self-reported COVID-19, but not significantly so [33]. These findings suggest that one of the two explanations other than infection is likely to account for the lower than expected smoking rates among those hospitalized for COVID-19. Based on recent advice from the World Health Organization on COVID-19 and hookah use, we suggest that hookah or waterpipe smoking should be regulated to prevent COVID-19 transmission through hookah smoking [34].

Conclusions

The results of this study showed a slight relationship between smoking and symptoms of infection with COVID-19 and highly significant differences in the disease severity (COVID-19) in the male and female groups whose members had never smoked compared to former smokers ($P < 0.01$).

Recommendations

According to the results of this study and the previous studies, coronavirus is a respiratory disease caused by the SARS-CoV-2 virus; tobacco smoking was considered an a priori risk factor for SARS-CoV-2 infection and poor COVID-19 disease. Based on recent advice from the World Health Organization on COVID-19 and hookah use, previous studies suggest that shisha should be regulated to prevent COVID-19 transmission through its smoking. Our country should place restrictions on different types of smoking to minimize the risk of infection with these serious viruses.

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Ethical clearance: Ethical approval was obtained from the College of Medicine / University of Diyala ethical committee for this study.

Conflict of interest: Nil

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الارتباط بين نوع التدخين وعدد مرات التدخين وتأثيره على الأشخاص المصابين بـالكوفيد ١٩

مها مصطفى عاصم[١] ، داليا فالح احمد[٢] ، مريم بدر محمود[٣]

الملخص

خلفية الدراسة: للتدخين دور كبير في إضعاف مناعة الجهاز التنفسي . يمكن أن تكون نتائج العدوى بالعوامل المعدية انتهازية و أكثر خطورة في الجهاز التنفسي بين الأشخاص الذين يدخنون أكثر من غيرهم.

اهداف الدراسة: تهدف هذه الدراسة إلى إظهار العلاقة بين التدخين وتطور العدوى بفيروس COVID-19.

المرضى والطرائق: تم تصميم نموذج استبيان من Google وتم جمع المعلومات من كلا الجنسين بما في ذلك: العمر ، عدد مرات التدخين ، نوع التدخين ، فصيلة الدم ، والأمراض المصاحبة.

النتائج: أشار التحليل الإحصائي إلى وجود فروق ذات دلالة إحصائية بين الذكور والإناث ، والمرضى الاكبر سنا فقد شملت الدراسة المرضى المصابين وغير المصابين بـ COVID-19 ($P\leq0.01$)، وهناك فروق ذات دلالة إحصائية بين فصائل الدم ($P<0.05$). فيما يتعلق بحالة التدخين ، توجد فروق ذات دلالة إحصائية كبيرة في شدة المرض بين مجموعة غير المدخنين مقارنة بالمدخنين ($P\leq0.01$، $P\leq0.05$) على التوالي ، وتزداد هذه الفروق مع زيادة عدد مرات التدخين ، ولا توجد فروق معنوية بين أنواع التدخين بين المجموعات المصابة وغير المصابة بـ COVID-19. وفقًا للصورة السريرية لـ COVID -19 ، أشار التحليل الإحصائي إلى وجود فروق ذات دلالة إحصائية في شدة المرض في مجموعات الذكور والإناث الذين لم يدخنوا أبدًا مقارنة بالمدخنين السابقين ($P<0.001$).

الاستنتاجات: COVID-19 هو فيروس انتهازي له دور حيوي في صحة الإنسان ، أشارت الدراسات الحديثة إلى الدور الحقيقي للفيروس في الحالة الصحية للمدخنين ، وأظهرت هذه الدراسة عدم وجود علاقة قوية بين الفيروس والمدخنين.

الكلمات المفتاحية: كوفيد-١٩، مدخن ، شيشة ، (سجائر إلكترونية) ، سيجار

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