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Original Article

Association between the Clinical Presentation and Outcome among Smokers vs Non- Smokers COVID-19 Patients in Kirkuk Health Care Centers/Iraq

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Abstract

Background: Smoking is regarded as one of the most important reasons of diseases and lead to eight million deaths all over the world. Both factors including corona virus disease 2019 (COVID-19) infection related to smoking can primarily affect the lungs. COVID-19 is mainly transmitted through the respiratory tract (saliva), and the smoker group may be at a higher risk of contracting the virus because of impaired immune systems, reduced lung function, susceptible hygiene habits, and cross-infection. **Objectives:** The study aimed to determine if there is any association between the current smoking and the clinical severity and outcomes of disease among patients suffering from COVID-19 in Kirkuk city. **Materials and Methods:** Retrospective case series include 973 adult (≥18 years old) patients (525 male and 448 female) with confirmed COVID-19. The studied patients were diagnosed by reverse transcriptase-polymerase chain reaction (PCR) by taking a swab from a nasopharyngeal region during the period from July 2020 to October 31, 2021. The data were collected from patients on the first visit to health services system (Private Clinic, AL-shifaa Hospital, and Kirkuk General Hospital). The diagnosis of patients with COVID-19 pneumonia was based on symptoms. Presence of infiltration on chest X-ray or high-resolution CT scan with positive PCR swab for SARS-CoV-2 was found. The total number of patients included in the study was 974 (646 smokers and 328 non-smokers). A special questionnaire form was prepared for this purpose and distributed after receiving the verbal consent from them, and the data were collected by interviewing with them. **Results:** This study shows that the most affected age group between smokers and non-smokers was 50–59 years (47.35%–49.8%), respectively, with *P* value = 0.444. Regarding the sex, males from smoker group constitute a higher percentage (75.6%), in comparison to non-smoker group in which the females constitute a higher percent (56.2%) with a *P*-value = 0.000. Also the study reveals that the smoker group shows a high percent among patients with hypertension (33.8%), in comparison to the non-smoker group in which diabetes mellitus was the most comorbid disease (37.9%) with a *P*-value = 0.000. **Conclusion:** The study recommended that physicians can provide more attention to the smoking history in patients with COVID-19 infection, and more future advanced research is aimed to explain the mechanisms that decrease this risk.

Keywords: Association, COVID-19 outcome, health care centers, smokers

Introduction

Corona virus disease 2019 (COVID-19), usually caused by severe acute respiratory syndrome corona virus 2 abbreviated as (SARS-CoV-2), was first mentioned in Wuhan, China, during December 2019. It later on spread all over the world, leading to a global pandemic.[1,2]

This highly infectious disease has thus far infected more than 23.4 million people all over the world and led to the death of approximately 808000 patients, resulting in high case fatality rate (CFR), which varies between 0.7% and 12.7% (average: 3.4%).[3]

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COVID-19 mainly affects the lung epithelial cells, causing acute respiratory distress syndrome (ARDS), and viral pneumonia is concentrated in elderly patients. For that reason, the mortality rate is much higher in patients with at least one accompanying comorbid disease and in the elderly age groups.[4]

Two recent studies showed that 29%–31% males and 3%–4% females of Iraq population are active smokers. Although the reported percentage of cigarette smoking in Iraq population is not the highest in the Arab countries, we believe the incidence is higher than that reported.[5]

However, it has been mentioned that COVID-19 leads to more progression effects in patients suffering from chronic – obstructive pulmonary diseases (COPD).[5] Given that smoking can play an essential role in the etiopathogenesis of this disease, and it may have the same characteristic effect on signs and symptoms. The definitive relation between the two variables (smoking and COVID-19) prognosis has yet to be established and documented.[6]

In a recent updated meta-analysis study of smoking and COVID-19 severity, it was shown that smoking does not increase the severity of COVID-19 (odds ratio [OR], 1.69; 95% CI: 0.41–6.92).[7]

Various comorbidities represent risk factors for severe COVID-19. The outcome of smoking on COVID-19 severity has been previously mentioned in several meta-analyses studies limited by poor methodology and small sample sizes.[8]

The harms of tobacco use are well-known. Tobacco leads to more than 8 million deaths yearly from cancers, diabetes, hypertension, cardiovascular diseases, and lung disorders. Smoking tobacco is also a reportable risk factor for death and severe disease occurrence like cancers, diabetes, and hypertension and death from many respiratory infections. In the pandemic of COVID-19, questions have been asked about clinical impacts for smokers, and if they have an equal chance of infection susceptibility, and if nicotine has any biological role on the SARS-CoV-2 virus (the virus that causes COVID-19).[8]

Most people who develop COVID-19 fully recover, but current evidence suggests that approximately 10%–20% of people experience a variety of mid- and long-term effects after they recover from their initial illness. These mid- and long-term effects are collectively known as post COVID-19 condition or "long COVID." Post COVID-19 condition is usually diagnosed three months after COVID-19. This allows the healthcare provider (HCP) to rule out the normal recovery process after the illness. The symptoms and effects last for at least two months. We are still learning about COVID-19 and post COVID-19 condition. (The most common symptoms of post COVID-19 condition include fatigue, shortness of breath or difficulty breathing, memory, sleep or concentration problems, persistent cough, chest pain, trouble speaking, muscle aches, loss of smell or taste, depression or anxiety, and fever).[9]

Patients with any history of smoking are more dangerous for developing a severe COVID-19 and lead to more progressive outcome in-hospital admission. In the absence of the preventative and supportive strategies, the current targeted therapies to reduce morbidity and mortality in former and current smokers are crucial.[10]

Recently, one new updated clinical trial study was conducted to detect the effects of nicotine which has been announced, but still no trial registration record was found as of May 12, 2020, regarding the following parameters:

1) risk of infection by SARS-CoV-2
2) hospitalization with COVID-19
3) severity of COVID-19 outcomes among hospitalized patients such as admission into intensive care units (ICU), use of ventilators, and death.[10,11]

The goal of the study was to verify any correlation between the current smoking and the clinical severity and outcomes of disease among a sample of patients infected with COVID-19 in Kirkuk city.

Materials and Methods

Official permission was taken from Kirkuk health directorate before starting the study.

Study design and setting

Retrospective case series include 973 adults (≥18 years old) patients (525 male and 448 female) with confirmed COVID-19. The patients were further diagnosed by a special test called reverse transcriptase-polymerase chain reaction (RT-PCR) by taking a nasopharyngeal swab specimens which was done in Private Clinic, AL-shifaa Hospital, and Kirkuk General Hospital among patient suffering from COVID-19.

Sampling techniques

A descriptive study was conducted among patients suffering from COVID-19. The data were collected from them on the first visit to health services system including Private Clinic, AL-Shifaa Hospital, and Kirkuk General Hospital.

Diagnosis of COVID-19 pneumonia

The diagnosis of patients with COVID-19 pneumonia was based on (1) symptoms present, (2) the infiltration spaces found on chest X-ray or high-resolution CT scan (HRCT) with a positive polymerase chain reaction swab for SARS-CoV-2. Patients admitted to hospital with possible COVID-19 had HRCT upon admission. All images of CT scan were reviewed and analyzed by a

specialist radiologist. The severity score was dependent on the percentage of lung involvement for each patient.[12]

A questionnaire form was prepared for this purpose and distributed after obtaining the verbal agreement from them, and the data were collected by interviewing them after giving a full detail of the study aim.

Patient demographics, symptoms, comorbidities, blood test results, radiographic findings, clinical course, and hospital mortality were collected during hospital stay and manually extracted from nonelectronic health records, and supplementary efforts were done to directly contact patients (or their relatives) to confirm outcome diagnosis. 509 out of 700 patients/relatives were actually successfully reached by phone communication. The age of them ranged from 18 to 85 years.

Study period

The period of the study was started from June 30, 2020, and finished by November 30, 2021.

Data collection tool

A certain specific questionnaire form was designed by the investigator reviewing all current related updated literatures from new books correlated to the questionnaire items included four main parts:

Part-1—Demographic characteristics like sex, age, residence, occupation, educational level, and comorbidities.

Part-2—Includes the main symptoms related to COVID-19 disease, complete laboratory investigation, body mass index (BMI), chest X-ray, O_2 saturation.

Part-3—Complete detail information about the final outcome of the disease in both groups (smokers and non-smokers) regarding the recovery, death, and admission to respiratory care unit for mechanical ventilation).

All the statements are with either yes and no answer, the number and percent will be calculated.

The statistical test was χ^2 , which was used to assess the relation between the studied indicated variables, $P < 0.05$ was regarded significant, and OR was calculated to determine the clinical relationship between the smoking and the disease severity.

Relative risk (RR) was determined between two groups and the results above 1 was regarded as a significant value.

Ehical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients. Verbal and analytical approval was taken before sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to the document number 1.

Results

Table 1 shows that the most affected age group between smokers and non-smokers was 50–69 years (47.3%–49.8%), respectively, with *P* value = 0.444.

Regarding the sex, males from the smoker group constitute a higher percentage (75.6%), in comparison to non-smoker group in which the females constitute a higher percentage (56.2%) with a *P*-value = 0.000.

With regard to the occupation, the most affected group among smokers was earners (39.7%) in comparison to housewives from non-smoker group (45.9%) with a *P*-value = 0.000.

For residence, both groups (smokers and non-smokers) show a high percent among those living in the city (72.9%, 77.7%), respectively, with a *P* value = 0.094.

Regarding the educational level and its effect on disease occurrence, both groups (smokers and non-smokers) show a high percent among those who are illiterate (46.6%, 46.4%), respectively, at a *P* value = 0.953.

With regard to the main symptoms, fatigue and SOB were more prevalent symptoms among smoker groups (75.6%, 97.5%) with a *P* value = 0.001, while fever and SOB are the most present symptoms among non-smokers group (86.1%, 96.5%), respectively, with a *P* value = 0.409.

Concerning BMI, both groups (smoker and non-smoker) show a high percentage among those who are overweight (25–29.5), (43.9%, 42.4%) with a *P* value = 0.658.

Regarding the laboratory diagnosis, both groups (smokers and non-smokers) show a high percent among those with the ferritin level above 300 mg (64.3%, 10.5%), respectively, with a *P* value = 0.002, with normal white blood cells in both groups (51.3%, 46.9%), but the lymphocytes show a low percent in non-smoker groups (10.5%), in comparison to smoker group in which the lymphocyte show a normal level (41.2%) with a *P* value = 0.000.

Concerning the comorbidities, the smoker group shows a high percent among patients with hypertension (33.8%), in comparison to non-smoker group in which diabetes mellitus was the most comorbid disease (37.9%) with a *P* value = 0.000.

The main diagnostic method for disease was the nasal swab among the smoker group (42.9%), while it was 39.9% for chest X-ray and nasal swab in the non-smoker group with a *P* value = 0.542, and the main CT chest findings show more than 25.5% infiltration in the lungs in both smokers and non-smokers (41.5%, 32.2%), respectively, with a *P* value = 0.259.

Table 1: Demographic characteristic of the study sample

Characteristic	All patients, *N* = 974	Non-smoker, *N* = 646	Current smoker, *N* = 328	*P* value
Patients age				
<30	53	33 (5.1%)	20 (6.1%)	0.520
30–49	247	158 (24.5%)	89 (27.1%)	0.364
50–69	477	322 (49.8%)	155 (47.3%)	0.444
≤70	197	133 (20.6%)	64 (19.5%)	0.692
Male	531	283 (43.8%)	248 (75.6%)	0.000
Female	443	363 (56.2%)	80 (24.4%)	
Occupation				
Housewife	371	297 (45.9%)	74 (22.5%)	0.000
Earner	285	155 (23.9%)	130 (39.7%)	0.000
Student	14	8 (1.3%)	6 (1.8%)	0.464
Retired	96	61 (9.5%)	35 (10.7%)	0.543
Employee	208	125 (19.4%)	83 (25.3%)	0.032
Residence				
City	741	502 (77.7%)	239 (72.9%)	0.094
Rural	233	144 (22.3%)	89 (27.1%)	
Educational level				
Illiterate	453	300 (46.4%)	153 (46.6%)	0.953
Primary school	234	151 (23.5%)	83 (25.4%)	0.505
Secondary school	148	102 (15.7%)	46 (14.0%)	0.468
Postgraduate	139	93 (14.4%)	46 (14.0%)	0.875
Symptoms				
Fever	830	556 (86.1%)	274 (83.5%)	0.292
Rigor	620	399 (61.7%)	221 (67.3%)	0.085
Cough	733	481 (74.4%)	252 (76.8%)	0.480
Loss of smell	577	377 (58.3%)	200 (60.9%)	0.009
Loss of taste	593	379 (58.6%)	214 (65.2%)	0.046
Abdominal pain	517	319 (49.3%)	198 (60.3%)	0.001
Frequent bowel motion	455	298 (46.1%)	157 (47.8%)	0.607
SOB	944	624 (96.5%)	320 (97.5%)	0.409
Fatigue	719	471 (72.9%)	248 (75.6%)	0.001
Bone pain	621	404 (62.5%)	217 (66.1%)	0.266
Body mass index				
Under WT <18	0	0 (0.0%)	0 (0.0%)	
Normal (18–24.9)	313	202 (31.3%)	111 (33.9%)	0.417
Over WT (25–29.5)	418	274 (42.4%)	144 (43.9%)	0.658
Obese (30–34.9)	209	150 (23.2%)	59 (17.9%)	0.060
Above = 35	34	20 (3.1%)	14 (4.3%)	0.346
Laboratory				
Increased D-dimer	574	374 (57.9%)	200 (60.9%)	0.355
Increased CRP	646	440 (68.1%)	206 (62.8%)	0.079
Ferritin >300	686	475 (10.5%)	211 (64.3%)	0.002
WBC count				
Normal	471	303 (46.9%)	168 (51.3%)	0.202
High	406	275 (42.6%)	131 (39.9%)	0.431
Low	97	68 (10.5%)	29 (8.8%)	0.405
Lymphocyte count				
Normal	307	172 (26.6%)	135 (41.2%)	0.000
High	262	171 (26.5%)	91 (27.7%)	0.699
Low	405	303 (46.9%)	102 (31.1%)	0.000
Comorbidities				
Hypertension	331	220 (34.1%)	111 (33.8%)	0.946
Cardiovascular disease	175	119 (18.4%)	56 (17.1%)	0.604
Diabetes melitus	298	245 (37.9%)	53 (16.2%)	0.000

Table 1: Continued

Characteristic	All patients, *N* = 974	Non-smoker, *N* = 646	Current smoker, *N* = 328	*P* value
Chronic obstructive pulmonary disease	117	78 (12.1%)	39 (11.8%)	0.933
Renal disease	184	127 (19.6%)	57 17.5%)	0.930
Cancer	23	11 (1.7%)	12 (3.6^)	0.057
Diagnosis				
Computerized Tomography (CT scan)	219	149 (23.2%)	70 (21.3%)	0.542
Swab	380	239 (36.9%)	141 (42.9%)	0.070
Both	375	258 (39.9%)	117 (35.7%)	0.008
Computerized tomography (CTscan)				
Normal	126	88 (13.6%)	38 (11.6%)	0.370
<5%	132	92 (14.2%)	40 (12.2%)	0.377
5%–25%	344	208 (32.2%)	136 (41.5%)	0.259
26%–50%	201	137 (21.2%)	64 (19.5%)	0.536
51%–75%	140	100 (15.5%)	40 (12.2%)	0.167
>75%	22	21 (3.3%)	10 (3.0%)	0.865
O_2 saturation				
Normal	425	260 (65.9%)	165 (50.3%)	0.004
<95%	40	30 (6.3%)	10 (3.0%)	0.235
90%–80%	246	93 (38.2%)	153 (46.7%)	0.000
60%–79%	263	263 (40.7%)	0 (0.0%)	0.000

Lastly, with regard to O_2 saturation, the smoker group shows a normal percent (50.3%) in comparison to another group which show that there is 80–90% O_2 saturation with a *P* value = 0.000.

RR is more than 1 and that means that there is a statistical relation between smoking and the final outcome regarding the mechanical ventilation because smoker patients were more having a chance of 1.76 to have a mechanical ventilation during admission to respiratory care unit non-smokers with a *P* value = 0.065.

Discussion

A study was done by Leung *et al.*[13] mentioned that ACE-2 gene expression was upregulated in the airway epithelium of active smokers, and COPD patients therefore the explanation mechanism by which the risk for increasing the severity of severe COVID-19 in smoker patients.

This upregulation (abnormal mechanism) may happen through the α7 subtype of nicotine acetylcholine receptors (α7-nAChR) in people who consequently consume nicotine and smoking, for that reason, α7-nAChR antagonists (e.g., α-conotoxin and methyl lycoconitine) may be used to support with smoking cessation, this should be investigated and followed up for their potential impact and outcome on ACE-2 expression till the entry of SARS-CoV-2 into the host. Furthermore, recent updated studies explained that ACE-2 and α7-nAChR may be having potential therapeutic targets that need more advanced investigation.

The most frequent comorbidities include chronic kidney disease, hypertension, cardiovascular disease, and diabetes, which are associated with increased risk for severe COVID-19. At the same time, COPD is also related to a poor, bad clinical progression and deteriorated outcomes in spite of treatment, and it was documented that the risk of severe COVID-19 was seen 4 times much higher in patients with preexisting COPD than in patients not having COPD.

These results can be explained by diminished respiratory function and capacity, chronic and systemic chronic inflammation, and COPD-related respiratory failure in some patients. The COPD prevalence was previously revealed to be 15.2% in active smokers, 7.6% in former smokers, and 2.8% in non-smokers.[14]

Jessica *et al.*[15] published a study on the same subject, and the data were collected between March 31, 2020 and February 20, 2021. The final results included hospitalization, ICU admission, mechanical ventilation, mortality, and severity of illness. They found that the smoking correlated for elevation of high risk of hospital admission among 7 of 10 participants and the admission for ICU between 6 out of 9 researches. The 2 of 6 studies needed mechanical ventilation, and illness degree severity in 3 out of 9 studies which is in agreement with the current results finings. Furthermore, the study mentioned that tobacco history led to an elevated risk of pulmonary embolism occurrence among patients suffering from COVID-19.

The use of tobacco was found to be very risky because it cause compound risks related to the development of chronic liver disease (*n* = 1), diabetes (*n* = 1), and cancer (*n* = 2). They concluded that there is a high relationship with a strong evidence that tobacco use increases the

risks of disease severity/progression and mortality among corona patients [Table 2].[15]

Recently, a number of updated observational studies revealed that there is an inverse relationship between SARS-CoV-2 infection (COVID-19) and smoking, causing a (social) media hype and confusion among scientists and to some extent the medical community.[15]

The finding that smoking is not associated with SARS-CoV-2 infection contradicts earlier studies which reported that smokers are more liable to infections in general and to respiratory infections in particular. Smoking is known to increase the risk of infection of both viral and bacterial diseases, such as tuberculosis, the common cold, and influenza. Smoking is a putative risk factor for Middle East respiratory syndrome corona virus infection. Till now, and up to date time, there is no strong evidence study (i.e., evidence based on causal research) that smokers are protected against SARS-CoV-2 infection. Furthermore, there is a continuous evidence that smokers have worse impacts after infection with the virus than non-smoker patients.[16,17]

Naomi *et al.*[18] suggested in their research that it is imperative for any myths about COVID-19 and smoking among the general public are expelled, mainly taking into consideration the growth factors that smokers have worse results once infected. There is still now no suitable solution to the rapid spread of health misinformation through social media, but the important role of primary HCPs can play in mitigating its future harmful effects.

They reported in their paper that the main steps for HCPs activities are

First, according to national guidelines, the primary HCPs can choose to ask patients about their smoking habits during consultations, advise smokers to quit smoking, inform smokers about the health effects of smoking, and offer possible cessation support to all smokers. As face-to-face support cessation may nowadays be restricted, also they can point out the availability of support at a distance, like Health interventions or telephone quitlines.

Second, the another activity of the primary HCPs can inform patients about the dangerous relationship between COVID-19, smoking, and other critical illnesses, for example, by addressing the issue on their posters/television screens or websites in the waiting room.

Third, health misinformation on social media is becoming more prevalent among youth and young adults, and for this reason, the primary HCPs may choose to actively bring up the subject of COVID-19 and smoking in consultations with their ages and advise the non-smokers to never start smoking.[18]

One of the significant recent studies about the correlation of the COVID-19 severity and smoking, a study was conducted[19] to associate the relationship between them during the first initial wave of this epidemic in Malaysia. They established the study since 2020 on clinical records of all real-time polymerase chain reaction (RT-PCR)-diagnosed COVID-19 cases who are smoking, with positive history of comorbidities, typical clinical signs and symptoms, and disease management were reviewed. The severity of the disease was evaluated by the detection of future complications and outcomes of COVID-19 infection. The main statistical interference in this study was busing the logistic regression test which was indicated to verify the linkage between smoking status and COVID-19 disease severity.[19]

They found that smoking groups had a much higher risk of developing COVID-19 complications, like renal injury (OR 1.55; 95% CI 1.10–2.14, ARDS (OR with 1.69; 95% confidence interval [CI] 1.09–2.55), and acute liver injury (OR 1.33; 95% CI 1.01–1.74), in comparison to another similar groups who had never smoked. In spite of this, there were no real differences between the two groups in terms of disease outcome [Table 3].

Table 2: Distribution of study groups according to final outcome after follow-up

Studied	Out come after follow up years		Total	RR	95% CI	*P*-value
	Long COVID-19	Cured				
Smokers	26 (33.7%)	243 (34.4%)	269 (34.3%)	0.976	0.623–1.529	0.968
Non-smoker	51 (66.3%)	464 (65.6%)	515 (65.7%)			
Total	77	707	784			

The table show that the relative risk (RR) between the outcome of two groups (smokers and non-smokers) was 0.976 which is less than one and indicated that there is no statistical relation between the two variables with *P* value = 0.968

Table 3: Frequency distribution of study groups regarding the death occurrence

Studied	Out come after follow up year		Total	RR	95% CI	*P*-value
	Death	Cured				
Smokers	28 (34.5%)	243 (34.4%)	271 (34.4%)	1.008	0.653–1.555	0.972
Non-smokers	53 (65.4%)	464 (65.6%)	517 (65.6%)			
Total	81	707	788			

Shows that the relative risk (RR) between the outcome of two groups (smokers and non-smokers) regarding the death occurrence it was 1.008 which indicated that there is statistical relation between the two variables with *P* value = 0.972

Table 4: The distribution of study sample according to mechanical ventilation out come

Characteristic	Smokers, *N* (%)	Non-smokers, *N* (%)	RR	*P* value
ICU mechanical ventilation	12 (38.7%)	17 (21.5%)	1.76	0.065
ICU-nonmechanical ventilation	19 (61.3%)	62 (78.5%)		

RR = 1.76

In April 2020, the recent reports from WHO show that smoker patients were having a high chance to develop serious diseases from COVID-19 complication than non-smokers.[20] More updated recent articles have shown that smoking is directly related to the future progress of COVID-19 infection, and the currently existing and former smoking are significantly correlated with the higher risk of critical illness.[21] During the past few years ago from influenza epidemics (Mediterranean SARS– Covid-19), smokers were twice more prone to be infected with influenza and had more diagnostic signs and symptoms in comparison to non-smokers, with a high percent mortality rate.[22] Smokers may need mechanical ventilation more often than non-smokers or they may be more likely to die. In a single-center study, Zhao *et al*. found that on-going smoking will lead to an elevated risk of developing severe COVID-19 by approximately twofold (OR = 1.98; 95% CI: 1.29–3.05), and COPD patients who developed severe COVID-19 symptoms had a >4-times fold higher risk (OR = 4.38; 95% CI: 2.34–8.20).[8]

Furthermore, a study was done in Basrah/Iraq by Albazoony[23] to study the fasting as an immune modulator in correlation to the severity and mortality of COVID-19 patients and to detect the effect of smoking on such patients by participating all the polymerase chain reaction-confirmed COVID-19 patients during April, May, and June 2020 in Basrah city.[23]

He found that there was no significant difference in the mean age of patients over the three months including the fasting month, and there was no significant statistical differences in relation to gender, address, or previous medical history [Table 4].[23]

He also mentioned that significantly less severe cases (6.1%) and lower mortality (0.5%) were reported in patients with COVID-19 in the fasting month of Ramadan, as compared with patients who were diagnosed 1 month before and after. He supported his finding by the fact that current smoking was significantly more shown among mild to moderate cases as compared to severe cases. These results may mention that there was a protective role of smoking in COVID-19 patients. The sex and age effect were not significantly different between the two severity groups. The overall smoking (5.3%) is still highly below what is expected in comparison to other previous studies.

Previous studies revealed that 3%–4% females and 29%–31% males of Iraq population are active smokers. With the belief that the incidence are much higher than reported[24,25] in contrast to WHO that revealed the final results of post COVID-19 outcome.[26]

Conclusion

There is strong evidence that tobacco use elevated the risks of mortality and disease severity/progression among COVID-19, and hospitalized patients.

1. Future researches and papers must be done in order to determine the amount of nicotine taken and the number of the total number of cigarettes smoked daily, degree of COPD, and passive exposure.
2. Assessment of the dynamics of reaction between COVID-19 and cigarette smoking and also the impact of different smoking habits and behavior (e.g., mild versus heavy consumption, electronic cigarettes use, and water pipe) on the SARS-CoV-2 transmission.
3. Physicians can provide more time concentrating to the history of smoking and its association with the disease among COVID-19 patients, and further detailed studies may determine the main possible ways of mechanisms that stop or decrease this risk factor.
4 More advance efforts for public health education during the outbreak occurrence, and all smokers can be encouraged to stop tobacco for better health with the promotion of taking vaccination for those people with a positive history of tobacco smoking

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Conflicts of interest

There are no conflicts of interest.

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