Does Smoking and Alcohol Increase the Risk of COVID-19? A Review

Ankit Mahajan1  Ujwal Singh2  Pankaj Kumar3

1 Department of Periodontology and Implantology, H. P. Government Dental College and Hospital Shimla, Shimla, Himachal Pradesh, India
2 Department of Periodontology and Implantology, Private Dental Practitioner, New Delhi, India
3 Department of Dentistry, Tata Central Hospital, Jharia, Jharkhand, India

Address for correspondence Ankit Mahajan, MDS, Department of Periodontology and Implantology, H. P. Government Dental College and Hospital, Shimla, Himachal Pradesh, 171001, India (e-mail: umasharma957@yahoo.com).

Abstract

The first case of pneumonia of unknown origin was identified in Wuhan, the capital city of Hubei Province situated in the Republic of China. The pathogenic organism that has been identified as a causative organism is a novel enveloped RNA betacoronavirus, which has been designated as severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). This virus has been found to have a similar phylogeny to SARS-CoV. The novel coronavirus or COVID-19 can be symptomized through clinical manifestations like pyrexia or fever, cough, dyspnea/difficulty in breathing, myalgia/muscle pain, and constant fatigue. COVID-19 can be transmitted through respiratory tract secretions. It mainly results in respiratory tract infections and the development of severe pneumonia in infected patients. Severe disease may ultimately cause death due to progressive respiratory failure. The coronavirus disease 2019 or COVID-19 has been declared a public health emergency of international proportions by the World Health Organization (WHO). Thus, an analysis of cases might help in the identification of the disease's defining clinical characteristics, association with underlying lifestyle habits, and disease severity. This present article studied the probability of smoking and alcohol consumption in increasing the risk involved in increasing the severity of COVID-19 infection among affected subjects.

Keywords

► COVID-19
► SARS-CoV-2
► smoking
► alcohol
► risk

Introduction

The coronaviruses were first discovered in the 1960s. These viruses were classified under the Coronaviridae family. This family of virus forms the largest among the “Nidovirales” order. This family is composed of two subfamilies: Orthocoronavirinae and Torovirinae. Orthocoronavirinae includes a total of four genera—α-, β-, gamma-, and delta-Coronaviruses. The α- and the β-coronaviruses are found among mammals which include bats. The gamma variety infects all avian and few mammalian species, while the delta variety infects both the avian and mammalian species.1

The designation severe acute respiratory syndrome Coronavirus-2 (SARS-CoV-2) was proposed by the “Coronaviridae Study Group of the International Committee on Taxonomy of Viruses,” according to its taxonomy and phylogeny. Till
April 4, 2020, there were overall 1,117,942 confirmed cases of COVID-19 cases with 59,201 fatalities across the world. The novel coronavirus (also known as, 2019-nCoV and SARS-CoV-2) was first described in December 2019 in pneumonia-affected subjects belonging to Wuhan, which is situated in China. The rapidly rising numbers of cases of this virus around the world have led to a generalized fear, which has been termed as “corophobia.” This virus is transmitted through the respiratory tract route. COVID-19 can manifest clinically as asymptomatic carrier, acute respiratory symptoms, and pneumonia. Neonates, children, and older adults can also get infected alongside. The most prevalent preexisting diseases found in COVID-19 patients under hospitalization include hypertension, cardiovascular disease, habit of smoking, chronic renal diseases, malignancies, and chronic obstructive pulmonary disease (COPD). This is the third most common contagious disease following Middle Eastern respiratory syndrome (MERS) and SARS. The most challenging scenarios which make tackling of this virus include its diagnosis, exact mode of transmission, the longer incubation period (lasting from 3–14 days) and insufficient numbers of available protective resources. The exact rate of SARS-CoV-2 is unknown, although human-to-human transmission is evident due to its wide prevalence among family members and workers related to health care services. Since it is a newly identified pathogenic microorganism, there is no acquired immunity existing among humans. Moreover, curative methods are still unknown. Persons suffering from cardiovascular diseases are at the highest risk of COVID-19. At present, there is no evidence for its transmission route through vaginal delivery or through breastfeeding, although newborn infants should be protected from exposure to infected subjects.

Structure of SARS-CoV-2 or Novel Coronavirus
The SARS-CoV-2 is a spherical-shaped, positive, single-stranded ribonucleic acid (RNA) viral particle. This virus has spiked projections. It has been derived from Latin “corona” which means “crown.” This conforms to their shape resembling a royal crown under an electron microscopic view. These are enveloped viral particles. The envelope is composed of a lipid bilayer, which is derived from the host cell membrane with structural proteins embedded within this envelope. These include spike (S), membrane (M), envelope (E), and nucleocapsid (N) proteins. The “N” protein is bound to the RNA forming nucleoprotein. S protein is a glycosylated protein forming homotrimeric spikes. This protein mediates the entry of viruses within host cells. The M protein is responsible for providing the virus its shape and is found in the largest amount. The E protein causes the release of viral particles from host cells. The CoV genome is the second largest among all RNA viruses. Its weight ranges between 26 to 32 kilobases (kb). Angiotensin-converting enzyme (ACE)-2 associated genes are responsible for various biological processes such as viral metabolism and immunological responses. Elevated ACE-2 levels have been found in oral epithelial cells and intrapulmonary airway but not among nonsmokers or those who had quit the habit of smoking. The binding process has been demonstrated between the SARS-CoV-2 spike (S) glycoprotein receptor-binding domain (RBD) and ACE-2 receptor. The “S” protein of COVID-19 has structural similarity with SARS-CoV; hence, it exploits the ACE-2 receptor for infection among hosts.

Respiratory Diseases in COVID-19
Analysis of viral genome analysis has suggested that SARS-CoV-2 is a recombinant virus between bat and an unidentified origin coronavirus. The human transmission is thought to be possibly taken place from animals. However, there is still an inconclusiveness regarding the exact animal origins of human beings from bats, snakes, or any other animal in a chain of transmission. But there are positive findings about this virus from samples obtained from the seafood industry and markets. Respiratory droplets derived from coughing or sneezing are primary sources for human-to-human transmission. Most frequently reported symptoms of respiratory illnesses due to COVID-19 include higher fever than 38.1°C (98% cases), severe cough (in 76% cases), and severe fatigue or myalgia (observed in 44% patients). Dyspnea appears in 55% infected subjects after 8 days and is the first severe complication of this disease. Also, diarrhea, headache, dyspnea, and hemoptyis have been reported as other clinical symptoms of COVID-19.

The pneumonia caused by SARS-CoV-2 is a highly contagious condition. Zhou et al conducted a study on 62 patients with COVID-19 pneumonia in Wuhan, China. They found that symptoms of fever (87.1% cases), cough with sputum (45.2% cases), pain in muscles (32.3% cases), difficulty in breathing (24.2% cases), fatigue (22.6% cases), and gastrointestinal symptoms such as pain in abdomen and diarrhea (14.5% cases) constitute presenting symptoms of COVID-19 infection. These findings suggested the binding of viral particle to ACE receptor, which is highly expressed in the gastrointestinal tract of humans. These investigators found diverse patterns using computed tomography (CT) among COVID-19 pneumonia-affected patients. These include consolidation, reticular pattern, vacuolar sign, subpleural lines, and bronchial changes such as distortion, pleural retraction sign, and pleural effusion. Mixed patterns are found to affect both lung interstitium and parenchyma. Chest imaging shows bilateral ground-glass opacities.

The Coronavirus pandemic has been preceded by two epidemics caused by betacoronavirus family, namely, MERS-CoV and SARS-CoV-2.

Effects of Smoking and Alcohol in Disease Progression in SARS-CoV-2 Infection
Smoking has been closely linked with adverse prognosis due to its bad effects on pulmonary health and the immune system. Such an association has been earlier seen in the MERS-CoV epidemic. However, there have been contradictory findings reported by few investigations in COVID-19.
Due to these existing variations in published data, we performed a systematic analysis of studies involving the risk factors of smoking and alcohol consumption and their impact on the severity of COVID-19 infection among hospitalized subjects. A literature search was conducted on search engines PubMed and ScienceDirect, using the following terms for conducting a literature search: “AND” and “OR.” Terms searched included “Smoking” AND “COVID-19”; “Smoking” OR “COVID-19”; “Alcohol” AND “COVID-19” and “Alcohol” OR “COVID-19,” etc. All studies published in 2019 and 2020 were included in this systematic review. A total of five studies were included in this review analysis (Table 1).

Zhou et al found no statistically significant difference between the rate of smoking among those who survived and nonsurvivors (p-value = 0.21). Similar findings were published by Zhang et al in 140 COVID-19 patients. They showed that of severe cases, smokers constituted 3.4%, while 6.9% of cases were former smokers. In contrast to nonserious COVID-19 patients, there were no current smokers and 3.7% were ones who quit smoking.

Lippi and Henry in their meta-analysis observed that there is a contradiction regarding whether the smoking habit was associated with COVID-19 severity. Many investigators reported with no statistical association between active smoking and disease severity. A total of five studies were included in this meta-analysis, comprising 1,399 COVID-19 patients 288 (20.6% cases) who suffered from severe disease. Only one study demonstrated that active smoking was a significant predictor of COVID-19 disease severity. However, the rest of the studies (total four in number) showed no statistically significant association. Although smokers are at higher risk, no significant association could be found between active smoking habit and SARS-CoV-2 severity on pooling data of studies included (odds ratio [OR] = 1.69; 95% confidence interval [CI] = 0.41–6.92 and p = 0.254). However, decreased ACE-2 levels have been reported in COVID-19 patients.

One of the common measures employed by governments all over the world is isolation and distancing. However, long-term isolation can result in stress due to which chronic alcohol use increases. There is also a risk of relapse among past alcohol users and an increase in alcohol abuse.

Consumption of alcohol concomitantly with smoking suppresses host immunity. This increases the risk of infections of the upper respiratory tract. There is evidence that the consumption of alcohol raises the chances of infection. Various complications associated with alcoholism include hepatic cirrhosis, deficiency of nutrients, and personal hygiene along with lifestyle changes. In a study conducted by Cohen, 36% nonsmokers, 40% light smokers (consuming one to fifteen cigarettes per day), and 48% heavy smokers (consuming more than fifteen cigarettes per day) developed cold symptoms. This study concluded that smoking habit was associated with an increased risk of infection of the upper respiratory tract, while consumption of alcohol is associated with a reduced risk of respiratory illness. However, this correlation is completely dependent on dosages and their response.

Patwardhan et al in their study concluded that the long isolation period, which is observed in COVID-19 spread, can create mental stress, leading to an increase in frequency of smoking and even relapse in individuals who had quit the habit. Lai et al in their study observed that pneumonia was diagnosed in older patients who had higher smoking prevalence. They reported systemic conditions of hypertension (in 20.5% cases) and diabetes (in 14.4% cases). It was also observed that similar viral loads were found in both symptomatic and asymptomatic carriers. However, high-viral load was detected from patients with severe manifestations.

Literature has proven studies to show that carcinogenesis is often associated with low-immunological status, which is characterized by overexpression of immunosuppressive cytokines along with suppressed induction of proinflammatory signals associated with apoptosis, impairment of maturation of dendritic cell, and increased immunosuppressive leukocytic populations. These chains of events are contradictory to presentations in severe COVID-19 patients. This difference in susceptibility along with prognosis is due to higher rates of smoking habits in patients with cancer. Also, tobacco usage has been found to significantly increase gene expression of ACE-2, which acts as a binding receptor for SARS-CoV-2. This can very well explain elevated susceptibility toward COVID-19 infection among smokers. Besides, smoking cigarettes is one of the leading reasons for COPD. COPD has been labeled as an independent risk factor in severe COVID-19 infections.

Transmission of SARS-CoV-2 has been attributed via respiratory droplets and contamination of surfaces and hands. This microbial strain can persist upon inanimate surfaces for up to 9 days period at room temperature. As per recommendations by WHO, environmental cleaning and disinfection procedures should be followed constantly and correctly. Thorough cleaning of surrounding surfaces using
water and detergents along with the application of common hospital-level disinfectants are both effective and sufficient for eradicating the virus. However, effective inactivation can be achieved by the use of surface disinfectants like 0.5% hydrogen peroxides, 0.1% sodium hypochlorite, or 62% to 71% ethanol. Carrier tests have shown that the use of 62% to 71% ethanol reduces viral load by 2.0 to 4.0 log₁₀. As much as 0.1% to 0.5% sodium hypochlorite and 2% glutaraldehyde also demonstrates a decrease up to more than 3 log₁₀. All of the above-mentioned surface disinfectants show their effectiveness within 1-minute duration of application. Current testing methods employed for SARS-CoV-2 include polymerase chain reaction (PCR)-based assay.¹⁰

### Conclusion

The SARS-CoV-2 or COVID-19 infection is an ongoing pandemic that is infecting increasingly numbers of people across the world. At present, there is no specific treatment, and it is expected to come after some time. Preventive and protective measures observed at individual levels lessen the risk of acquiring this disease. The virus-infected hosts have widely different metabolic systems, which are largely determined by dietary factors, nutritional level, age at time of infection, gender, underlying medical or systemic conditions, lifestyles and, most importantly, environmental factors. These variations govern the body’s response to the clinical severity of COVID-19 disease infection. Thus, an individual’s assessment of these factors is right now the best way in dealing with the SARS-CoV-2 pandemic. Studies utilizing data obtained from positive disease shows that active smoking can lead to adverse outcomes but was not significantly associated with a greater risk of progression toward COVID-19 severe disease. Further, due to the limited availability of data research should be performed to assess the outcome of smoking and consumption of alcohol on the outcome of COVID-19 severity.

### Conflict of Interest

None declared.

### References