COVID 19 - a new threat for smokers and vapers?

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Abstract

The new pandemic disease Covid-19 compelled all the researchers to investigate for early identification of the potential risk factors. Further, the relation between smoking and infections are well known. The authors are trying to find the epidemiological links, the pathogenic mechanisms and also the impact of this coronavirus on different respiratory chronic diseases, based on the last published data about the consequences of smoking and vaping on consumers.

Keywords

vaping • e cigarette • smoking • COVID-19

Introduction

The year 2020 started with a new pandemic disease COVID-19, spreading rapidly everywhere. More and more alarming signals and information reached the world from Wuhan, Hubei Province (China) in the last few months. After extensive research made by my Chinese colleagues, the first case (case zero) was reported in November 2019 and officially recognized in December (1). A wet market in this town is found to be and considered as the possible pandemic origin. The link between humans and the intermediate host is still unclear even though the researchers found a 96% DNA match between bat and human coronavirus or they suspected pangolins earlier for the disease (1). It is one of the important epidemic diseases of the last century. Other pandemic diseases in the last 40 years prior to this challenging disease were: Ebola virus disease in 1976 in the Democratic Republic of Congo and Sudan (fatality rate around 50%), Severe Acute Respiratory Syndrome (SARS) (caused by SARS coronavirus) in 2002–2003 (first case in
China a mortality rate of 9.6%), Asian Flu (N1H1) in 2009 (first case in California, with 0.001–0.007% deaths of the world population) and Middle East Respiratory Syndrome (MERS) (caused by a coronavirus (MERS-CoV) in 2012 (first case in South Arabia, 35% of reported patients died) (2). Now, we have the initial statistics on COVID 19 infection and the researchers are working to evaluate the social, health and economic impact on every nation. On March 13, WHO said that Europe was the centre of the pandemic (3) but every European nation approached and tackled this contagious disease and its consequences with different strategies. On 29 March 2020, the pandemic statistics were: more than 6,84,000 are confirmed with infection, over 32,000 are dead and more than 1,45,000 are recovered (4). We have now started to learn many lessons from the obtained data. When risk factors are considered even with the available limited data taking into account risk and prognostic factors, smoking is found to be a recognized detrimental factor to the immune system as well as the responsiveness to a variety of infections. The outbreaks of influenza, MERS, and SARS brought more severe cases and increased mortality rates of smokers or the risk of infections (5,6). But the following question ‘what are the links between smoking, vaping, and COVID 19’ is still remaining unanswered. We are trying to find the answers starting with the epidemiology of the disease, possible pathogenic interactions, or therapeutically aspects.

Epidemiology connections

Vardavas and Nikitara (7) conducted a research about the possibility of the association between smoking-severity of COVID 19 infections, the need for ventilation, need of ICU, and rate of hospitalisation or death. They screened 71 studies and finally, only 5 from China were selected by the authors. Their idea was following the first 2 months of the COVID 19 pandemic (December 2019 and January 2020) and registering the data, and found that the mortality rate of smokers is 9% (8). General mortality in China due to COVID 19, was 3.6% (9). Consistent with these 5 studies, analysis of deaths due to coronavirus in China shows that men are more likely to die than women, which may be probably related to the fact that many Chinese men smoke more than women (10). WHO report on the global tobacco epidemic, China Country Profile 2019, demonstrated that 52.1% of Chinese men smoke compared to just 2.7% of women. Another strong argument coming from these studies is about the factors and chances against and of disease progression (including death). Moreover, Chinese authors reported that there is a 14 times higher risk of having pneumonia among people with a history of smoking compared to those who did not smoke (11). Zhang and et al. (12), after analysing the mortality and the severity of diseases, found that among the severe vs. nonsevere patients, 3.4% were current smokers and 6.9% were former smokers, and on the other sample 0% and 3.7% (QR-2.3). In another study (13) the number of smokers with severe disease was much higher (12.6%). Further, it is mentioned by Liu et al. in his paper that smoking, as a risk factor, is responsible for adverse outcome in patients with a history of smoking (27.3%) than the group that showed improvement or stabilization (3.0%) \( p=0.018 \) level (11). Most of these studies were on small samples. The largest study was conducted by Guan et al. (13). They identified that higher percentages of current and former smokers among patients needed ICU support, mechanical ventilation or who had died, and a higher percentage of smokers among the severe cases. Further, they quantified the risk for smokers vs. non-smokers; for the first group, it is 1.4 times more likely to have severe symptoms of COVID-19 and 2.4 times more likely to be admitted to an ICU (on ventilators or to die).

Hypothesis of susceptibility to COVID 19 of smokers

It is well known that smokers are vulnerable to virus and countless studies confirming the same. Angiotensin converting enzyme 2 (ACE2) has been identified as a receptor for HCoV-19. This generates the hypothesis that smoking is responsible for the differences in the expression pattern of ACE2 in the respiratory tract, with consequences related to the susceptibility to the virus. Three datasets (GSE994, GSE17913, and GSE18344) have been evaluated. Elevated ACE2 were found in intrapulmonary airways (GSE994) and oral epithelial cells (GSE17913) of smokers; it was not the same for non-smokers or former smokers (14). Data on human as well as experimental research on rats confirmed that smoking is increasing ACE2 in the respiratory tract and also the susceptibility to HCoV-19. Smoking can upregulate this angiotensin-converting enzyme-2 (ACE2) receptor which is recognized as a receptor for other coronavirus infections such as SARS, SARS-CoV, and the human respiratory coronavirus NL638 (15). ACE2 could be a novel adhesion molecule for SARS-CoV-2 causing COVID-19. So, it can be a potential therapeutic target for the prevention of fatal microbial infections and the same type of upregulation is possible for e-cigarette and heat-not-bum IQOS devices (15). Using RNAseq, Leung et al. (16) determined gene expression levels in bronchial epithelia obtained from cytological brushings of 6th to 8th generation airways in individuals with and without COPD. They found an increasing ACE2 expression in lower airways in patients with COPD and current smokers. Moreover, they suggest that these patients are at increased risk of serious COVID19 infection and highlight the importance of stopping smoking in reducing the risk. The other well-known effect of smoking is vasospasm and transient hypoxia of organs induced by a high level of blood nicotine. The depletion of oxygen in the viscera and the respiratory tract will reduce immunity significantly (17).
Dissemination of COVID 19 infection depends also on the one’s smoking habits; that’s why in between the personal precarious recommendations coming from the specialist it’s also included quitting smoking

E cigarette and COVID-19
Just like the discussion on vaping divided the medical schools between the defenders and opponents for 2 years, the discussion on the consequences of COVID 19 on the dangers of vaping and e-cigarette have split the community into two. For some authors (18) there is no evidence that vaping (intrinsically) increases the risk of infection or progression to severe condition of COVID-19; they consider it as an effect of COVID19 linked to previous conventional smoking. They sustained also that even propylene glycol is working as a disinfectant. That’s why these authors have only some plain recommendations like (18,19):

• If you vape, do not revert to smoking (if you are a dual user try to become an exclusive vaper).
• If you enjoy vaping and do not smoke, quitting vaping must be a personal choice and not an obligation.
• Be discreet and do not call unwanted attention (bear in mind that these are difficult times and that a lot of non-vapers have been exposed to a lot of misinformation).
• Avoid big clouds in public no matter what (even outdoors).
• Use low powered devices whenever possible and when others are around. The risk of spreading the virus with discrete vaping in low powered devices is roughly equivalent to the risk of spreading it through normal sedentary breathing.
• Avoid vaping in enclosed public spaces and try to keep at least 2 m distance from others when vaping outdoor.

Contrarily Prof Glantz (20), recognized as an objective researcher (without any sponsoring activities from the tobacco industry), stated that vaping like smoking may also harm lung health; his arguments are based on the well-known demonstrated effects of e-cigarette on the lung (21,22) (impaired mucociliary clearance and neutrophil phagocytosis, damage of the lung by the effect of neutrophils lyse DNA and release it into the extracellular environment to help to immobilize bacteria and enhance of upper airway colonization and tissue damage by aerosol exposure). Because of these, including stopping smoking, vaping, and avoiding second-hand exposure are the lists of important preventive measures according to his opinion. Even the FDA stated in an email that e-cigarette use can damage lung cells (23).

Smoking and different diseases. What we already know
In lung cells including bronchial epithelial cells, alveolar macrophages, pulmonary endothelial cells, and interstitial fibroblasts, nicotinic receptors are co-expressed with most components of the RAS (renin–angiotensin system) (24). Smoking is already known for increasing the risk for the development of acute respiratory distress syndrome (ARDS) on people associated with different risk factors like: severe infection, non-pulmonary sepsis (blood infection), or blunt trauma (25, 20). Regarding the cancer patients with smoking, the higher rate of smoking history can be one of the potential factors of higher risk of COVID-19, and with a poorer prognosis than those without cancer (26). The same authors describe these cancer patients in the sample are prone to severe conditions and events (admission to the intensive care unit requiring invasive ventilation, or death) due to COVID-19. For COPD patients after adjusting for age and smoking status, COPD hazards ratio (HR) was 2.681(95% confidence interval 1.424–5.048) (27). The number of comorbidities is also an important factor. Smoking is a trigger for asthma and at the same time smoking can weaken lung function. When you have asthma, if you smoke, your lung capacity will deteriorate very rapidly. Smoking when children with asthma are near to you will affect their lung function too (28).

Conclusions
Many relations are established at this moment by demonstrating the smoker’s vulnerability to COVID-19. If more cases are being examined in the coming months (from different ethnic and genetic backgrounds worldwide), probably this ACE2 expression variation can be better analysed and compared to establish whether and when it contributes to susceptibility to COVID-19 and it will be more simple to find some subgroups or phenotypes.(29) But, it is certain for smokers with COVID 19 infections, there will be a greater impact on disease progression; association with smoking is most likely with the negative progression and adverse outcomes of COVID-19. Further, WHO and all countries should ensure for the next pandemic waves that the smoking status of patients identified with Covid-19, including deaths, is recorded and incorporated in data sets for determining this important relationship of the virus with an external risk factor. Data on vaping and COVID-19 are now collected and we are confident that the next articles will be able to analyse on deleterious effects of e-cigarette effectively and more convincingly.

References


