Editorial

Smoking amid Covid-19; a jeopardy calling attention

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In December 2019, pneumonia of unknown etiology were reported in Wuhan, Hubei Province, China, which was linked to Huanan Seafood Wholesale Market.[1] The virus was recognized as a novel coronavirus and officially named by the WHO as 2019-nCoV. As the cases increased and many patients died,[2] the disease was called corona virus disease 2019 (COVID-19) specified as SARS-CoV-2, which was discovered through whole-genome sequencing, polymerase chain reaction (PCR) as well as culture of bronchoalveolar lavage fluid obtained from affected patients.[1] WHO on March 11, 2020 declared COVID-19 as a global pandemic.[3] As of May 10th, there are more than four million confirmed cases of COVID-19 and almost 280,000 deaths globally.[4] Immunocompromised individuals are more susceptible to severe COVID-19 complications.

Tobacco smoking is the cause of annually 6 million deaths worldwide and is estimated to surpass 8 million by 2030, according to statistics from the World Health Organization (WHO).[5] Around 5.7 million die as a result of direct tobacco use and 1.2 million are consequent to non-smokers being presented to tobacco smoke.[6] Smokeless tobacco use is a significant health risk and cause of disease.[7] Along with conventional cigarette smoking, e-cigarettes including Electronic Nicotine Delivery Systems and JUice USB Lighting (ENDS and JUUL), waterpipe as well as other tobacco smoking devices have nicotine products. Smoking leads to diseases such as chronic obstructive pulmonary disease (COPD) as well as lung cancer.

Tobacco smokers have an increased risk of severe COVID-19 infection due to compromised lung function.[6] Eroding of the protective lining of lungs, damage of blood vessels as well as irregular endothelial surface leading to blood clotting and lethal embolism is attributed to nicotine chemical derivatives. Hence, in the presence of existing chronic lung disease, the bronchial status of patients can rapidly aggravate with COVID-19 infection[6].

Tobacco smoking incorporating use of cigarettes as well as other devices including electronic cigarettes, Vape, JUUL as well as water-pipes, endangers the spread of salivary droplets as aerosols peculiarly in cases of SARS-CoV-2 infected asymptomatic smokers. There is also a possibility of onset of passive infection by means of smoke as well as aerosol from asymptomatic infected smokers.[6] With Middle East respiratory syndrome coronavirus (MERS-CoV) outbreak, smoking was noted to be a risk factor and linked with high mortality.[8]

The mechanisms by which smoking elevates the risk of worsening pneumonia include transformed airway architecture, suppression of airway ciliary clearance as well as diminished immune function.[8] Smoking unfavorably affects immune system. In the first place, it lowers CD4+ T-cells, helper T-cells, that patronize antibody production in B-cells as well as activate killer T-cells to attack pathogens.[8] Nicotine stimulates the secretion of catecholamines as well as corticosteroids, can debilitate immune function and represses the ability to combat infections. Also nicotine curbs the formation of interleukin-22, which has role in lowering lung inflammation and rebuilding damaged cells.[8] Tobacco use may predispose to enhanced risk of suffering from serious symptoms due to COVID-19 illness.[9] Smoking may strongly elevate the likelihood of adverse health outcomes for COVID-19 patients related to intensive care and mechanical ventilation.[9]

Liu et al found that in patients with COVID-19 the adverse outcome group had an undoubtedly higher proportion of patients with a history of smoking (27.3%) compared to the group that exhibited improvement or stabilization (3.0%).[10] MERS-CoV infection is associated with dipeptidyl peptidase IV (DPP4) receptor while SARS-CoV-2 is associated with ACE2 receptor (angiotensin II conversion enzyme-2 receptor). For infecting the host, each virus attaches to its receptor, MERS-CoV to DPP4

and SARS-CoV-2 to ACE2, leading step for coronavirus infections.[11] ACE2 gene expression is more advanced in current as well as former smokers in contrast to those who never smoked.[11]

The ACE2 receptor yields a human cell-binding site for the S protein of the SARS-coronavirus , SARS-CoV, a virus which was initially recognized in 2003 in a southern province of China, the coronavirus NL63 and now SARS-CoV-2.[12] Modified S protein of SARS-CoV-2 has greater affinity for ACE2 and is 10- to 20-fold highly likely to bind to ACE2 in human cells as opposed to the S protein of the previous SARS-CoV. This is indicative of smokers being more prone to infection by SARS-CoV-2 and likely to have Covid-19 (smoke COV 3).[12] It is therefore not unanticipated to find a worse prognosis of COVID-19 in patients with COPD.[13]

Smoking is likely to be a potential factor correlated to COVID-19 severity. Irrespective of the association between cigarette smoking and COVID-19 severity, research in this area is scarce to date and hence it would be helpful to have more accumulation of evidence in this area. Furthermore evidence is also required regarding effect of second-hand smoke on the transmission of SARS-CoV-2. It is therefore recommended that all cigarette stub and devices and their attachments after single use should be disposed off. Smoking devices should be single use and no re-use or sharing of devices be practiced.[6] In summary, smoking has deleterious effect in COVID-19 and quitting smoking can help the body fight the virus better.

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