PERSPECTIVE

COVID-19's putative function in DNA damage induction

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ABSTRACT

The Coronavirus Disease-2019 (COVID-19) produced by the Severe Acute Respiratory Syndrome coronavirus-2 (SARS-CoV-2) is putting the world's health and economic systems in jeopardy. COVID-19 can produce a wide range of symptoms in certain people, affecting several organs including the lungs, heart, intestines, kidneys, and brain, resulting in multi organ failure, sepsis, and death. These consequences are linked to direct viral infection of these organs, immunological dysregulation, hyper coagulation, and the possibility of developing cytokine storm syndrome. Because COVID-19 is a new virus, the long-term repercussions on the health of recovered individuals are unclear. We focused on current evidence of coronavirus-mediated DNA damage pathways in this study. The evidence suggests that these viruses can cause DNA damage, genomic instability, and cell cycle disruption during replication in mammalian cells. Because the generation of DNA damage and abnormal DNA repair pathways is linked to the development of chronic illnesses such as cancer, diabetes, neurological disorders, and atherosclerosis, it will be critical to investigate comparable effects and outcomes in recovered COVID-19 patients.

Key Words: *COVID-19; SARS-CoV-2; DNA damage*

INTRODUCTION

The disease caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) was identified for the first time in December 2019 in Wuhan, Hubei Province, China. The World Health Organization termed this illness COVID-19, and it has already spread to more than 200 nations, with over 260 million confirmed cases and more than 5 million fatalities globally. The primary modes of SARS-CoV-2 transmission have been found to be from person to person via respiratory droplets, airborne transmission via aerosols, and contact with infected surfaces. People over the age of 60, as well as those with comorbidities such as cardiovascular disease, Type 2 Diabetes (T2D), obesity, or cancer, have been found to be at a higher risk of developing more severe symptoms. COVID-19. Surprisingly, it has been discovered that 20%–60% of SARS-CoV-2 infected individuals are asymptomatic or exhibit relatively moderate signs of the illness. Despite this, these individuals can develop lymphopenia, increased alanine aminotransferase and C-reactive protein levels, and lung abnormalities include opacities, shadows, and widespread consolidation. Furthermore, the number of patients reporting symptoms for more than 10 months after the first infection has increased, which is now recognized by the WHO as long COVID or post COVID syndrome. Although the incidence of this persistent condition is unknown, reports indicate that it might occur in people who were not hospitalised or who had very minor symptoms during the acute viral infection. As a result, it is important to investigate the long-term implications of the original viral infection. In this paper, we examine the current data on SARS-probable CoV-2's methods for inducing DNA damage, which may lead to long-term repercussions.

The most severe types of COVID-19 have been linked to cytokine storm syndrome, a dysregulated pro-inflammatory response. Although interleukin-6 (IL-6) is the most often overexpressed cytokine in COVID-19 patients, its levels are many orders of magnitude lower than in other inflammatory syndromes such as Acute Respiratory Distress Syndrome (ARDS), Cytokine Release Syndrome (CRS), and overt sepsis. Other usual cytokines implicated in the cytokine storm syndrome generated by ARDS, CRS, or sepsis (such as IL-8, TNF, IFN, and sIL-2R) are less prevalent in COVID19 patients, raising issues regarding their importance in the fate of COVID-19 patients. C-reactive protein levels, on the other hand, are much greater in COVID-19 individuals than in other cytokine storm syndromes. The liver produces C-reactive protein in response to tissue injury, infections, and inflammatory signals. This protein binds to phosphatidyl choline in dying cells' and bacteria's membranes, causing phagocytosis by circulating macrophages and increasing systemic inflammation. High amounts of C-reactive protein have been linked to high levels of oxidative damage in the DNA of people suffering from psoriasis, obesity, pancreatic cancer, and cardiovascular disease. Furthermore, chronic inflammation is linked to the generation of reactive oxygen species, which can increase the development of some cancers, insulin resistance, and vascular lesions. Serum glutathione and total thiol levels are lower in COVID-19 patients, whereas superoxide dismutase, catalase, malondialdehyde (a marker of oxidative stress and lipoperoxidation), and total oxidant status are higher. Oxidative stress levels were shown to be connected to blood oxygen saturation levels, illness severity, prognosis, and the viral variety causing the infection. An examination of transcriptome alterations seen in diverse data sets from blood cells, lung biopsies, or leukocytes from healthy participants and COVID-19 patients revealed a considerable elevation of pro-oxidant genes, particularly myeloperoxidase and calprotectin genes. Another study discovered that peptides derived from the S protein of SARS-CoV-2 increased the levels of nitrites, hydrogen peroxide, and Reactive Oxygen Species (ROS), as well as upregulated the activities of superoxide dismutase and catalase in a tadpole model, indicating that the S protein can cause oxidative stress in animals. Overall, these findings show that oxidative stress plays a crucial role in COVID-19 aetiology, and all of these clinical findings warrant further investigation.

The imbalance between ROS generation and antioxidant molecule levels in cells causes oxidative stress. This pro-oxidant condition damages macromolecules including lipids, proteins, and nucleic acids. Single- and double-strand breaks, DNA-protein crosslinks, and base and sugar oxidation products, such as Guanine Oxidized Species (GOS), can all be caused by oxidative stress in the DNA. GOS was found in the serum of critical COVID-19 patients in intensive care units, according to one research. The scientists discovered that GOS levels in the blood of non-surviving COVID-19 patients were greater than in those who survived, indicating that oxidative DNA damage might be a role in COVID-19 mortality. The study, however, lacked groups of asymptomatic COVID-19 patients and control groups of non-infected people, which are required to establish that oxidative DNA-damage is prevalent in COVID-19 patients regardless of illness severity. Another research evaluated blood malondialdehyde and GOS levels in COVID-19 patients who were hospitalized with those who were not. The authors discovered that malondialdehyde rises at the time of hospitalization and rapidly drops during the time-course studied, whereas GOS peaked 7 days after admission. These data showed that oxidative stress comes before DNA damage during the aetiology of COVID-19. Surprisingly, malondialdehyde levels were greater in non-hospitalized patients, whereas serum GOS levels

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were higher in hospitalized patients; however, the investigators could not link this finding with oxygen treatment or subsequent disease severity.

This latest research implies that there is a larger chance of acquiring DNA oxidative damage in severe COVID-19, possibly because to persistent oxygen supplementation during in-hospital patient care. Similarly, a study aimed at determining DNA fragmentation in sperm cells and sperm quality in patients with mild cases of COVID-19 discovered that DNA fragmentation in sperm cells and oxidative stress markers were higher 14 days after diagnosis than levels in the same patients 120 days after diagnosis. However, like with earlier investigations, this one lacked a non-infected control group. To determine the extent of oxidative DNA damage caused by COVID-19 infection and/or treatment, future studies comparing markers of oxidative DNA damage between non-infected subjects and patients from the entire spectrum of COVID-19 patients undergoing both hospitalization and home recovery are required. Oxidative DNA damage has long been linked to an increased risk of developing neurological disorders and cancer. This raises the notion that severe COVID-19 individuals with pro-inflammatory and pro-oxidative states may have a higher chance of acquiring other chronic illnesses in the long run, and that SARS-CoV-2 virus infection may increase these comorbidities in patients with comorbidities. Future research is required to address the risks of acquiring such diseases in both sick and well COVID-19 individuals.

CONCLUSION

The worldwide SARS-CoV-2 pandemic is posing challenges to many aspects of human civilization, and the immediate and long-term effects on human health are yet unknown. The risk of chronic illness development in recovered COVID-19 patients is unknown. Coronaviruses from the same family as SARS-CoV-2 can cause DNA damage and impede DNA repair pathways, leading to genomic instability. DNA damage and abnormal repair processes are recognized to have a role in the aetiology of many chronic illnesses, including cancer, obesity, diabetes, atherosclerosis, and metabolic syndrome. These possible consequences underline the need of assessing the long-term implications of this unique viral illness in both recovered COVID-19 patients and asymptomatic but infected persons.