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Nutritional Impact of COVID-19 and Its Implications on Atherosclerosis

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Abstract The coronavirus disease 2019 (COVID-19) pandemic has affected people from every part of the world, with the elderly and minority groups being at a higher risk owing to the high prevalence of underlying health conditions such as atherosclerosis among these groups. This study discusses numerous implications of the COVID-19 pandemic on people consuming more unhealthy foods due to restricted access to stores and stores closing early. A lot of people currently live a sedentary lifestyle because of the strenuous quarantine measures. COVID-19 impacts the nutritional status of people leaving them at risk of atherosclerosis and worsens the health condition of individuals suffering from it. Also, the use of COVID-19 medications has been observed to have side effects in patients with atherosclerosis. Thus, we review the nutritional impact of COVID-19 and its implications on atherosclerosis at length and make recommendations for reducing future implications. This study would serve as a foundation for further research on COVID-19 and its implications on atherosclerosis as only a limited number of studies have addressed it.

Keywords: *atherosclerosis, COVID-19, Sars-CoV-2, cardiovascular disease, heart failure*

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1. COVID-19 and Atherosclerosis

Cardiovascular disease (CVD), mainly triggered by atherosclerosis, is a prominent cause of deaths globally [1]. Atherosclerosis is an inflammatory disease of the arterial wall [2,3], characterized by the gradual buildup of lipids within the intima of both medium and large sized arteries [4,5]. There are several risk factors for atherosclerosis, and their impact on health have been identified [6]. Some of these risk factors include hypertension, increased level of low-density lipoprotein cholesterol (LDL-C), high total body cholesterol, reduced level of high-density lipoprotein (HDL) and a sedentary lifestyle [5,7]. Diet and physical activity play a major role in the prevention, development, and treatment of atherosclerosis [5]. There is an increased risk of COVID-19 related mortality in patients with atherosclerosis due to their fragility and increased vulnerability to myocardial incidents [8]. The severe acute respiratory syndrome coronavirus 2 (Sars-CoV-2) have been found to affect coronary microvascular function in patients with heart failure [8]. Sars-CoV-2, the causal agent of COVID-19 is a single-stranded ribonucleic acid **(**RNA) virus primarily transmitted through respiratory droplets from one individual to another [9,10,59]. In some patients, Sars-CoV-2 has been observed to cause respiratory infection leading to pneumonia and acute respiratory distress syndrome [9,11]. Multiorgan damage and thromboembolic events have also been reported in patients with uncontrolled COVID-19 infection, due to the immune system oversecreting pro-inflammatory cytokines and chemokines, and presence of coagulation abnormalities [12,13,14]. Several studies have been able to show a link between pro-inflammatory cytokines and the development of atherosclerosis. COVID-19 causes hyperactivation of the immune system resulting in increased number of cytokines, and the occurrence of vascular inflammation contributing to the progression of atherosclerosis [15,16,17,18,19]. This review focuses on the nutritional impact of COVID-19 and its implication on atherosclerosis.

2. Background of COVID-19

In the past, countries have witnessed several epidemics that caused enormous destruction. Some of these

deadly outbreaks include the Spanish Flu of 1918, HIV/AIDS (Human Immunodeficiency Virus/Acquired Immunodeficiency Deficiency Syndrome), SARS (Severe Acute Respiratory Syndrome), MERS (Middle East Respiratory Syndrome) and Ebola viruses. Prior to the identification of SARS-CoV and middle east respiratory syndrome coronavirus (MERS-CoV), coronaviruses were only thought to cause mild respiratory infections in humans. The exact pathophysiological mechanisms underlying the emergence of SARS-CoV-2 are still unknown and are still being studied in laboratories and until these studies yield any results, the precise mechanism of SARS-CoV-2 remains hypothetical [20].

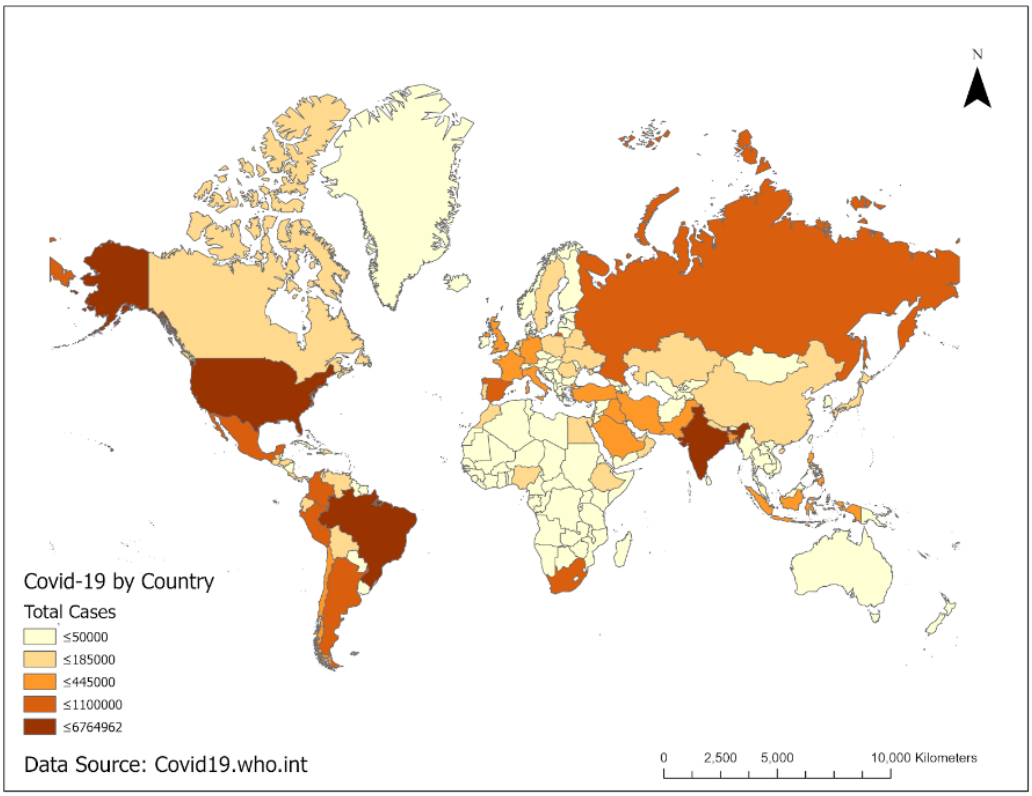

Figure 1. Total COVID-19 Cases across Different Countries as of September 20, 2020

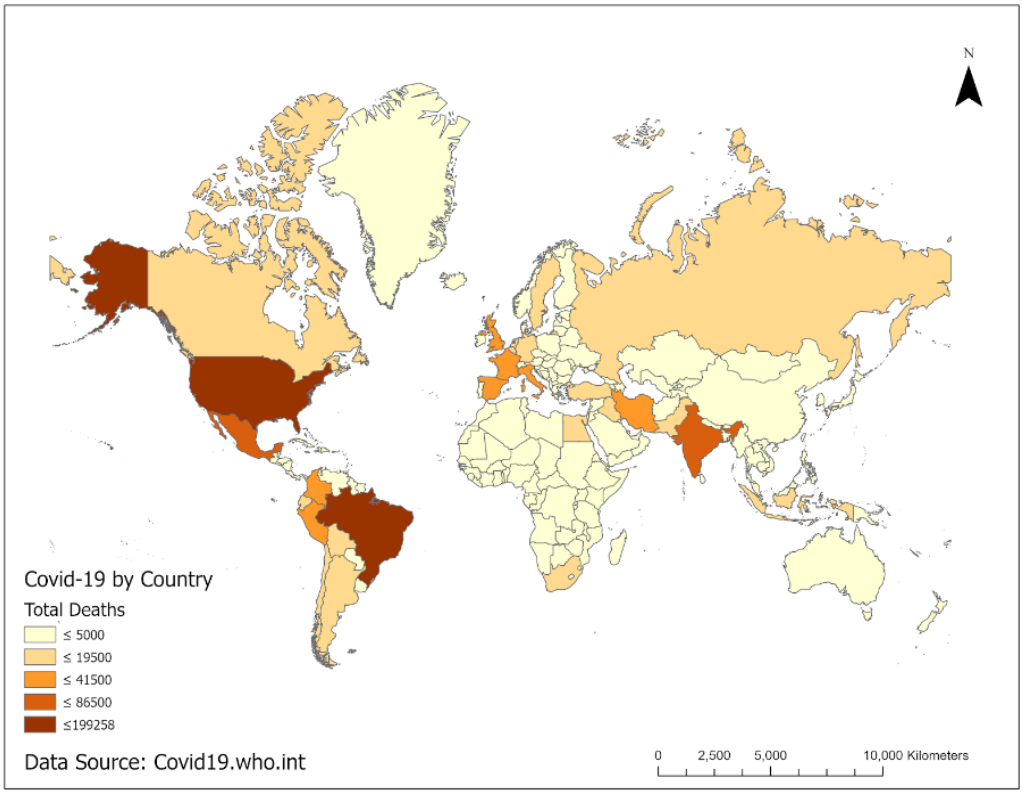

Figure 2. Total COVID-19 Deaths across Different Countries as of September 20, 2020

COVID-19, which had its origins in China reportedly around December 2019 rapidly and violently spread to over 200 countries. The first presumptive case which started with symptoms of pneumonia with an unknown cause was found close to a seafood market in Wuhan, the capital city of Hubei province, China, on December 8, 2019. Within a few days, many clusters emerged with patients having similar pneumonia-like symptoms around the region. With its continued spread, the pneumonia symptom was identified to be caused by a new coronavirus called SARS-CoV-2 [21]. Originating from China, coupled with inadequate containment and preventive measures taken, there was continued influx and outflux of people into and out of China. Perhaps, the most contributing effect to the rapid spread of the virus came as a result of the massive outflow of travelers during the Chinese Spring Festival travel rush in mid-January. This caused the spread of COVID-19 throughout China and to other countries [21].

3. Nutritional Impact of COVID-19

One of the major effects of COVID-19 on individuals is a change in lifestyle habit such as the consumption of unhealthy diets [22]. The restriction on the number of people in stores at a given time as well as restricted store hours resulted in reduced access to food and lots of people switching to convenient foods which were mostly unhealthy [22]. The demand for packaged foods, frozen foods and foods with a long shelf life was high resulting in a low demand of fresh foods, fruits, and vegetables. A major consequence of these changes is an increase in weight gain [22]. Oxidative stress occurring as a result of psychological stress due to individuals having inadequate essential necessities such as food, and vascular inflammation are one of causes of atherosclerosis [22,23,24].

Sars-CoV-2 have been observed to affect people of all ages, but the age group most vulnerable to this virus are the elderly population [25]. Children have been observed to be asymptomatic or develop mild symptoms when affected with COVID-19 and are therefore less vulnerable to COVID-19 compared to adults [26]. Minority groups comprising Blacks/African Americans and Latinos have been observed to be heavily impacted by the novel coronavirus especially those with underlying disease conditions such as atherosclerosis [25]. In developed countries such as in the U.S, consumption of diets high in saturated fat and low in fiber have been attributed to the development of atherosclerosis [27]. The consumption of unhealthy foods and the presence of underlying disease conditions increases COVID-19 risk in the elderly and minority groups which are the vulnerable groups due to their enhanced sensitivity to pro-inflammatory modulators and health discrepancies [25]. A change in food intake to healthy foods among these groups have been found to have an anti-inflammatory effect on them [25]. Increasing access to healthy foods, fruits and vegetables would help alleviate atherosclerosis among the vulnerable groups by boosting their immune function giving them a better chance at fighting off COVID-19 infection [28].

Due to lockdown measures imposed in most countries around the world, most individuals had little to no access to gym facilities, the typical day to day exercise most people undergo by leaving their homes to work was reduced as most people worked from home leading to a reduction in physical activity and thus a sedentary lifestyle. One of the benefits of adequate physical activity is that it reduces atherosclerotic risk [29,30]. Therefore, inadequate physical activity during COVID-19 lockdown increased the risk of atherosclerosis. There is also an increase in oxidative stress due to a decrease in physical activity resulting in increased vascular inflammation, reduced nitric oxide levels, increased oxidized LDL and foam cell accumulation [30]. Oxidative stress also increases the risk of endothelial cell dysfunction due to apoptotic cell death of endothelial cells resulting in inflammation [31]. The presence of pro-inflammatory properties in the body contributes to the development of atherosclerosis and a reduction in physical activity exacerbates atherosclerotic risk [22]. A long-term effect of COVID-19 implications is the difficulty in restoring previous healthy eating habits after a long period of consuming unhealthy foods. Lifestyle habits are difficult to change, and the presence of vascular inflammation due to an unhealthy food intake can activate pre-existing plaques in the body resulting in atherosclerosis [22].

4. Impact of COVID-19 on Atherosclerosis

Atherosclerosis is one the underlying diseases commonly found in patients with the coronavirus [32,33,34]. COVID-19 increases the rate of mortality in patients with atherosclerosis [10,35], with COVID-19 medications such as ribavirin and chloroquine causing cardiovascular atherosclerotic side effects such as hypotension and cardiomyopathy [8]. Other atherosclerotic side effects caused by COVID-19 medications include myocardial infarction (MI), hypertension, vasculitis, arrhythmia etc. [8]. COVID-19 was also found to cause heart failure in patients with atherosclerosis [36]. A recent study in Wuhan, China showed that heart failure was among the most recognized complications of patients with COVID-19 observed in up to 4% of patients [36]. Acute myocardial injury was also detected in about 7-20% of patients with COVID-19 [9,37,58]. Patients with myocardial injury are at risk of developing atherosclerosis [38]. Hypertension was the most reported complication of COVID-19 observed in about 40% of patients [39,40,41,42]. An observational study evaluating long-term effects of the corona virus infection established that lipid metabolism and the development of atherosclerosis is impacted by Sars-CoV-2 [10,43]. Increased number of free fatty acids and phosphatidylglycerol owing to an altered lipid metabolism was discovered in 25 patients evaluated about 10 years after they recuperated from SARS-CoV in 2003. Greater than 40% of these patients were affected by CVD [10,19].

Common atherosclerotic manifestations reported by patients with COVID-19 infection include a minor respiratory syndrome, hypoxemia, ischemic stroke, and inflammatory thrombophilia which may include

thromboembolism [37,44,45,46]. Other cases reported include an increase in serum cardiac biomarkers such as troponin, ST-segment–elevation myocardial infarction and acute myocarditis based on clinical discoveries using elevated biomarkers, cardiac magnetic resonance imaging and electrocardiographic changes [47,48,49]. Children with harsh symptoms of COVID-19 were found to have vasculitis similar to Kawasaki disease [46,50,51,52,53,54]. The Sars-CoV-2 virus which triggers an inflammatory response in patients with COVID-19 also enhances the activation of platelets and the expression of tissue factor in these patients [46,55,56].

Due to the spread of Sars-CoV-2, a lot of time and resources are being allocated to patients with COVID-19 with little to no time and resources spent on patients with atherosclerosis [8]. Patients with atherosclerosis experienced delays in the hospital, rescheduling of important therapies and elective procedures, limited access to medical care and facilities and a reduction in time spent on diagnosing each patient risking diagnostic errors by physicians [8]. Individuals with atherosclerosis are more susceptible to being infected with Sars-CoV-2 [57], and poor prognosis, maybe due to diagnostic errors can lead to increased atherosclerotic risk which may lead to death.

5. Conclusions

The COVID-19 pandemic caused by SARS-CoV-2 has reduced access to emergency services and health care facilities leaving individuals with atherosclerosis at risk of increased morbidity or mortality. Patients with COVID-19 suffering from underlying cardiovascular disease conditions such as atherosclerosis are at a higher risk of COVID-19 related mortality. The use of drugs to combat COVID-19 has also been observed to have atherosclerotic side effects on individuals with atherosclerosis. Due to a high demand of packaged and ready to eat foods which are more likely unhealthy compared to fresh foods, fruits and vegetables, and a reduction in physical activity as one of the implications of the COVID-19 pandemic, the risk of atherosclerosis development and COVID-19 related mortality increases. Access to healthy foods should be increased especially to the vulnerable groups to boost their antioxidant and immune function which would aid in fighting off COVID-19 infection and reduce atherosclerotic risk. Proper and early diagnosis of cardiovascular diseases, cardiovascular comorbidities and diseases that put individuals at risk of atherosclerosis is important to prevent the occurrence of COVID-19 related mortality in patients with underlying health conditions. People with underlying diseases need to be made aware and conscious of their health condition so extra precaution can be taken to prevent COVID-19 infection. The cost of health insurance should be subsidized making it more affordable to people with underlying health conditions and at risk of COVID-19 infection such as the vulnerable groups. Urgent care should also be given to these groups to prevent increased morbidity or mortality as a result of the COVID-19 infection exacerbating atherosclerosis.

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Conflicts of Interest

The authors have no conflicts of interest.

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This article is dedicated to all medical personnel around the globe who are in the front line of the COVID-19 pandemic.

Abbreviations

COVID-19, coronavirus disease 2019;
CVD, cardiovascular disease;
LDL-C, low density lipoprotein cholesterol;
HDL, high density lipoprotein;
SARS-CoV-2, severe acute respiratory syndrome coronavirus 2;
RNA, ribonucleic acid;
MERS-CoV, middle east respiratory syndrome coronavirus;
LDL, low density lipoprotein; MI, myocardial infarction

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