

Systematic Review on Association of Various Disease with Covid-19

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ABSTRACT

The SARS-CoV-2 virus, which causes COVID-19, initially emerged in Wuhan City, Hubei Province, China, in early December 2019. On January 30, 2020, the World Health Organisation proclaimed the pandemic a Public Health Emergency of International Concern. There have been 49,053 confirmed cases and 1,381 fatalities globally as of February 14, 2020. The risk of developing severe COVID-19 is increased in some congenital disorders, according to reported studies, because these conditions often come with pre-existing co-morbidities that impact the circulatory and respiratory systems, leading to potentially life-threatening pneumonia. A high risk for severe COVID-19 infection is not associated with congenital disorders other than those listed, even though they cause psychological discomfort in patients. A higher risk of developing new-onset diabetes among survivors was associated with COVID-19, according to this review. Glucose dysregulation must be closely monitored throughout the recovery process from SARS-CoV-2 infection. The SARS-CoV-2 virus has unleashed a COVID-19 pandemic that rivals the size of the 1918 flu pandemic. Although respiratory symptoms predominate, neurological symptoms are also being more acknowledged as a possible clinical manifestation. It is expected that SARS-CoV-2 will create many issues impacting the central nervous system or peripheral nervous system, based on what is known about previous coronaviruses like the ones that caused SARS and the Middle East respiratory syndrome outbreaks. Diabetes mellitus and arterial hypertension are risk factors for cardiovascular disease; the impact of COVID-19 on these conditions may not be fully recognised. Recent research suggests that a family history of COVID-19 infection may raise the risk of various cardiovascular diseases to a level comparable to those of established risk factors for this condition. Furthermore, the risk of developing CVD and the severity of its symptoms are both increased in patients with a preexisting condition, such as myocarditis, acute coronary syndrome, heart failure (HF), thromboembolic consequences, or arrhythmias. Direct viral penetration into cardiovascular tissue and activation of a robust systemic inflammatory response are two hypothesised methods by which COVID-19 may influence CVD and CV risk.

Keywords- COVID-19, Cardiovascular disorder, Diabetes mellitus, Hypertension, Neurological disorder.

I. INTRODUCTION

The COVID-19 pandemic, which started in Wuhan, Hubei province, China, in late December 2019, is currently sweeping the nation. Formerly known as Novel Coronavirus 2019, the virus was renamed by the International Committee on Taxonomy of Viruses (ICTV) because of its striking resemblance to Severe Acute Respiratory Syndrome-Coronavirus-2 (SARS-CoV-2). The CDC in China had originally assigned the original name. The SARS-CoV-2 pandemic has been renamed by the World Health Organisation (WHO) to Coronavirus Disease-2019 (COVID-19)[1]. Symptoms

such as fever, cough, and difficulty breathing are caused by COVID-19, which mainly affects the respiratory system because to its effects on lung tissue. Recent investigations have demonstrated that it can impact several organ systems and lead to the emergence of extra-pulmonary symptoms. Extra-pulmonary symptoms might delay the identification of COVID-19 and may result in misdiagnosis, which can harm patients[2]. Understanding the different ways COVID-19 might manifest clinically is crucial for early diagnosis and treatment to reduce the disease's negative impact on health and mortality rates.

II. HISTORY AND ORIGIN OF COVID 19

A positive sense genome characterizes coronaviruses, which are RNA viruses that are encapsulated. The name "coronavirus" comes from the fact that these viruses, which can be anywhere from 60 nm to 140 nm in size, have spike-like projections on their surfaces that, when seen under an electron microscope, look like a crown. Currently, four coronaviruses HKU1, NL63, 229E, and OC43 are making the rounds among humans and are known to cause mild to severe respiratory disease[3]. Serious human sickness has resulted from the transmission of animal betacoronaviruses on two separate occasions in the past twenty years. In the years 2002–2003, in the Chinese province of Guangdong, a new coronavirus of the β genus that originated in bats was passed on to people by palm civet cats[4]. Before it was contained, the severe acute respiratory syndrome coronavirus, or SARS, affected 8,422 people mostly in China and Hong Kong, killing 916 people (or 11% of the total)[5]. Saudi Arabia was hit by the bat-borne Middle East respiratory syndrome coronavirus (MERS-CoV) in 2012. The intermediate host, which was dromedary camels, inflicted mortality on 858 people out of 2494 (or 34% of the total)[6].

Transmission of COVID 19

Since most of the first cases were associated with being in wet marketplaces, zoonotic transmission seemed to be a real possibility. A greater percentage of people who were uninvolved with the market or any other person exhibiting respiratory symptoms caught the disease by the end of January 2020. The transmission of the sickness to individuals who had not traveled to Wuhan and to healthcare professionals indicated a human-to-human spread of the virus. The precise method of transmission of this virus remains unidentified. However, like other respiratory viruses, the main mechanism of transmission is likely through droplets, either directly or indirectly via contaminated objects. Currently, there is no proof of the virus being transmitted through the air[7,8]. A possibility for fecal-oral transmission has been suggested by the presence of viral particles in stool samples from both symptomatic and recovered individuals, however the danger is still unknown.

Interaction of COVID 19 with congenital heart disease

In the early stages of the COVID-19 pandemic, heart disease was identified as a potential killer. A high-risk population prone to numerous cardiovascular complications and concerns are adults with congenital heart disease (CHD), which affects roughly 1.5 million people in the US and 2.5 million in Europe. As a result, these individuals are seen as having a higher chance of adverse events and are consequently forced to adapt to a more constrained way of life and career paths[9]. Patients with congenital cardiac abnormalities, such as

cyanotic lesions or unrepaired cyanotic defects (Eisenmenger syndrome), were at significantly higher risk of contracting SARS-CoV-2, according to a recent clinical research involving 105 patients. According to research, SARS-CoV-2 binds more preferentially to the ACE2 receptor than SARS-CoV. It is possible that COVID-19 damages the cardiovascular system in part because ACE2 is present on myocardial cells and the virus is able to directly access these cells[10]. A higher mortality rate is observed in COVID-19 patients with pre-existing CVD compared to COPD patients, according to the research. The specific mechanism by which SARS-CoV-2 causes damage to cardiomyocytes is still unknown. A cytokine storm set off by COVID-19 has been proposed as a possible explanation. Cardiomyocytes in acute respiratory distress syndrome (ARDS) may be subject to oxidative stress due to an increased oxygen demand in hypoxic environments, according to another. Despite the lack of definitive evidence, recent research implies that COVID-19 may increase the risk of cardiovascular complications and intensive care unit hospitalization for individuals with CHD. When comparing patients with suspected COVID-19 to those with proven COVID-19 and coronary heart disease (CHD), the incidence of cardiovascular difficulties is higher in the latter group. Arrhythmias(22%), heart failure(55%), and stroke (22% of all cases) are among the most prevalent complications[11]. The risk of contracting COVID-19 during cardiac repair surgery has not been the subject of any research. Overall, 34% of patients with perioperative SARS-CoV-2 infection died after surgery, with pulmonary problems affecting 94.1% of patients undergoing heart surgery, according to a recent study on postoperative mortality. Male gender, advanced age (70 or more), ASA grades 3-5, malignant illness surgery, emergency surgery, major surgery, and emergency surgery were all identified as risk variables[12]. A separate study that focused on children proposes sorting congenital heart defects by kind in order of surgical importance. Operations can be classified as either urgent (within one to two weeks), scheduled for more than two weeks, or emergency (within 24-48 hours). Adolescents are mostly unaffected by SARS-CoV-2 infection, but older patients with coronary heart disease who are obese and have other comorbidities are at a higher risk of consequences[13]. Careful consideration of the potential risks is required before giving these patients surgery consent.

COVID- 19 impact on cardiovascular disease

As of January 2023, over 670 million people around the world have been affected by COVID-19, which was triggered by SARS-CoV-2 infection. The coronavirus has been associated with almost 7 million fatalities[14-17]. Further, evidence of Long COVID-19 is mounting; this form of the virus causes delayed issues or chronic symptoms to appear weeks after the first onset of COVID-19 symptoms. The severity of the illness is

associated with preexisting cardiovascular risk factors and diseases, as was seen shortly after the pandemic started[18]. Twenty to thirty percent of COVID-19 patients hospitalized also had elevated troponin levels. Patients already suffering from cardiovascular disease may see their clinical outcomes worse as a result of this[19]. This increased number of cases of sickness and mortality from COVID-19 is likely due to the unfavorable cardiovascular implications caused by both the direct and indirect effects of SARS-CoV-2 infection[20]. Although COVID-19 treatments and vaccines can alleviate symptoms to some extent, they also carry the risk of cardiac adverse effects that might compromise heart health[21-24]. According to the Expert Consensus Decision Pathways on cardiovascular sequelae of COVID-19 in adults, the most recent publication from the American College of Cardiology highlighted the clinical significance of the two-way relationship between COVID-19 and cardiovascular illness[25]. In this review, we look at how COVID-19 increases the risk of cardiovascular illness, how immunization and therapy affect COVID-19 and cardiovascular health, and how the two are related[26-28].

Interaction of COVID 19 with Diabetes mellitus

Getting SARS-CoV-2 while suffering from a greater death rate is the result of a cascade of interrelated complications caused by diabetes mellitus[29]. People with COVID-19 are at increased risk for developing high blood sugar, which can lead to an overabundance of sugar binding to ACE2 and, ultimately, the virus's rapid replication. Inflammation, dysfunction of the inner lining of blood vessels, and blood clot formation are all consequences of inflammation brought on by an imbalance in glucose processing and an increased tendency for blood clotting, which are all exacerbated by high blood sugar[30]. A worsening of thrombotic and ischemic effects connected to multiorgan failure and greater mortality rates can occur in patients with a propensity to vasculopathy and poor immunity when they have a severe illness. Inconsistent signaling in glucose metabolism-related pathways can diagnose all forms of diabetes mellitus. Because of the association between diabetes mellitus and the progressive damage to both big and small blood vessels, the condition is categorized as a vasculopathy similar[31]. There are numerous paths via which vascular disease develops in people with diabetes, including persistently high blood sugar levels and impaired insulin sensitivity. Oxidative stress and faulty AGE-RAGE signaling are involved in these[32]. Vascular damage can be accelerated by the AGE-RAGE axis, which involves increased synthesis of AGEs and stimulation of RAGE receptors. Endothelial cells of large and small arteries undergo changes in mitochondrial superoxide expression as a result of metabolic issues associated with oxidative stress. Even if blood glucose levels normalize, proinflammatory pathways are still activated due to a cascade of

epigenetic alterations caused by elevated superoxide production over time.

Abnormal cytokine responses, decreased leukocyte recruitment, and neutrophil dysfunction are some of the alterations that occur in the innate and adaptive immune systems as a result of chronic hyperglycemia. Acute viral infections can worsen chronic hyperglycemia because the immune system reacts by producing systemic insulin resistance, which in turn makes hyperglycemia worse[33]. Severe COVID-19 progression is highly associated with elevated blood glucose levels. In patients with SARS-CoV-2 infection and diabetes mellitus, monitoring blood glucose levels is more useful for prognosis than hemoglobin A1c (HbA1c) for tracking rising hyperglycemia. Findings suggest that high blood sugar declines rapidly and may cause rapid clinical deterioration in people with preexisting vascular disease and compromised endothelial function[34]. There is a complex relationship between COVID-19 and DM, and aberrant immune responses may exacerbate the disease by hastening the thrombotic and ischemic effects of the virus, which are associated with increased mortality rates and multi-organ failure[35].

COVID 19 impact on Neurological disorders

In cases of SARS, MERS, and COVID-19, neurological complications are rare. Even a tiny percentage could lead to a large number of patients because of how broad the present pandemic is. As for peripheral nervous system (PNS) complications, the lowest incidence varied between 0.05% for SARS and 0.16% for MERS, whereas the lowest incidence of central nervous system (CNS) complications varied between 0.04% for SARS and 0.20% for MERS. Estimates of the number of COVID-19 cases involving neurological problems were based on these numbers[36]. Out of 4.8 million cases of COVID-19 globally, an estimated 1,805–9671 people are dealing with issues related to the central nervous system (CNS), while another 2,407–7737 are dealing with issues related to the peripheral nervous system (PNS). Ignoring newly reported symptoms of stroke and COVID-19 infection, these numbers will rise if the pandemic continues. With 901 cases reported so far, the number of cases involving neurological symptoms and COVID-19 is rising. Symptoms can be viewed as neurological difficulties caused by COVID-19 as a whole, immune-mediated disorders that manifest around the time of infection, or direct effects of the virus on the brain system[37]. The three-week period was recorded by a nationwide registry for 125 individuals with COVID-19 and neurological or mental disorders. Alterations of mental state occurred in 39 patients (31%). Of the 16 patients (or 13% of the total) diagnosed with encephalopathy, 7 (or 6% of the total) had encephalitis. A total of 23 patients (18%) were also diagnosed with a neuropsychiatric disorder; among them, 10 patients (8%) were found to be suffering from psychosis, 6 patients (5%) from neurocognitive

syndrome, and 4 patients (3%) from emotion disorders. In the group of patients studied, 77 people, or 62% of the total, had a cerebrovascular event. Among them, 57 were ischemic strokes, nine were intracerebral hemorrhages, one was central nervous system vasculitis, and ten were other cerebrovascular events.

Interaction of COVID 19 with Hypertension

According to studies, between fifteen percent and thirty percent of COVID-19 patients had a history of hypertension. Hypertension was found in over 75% of the people who died in an Italian epidemic[38]. New evidence suggests that hypertensive patients are at a higher risk of contracting COVID-19 than the general population. A meta-analysis systematic review indicated that the risk of acquiring severe forms of COVID-19 is 2.5 times higher in people with hypertension (Odd Ratio, OR 2.49). With an odds ratio of 2.42, the mortality risk showed a continuous trend[39]. Because of their vasodilatory and vasoconstrictive characteristics, the ACE2 and ACE enzymes regulate blood pressure and pH. The enzyme angiotensin-converting enzyme 2 (ACE2) starts by using angiotensin 1 (Ang 1) as a substrate[40]. It breaks it down into angiotensin 1-9, which ACE2 uses again as a substrate to make angiotensin 1-7. Vasodilatory, anti-inflammatory, and protective actions are exhibited by these substances. One of ACE2's roles is to bind to the SARS-CoV virus[41-43]. When an infection starts in the respiratory system, it is usually because epithelial cells lining the airway express ACE2. Additional ACE2 generators include the kidneys, heart, and intestines. The spike protein is the mechanism by which the SARS-CoV-2 virus binds to ACE2 receptors[44]. Increased ACE2 production is associated with increased vulnerability to SARS-CoV infection, according to laboratory studies[45]. To control hypertension, many people take medications called angiotensin-converting enzyme inhibitors or angiotensin receptor blockers[46]. Although these medications mostly inhibit ACE1 activity, they have also been observed to increase ACE2 activity. The risk of infection and sickness contraction is raised when ACE2 levels are high, as is the case with SARS-CoV and SARS-CoV-2[47]. As a result, the risk of a life-threatening infection increases.

Although RAS inhibitors do not show any increased sensitivity to infections, ACEIs prescription may increase vulnerability to infections. Lisinopril increased ACE2 mRNA expression in animals while keeping ACE2 levels unchanged, according to the study[48]. By increasing the conversion of Angiotensin 2 to Angiotensin 1-7, ACE2 protects lung tissue, according to previous experimental study[49]. The inflammatory process in the lungs is reduced as a result. Reducing inflammation in the lungs, heart, and kidneys, angiotensin 1 receptor blockers reduce the likelihood of developing acute renal damage, myocarditis, or acute respiratory distress syndrome[50]. One possible alternative to ACE2 in the treatment of hypertension is

calcium antagonists. They do not seem to affect the inflammatory response in COVID-19 patients' lungs. The results were corroborated by a multicenter study included 1128 COVID-19 individuals with hypertension, 188 of whom were on an ACEI or ARB medication[51-53]. Those who used antihypertensive medication had a much reduced risk of death from any cause (3.7% versus 9.8%, $P = 0.01$). The link between COVID-19 and hypertension needs further investigation[54].

III. CONCLUSION

The widespread spread of the COVID-19 virus and the ensuing lockdown procedures have increased anxiety for many who suffer from autoimmune illnesses. Due to immunosuppressive treatment, organ dysfunction induced by auto antibodies, and less medical attention during the pandemic, patients with autoimmune diseases exhibit heightened anxiety in response to COVID-19. Restriction has a major effect on these people' physical and mental wellbeing. The exact antiviral and immunomodulatory therapy that individuals with COVID-19 infection and autoimmune disorders (ADs) receive can have a significant impact on their prognosis, either making their condition worse or better. According to the review, COVID-19 was found to be associated with an increased risk of developing new-onset diabetes in survivors. Whilst recovering from a coronavirus 2 infection with severe acute respiratory syndrome, it is critical to keep a close eye on glucose dysregulation. Coronary artery disease was associated with COVID-19 mortality and severity. However, these findings need to be confirmed by further thorough examinations. Anyone experiencing symptoms of COVID-19 or having come into contact with an infected person should seek medical advice immediately; this is especially true for those who have a history of cardiovascular disease, are elderly, or have other health problems. Doing so will aid in halting the decline in their health.

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