A Multi Organ Involvement due to COVID-19: A Study Report

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ABSTRACT

SARS-CoV-2, a new coronavirus strain, first appeared in China in November 2019 and has since spread to over 150 countries. SARS-CoV-2 not only causes pneumonia, but it also has the potential to affect other organs such as the heart, liver, and kidneys. The role of the lungs, kidneys, and heart in COVID 19, as well as the mechanism of lung, kidney, and heart damage and its influence on mortality, are discussed here. In late 2019, coronavirus-2, a significant human infection that causes the disease coronavirus disease in 2019, appeared as a major human pathogen (COVID-19). Acute respiratory failure, which is similar to the acute respiratory distress syndrome ARDS, is the most prevalent clinical manifestation of severe COVID-19. COVID-19 includes diseases of the airway, lung parenchymal, pulmonary vascular, and respiratory neuromuscular systems. The consequences of severe acute respiratory syndrome coronavirus-2 infection are discussed in this article. COVID-19 has the potential to have a major impact on the cardiovascular systems of individuals. For starters, those who have COVID-19 and prior cardiovascular disease are at a higher risk of developing severe illness and dying. COVID-19-related mortality is highly linked to cardiovascular disease, diabetes, and hypertension. Second, medications being tested for COVID-19 might cause arrhythmia in the cardiovascular system. Finally, COVID-19 has been linked to a variety of direct and indirect cardiovascular problems.

Keywords: *COVID19.*

1. INTRODUCTION:

As of March 24, 2020, there has been roughly 381,761 cases globally since the first case, which was discovered in Wuhan, China, in December 2019 by a novel strain of human coronaviruses, the severe acute respiratory syndrome coronavirus 2 (SARSCoV- 2). The World Health Organization (WHO) designated it as coronavirus illness 2019 on February 11, 2020. (COVID-19). It has already spread to over 150 nations throughout the world, claiming the lives of 16,558 people [1]. SARSCoV-2 is a Betacoronavirus, which means it's a positive-stranded RNA virus. It has a diameter of 60–140 nm and is frequently pleomorphic. Coronaviruses belong to the Coronavirinae family of viruses, which includes four genera: Alphacoronavirus, Betacoronavirus, Gammacoronavirus, and Deltacoronavirus. These are based on their genomic architecture and evolutionary connections. Only animals can be infected by alphacoronaviruses and betacoronaviruses. Gammacoronaviruses and deltacoronaviruses can infect animals in some cases. Human respiratory disorders are mainly caused by alphacoronaviruses and betacoronaviruses. SARS-CoV and MERS-CoV are highly pathogenic variants of human coronaviruses that may cause severe respiratory syndrome

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in humans, while other human coronaviruses strains such as (HCoV-NL63, HCoV-229E, HCoV-OC43, and HKU1) can also cause moderate upper respiratory infections [2]. The origin of SARS-Cov-2 is unknown at this time, however genomic investigations suggest that it evolved from a strain identified in bats (BatCov RaTG13) [3]. Human-to-human transmission, like that of other respiratory viruses, is mostly by aerosol, however transmission via faeces or direct touch has also been documented. According to information from examinations conducted by the China CDC, the incubation period is generally 3–7 days and can last up to 2 weeks [4]. The most common symptoms of COVID-19, according to clinical studies, are lethargy, fever, dry cough, sore throat, dyspnea, and diarrhoea [5]. The most prevalent laboratory anomaly is leukopenia. Consolidation or numerous ground-glass opacities involving both sides are shown on computerised tomography (CT) of the chest. COVID-19, which is primarily responsible for acute respiratory sickness, is not limited to the respiratory system; it may also harm other organs such as the kidneys, heart, gastrointestinal tract, immunological, blood, and brain systems. This paper aims to emphasise the impact of COVID 19 on the kidneys, with an emphasis on dialysis and renal transplant patients who are at risk [1].

Hypoxemia is a common clinical finding that arises out of proportion to the patients' perception of dyspnea. According to Gattinoni et al, substantial anomalies in ventilation-perfusion (V/Q) matching may accompany shunt physiology (perfusion of unventilated respiratory units), with disordered hypoxic vasoconstriction playing a crucial role. These researchers also noted differences between the severity of hypoxia and somewhat intact respiratory system compliance, indicating that significantly aberrant V/Q matching is a common hallmark of ARDS linked with COVID-19. Studies of larger patient populations with well-described associations between the disease course, clinical and radiographic findings, treatments, coinfections, and histopathology will be required to delineate the contribution of viral infection, immune-mediated damage, or ventilator-associated lung injury to the observed pathophysiology [6, 7].

Patients with COVID-19 have normal lung parenchyma, ground-glass opacities, localized consolidations, and pulmonary vascular perfusion anomalies, according to radiographic examinations. Although comprehensive evaluations imply that there is no pathognomonic CT pattern, ground-glass opacities in bilateral, peripheral, and lower lobe distribution appear to be the most prevalent pattern on computed tomography (CT) scanning. When COVID-19–associated pneumonia CT scans are compared to CT scans of other viral pneumonias, it seems that the peripheral distribution of opacities, a ground-glass look, fine reticular appearance, and vascular thickening are more apparent in COVID-19[8].

1.1 Epidemiology:

COVID-19 can induce viral pneumonia, as well as other cardiovascular issues. In early investigations in China, 32 percent to 46 percent of patients hospitalised with COVID-19 had underlying illnesses such as hypertension (15 percent to 31 percent), cardiovascular disease (14.5 percent to 15 percent, and diabetes (10 percent - 20 percent). The prevalence of hypertension, cardiac and cerebrovascular disease, and diabetes was found to be 17.1%, 16.4%, and 9.7%, respectively, in a meta-analysis of six COVID-19 investigations. The frequency of cardiovascular illness

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in COVID-19 populations varied greatly depending on the research scale, ranging from 40% in a study of 99 COVID-19 patients to 2% to 4% in large studies of more than 1000 COVID-19 patients [3, 9].

Male sex, advanced age, and the prevalence of hypertension, diabetes mellitus, cardiovascular illnesses, and cerebrovascular disorders, as well as consequences of acute cardiac injury, cardiomyopathy, and heart failure, are all related with death in COVID-19 patients[3,10]. Those with coronary heart disease had a fatality rate of 10.5 percent greater than the general mortality rate of 2.3 percent in a large cohort of 44,672 COVID-19 patients [3, 11]. The greatest death rate was linked to the presence of coronary heart disease and myocardial damage. COVID-19 has an effect on the following organs.

2. ORGANS EFFECTED DUE TO COVID-19

2.1 Infection of Lungs by SARS-CoV2

The viruses of the coronavirus family, including SARS-CoV2, are thought to enter the human body mostly through the mucosa of the nose and oropharynx, with some ending up in the lungs. Other organs that express angiotensin-converting enzyme 2 (ACE2) receptors on the cell surface, such as the heart, kidney, and liver, are also susceptible to SARS-CoV2 infection [1,12]. The most likely binding site of the SARS-CoV2 particles to the host cell is ACE2 receptors, which are largely membrane-bound aminopeptidases. When opposed to matured or differentiated cells, immature or undifferentiated cells have a very low level of ACE2 expression, making matured cells more sensitive to the virus (Figure 2) [1,13].

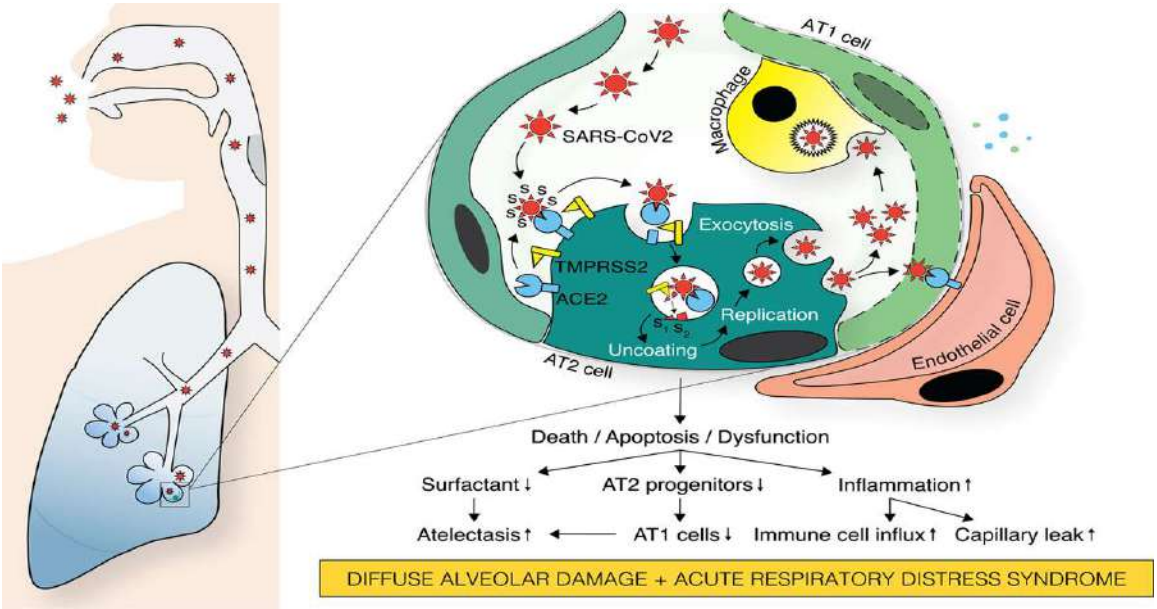

Figure 1. The pathobiological repercussions of severe acute respiratory syndrome coronavirus-2 (SARS-CoV2) infection on alveolar epithelial damage.

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2.2 Kidney involvement in COVID-19 infection

A worry for renal damage was expressed by chen et al. in a report of 710 COVID-19 patients, who found proteinuria and hematuria in 44 percent of patients and hematuria in 26.9% of patients at the time of admission. In 15.5 percent of individuals, the serum creatinine level was elevated. During the trial period, an overall incidence of acute kidney injury (AKI) was documented in 3.2 percent of participants. Another research by Zhen Li et al. found that proteinuria was more common in 63 percent of patients (32/51) and that serum creatinine and blood urea nitrogen levels were greater in 19 percent and 27 percent of patients, respectively. CT scans revealed renal abnormalities in 100 percent of the patients [2, 14].

2.2.1 Mechanism of kidney injury in COVID-19

The whole pathophysiology of COVID-19 kidney damage is yet unknown, however it appears to be complicated and heterogeneous (shown in Fig. 2). To begin with, certain antecodal findings showed PCR fragments of coronavirus in the blood and urine of patients infected with SARS and COVID-19, suggesting that new coronavirus might have a direct cytopathic effect on kidney resident cells. SARS-spike CoV-2's (S) protein employs angiotensin-converting enzyme II (ACE2) as a cell entrance receptor and TRMPSS as a cell entry receptor. In the kidneys, ACE2 is strongly expressed [2, 15-18].

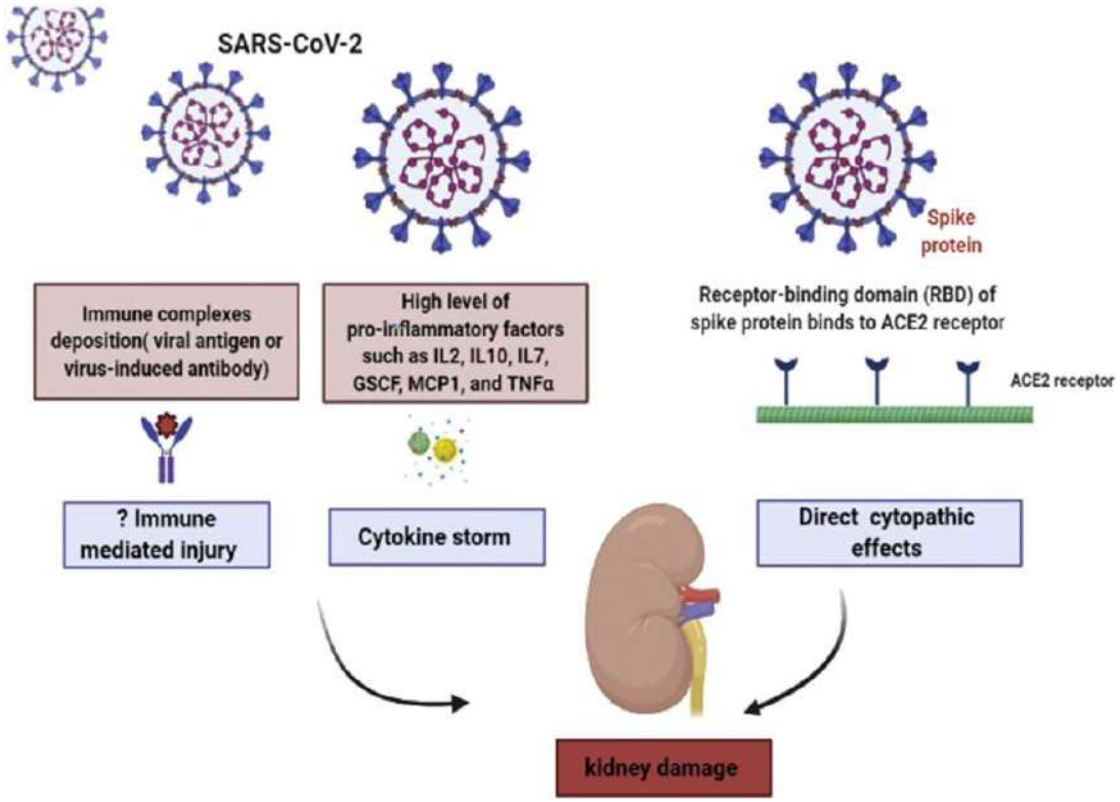

Figure 2: SARS-CoV-2 kidney damage pathogenesis

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2.3 HEART INVOLVEMENT IN COVID-19 INFECTION

2.3.1 Myocarditis

One of the most prevalent causes of myocarditis has been identified as virus infection. Acute cardiac damage complications were seen in 12 percent to 8% of COVID-19 patients, with the incidence being almost 13 times greater in ICU/severe patients than in non-ICU/severe patients [19].

Patients who died showed greater levels of troponin, myoglobin, C-reactive protein (CRP), serum ferritin, and interleukin-6 among 150 patients with laboratory-confirmed COVID-19 (IL-6). Some individuals died of fulminant myocarditis or "cytokine storm syndrome" caused by the virus, indicating that COVID-19 has a high inflammatory load and a potential increase in myocarditis-related cardiac events. The primary pathophysiological mechanism of fulminant myocarditis caused by COVID-19 is similarly an exceptionally powerful cytokine storm [3,20].

In extreme situations, acute heart damage as measured by increased high-sensitivity troponin levels is prevalent. Higher levels of N-terminal pro-brain natriuretic peptides (NT-proBNP) (27.5%) and cardiac troponin T (TnT) (10%) were linked to drastically increased plasma IL 6 levels in a study of 120 SARS-CoV-2-infected individuals. According to Guo et al., 28 percent of 187 COVID-19 patients hospitalised suffered an acute myocardial infarction (defined as elevated TnT). Inflammatory indicators such as leukocytosis, lymphopenia, dimer, CRP, and procalcitonin were also increased in patients with elevated TnT levels. In COVID-19, myocardial damage is a significant prognostic factor and is closely linked to death [3,21].

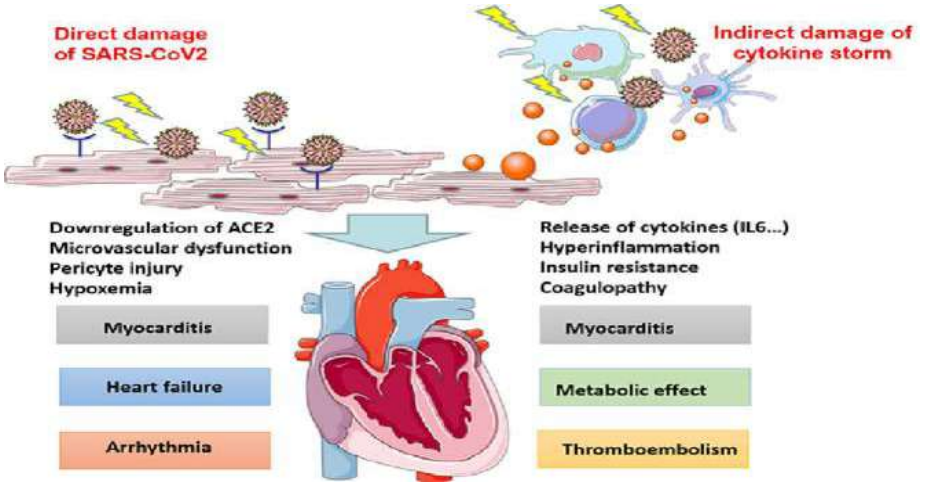

Figure. 3:. SRAS-CoV2 infection causes direct harm to the heart and circulatory system.

2.3.2 Heart Failure

In 23 percent of 191 inpatients from Wuhan, China, COVID-19 had the potential to induce heart failure. Following COVID-19, cases of severe myocarditis with impaired systolic function have been documented. Acute cardiac damage (72/94; 77 percent) and heart failure (41/83; 49 percent) were frequent cardiac consequences in a study of 113 COVID-2019 patients who died. In a case series of 150 COVID-19 patients, 7 percent of mortality were related to myocarditis with circulatory failure. It's still unclear whether heart failure is caused by an aggravation of

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underlying left ventricular dysfunction or by a new cardiomyopathy (either owing to myocarditis or stress cardiomyopathy) [3, 22-24].

Cardiac pericytes with strong ACE2 expression might be SARS-target CoV-2's cardiac cell.23 Pericyte damage caused by viral infection can cause capillary endothelial cell malfunction, which can lead to microvascular dysfunction. Patients with basic heart failure had greater levels of ACE2 expression in both mRNA and protein, indicating that if infected with the virus, they may be at a higher risk of heart attack and critical illness. The findings of this study explain why COVID-19 individuals with basic cardiovascular disease have such a high proportion of severe cases [24-26].

3. CONCLUSION:

A study on A Multi Organ Involvement due to COVID-19 is presented based on the current scenario of the pandemic COVID-19. It is observed that the major organs were damaged due to COVID-19 and present understanding of risk factors of respiratory tract infection indicates several approaches for primary prevention. In developed countries improved living standards, better nutrition, reduction of smoke and consumption of Alcohol will reduce the burden of mortality and morbidity associated with ARI.

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