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# Covid as an immunosuppressive disease a Review

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**Abstract-** The three symptoms of coronavirus disease (COVID-19) that patients most often mention are cough, fever, and shortness of breath. However, other significant illnesses that are frequently observed in COVID-19 patients (such as diabetes mellitus, hypertension, kidney disease, neurologic disorders, arterial and venous thromboembolism, and kidney disease) imply that the virus is specifically attacking the endothelium, one of the largest organs in the human body. We provide here a systematic and comprehensive analysis of both clinical and preclinical evidence in support of the idea that the endothelium is a major target organ in COVID-19 and give a mechanistic explanation for its systemic symptoms.

**Keywords**: heparin, endothelium, cytokine storm, cathepsin, coronavirus, COVID, acute kidney injury, ACE2, blood pressure,and Kawasaki disease.

## INTRODUCTION

Worldwide, the COVID-19 coronavirus disease outbreak represents a serious public health crisis. [1-7]. The first case of COVID-19, which is brought on by the SARS-CoV-2 coronavirus, was discovered in Wuhan, the capital of China's Hubei province, in December 2019 [1-7]. Covid-19 is a viral disease so only vaccine can be done as prevention. Viral diseases don't have any treatment [8-11] Cough, fever, and shortness of breath are the symptomsthat are stated the most often. Why respiratory symptoms are so widespread is explained by the disease's pathogenesis: In actuality, the virus is able to penetrate host cells thanks to the angiotensin-converting enzyme 2 (ACE2) protein, which is widely present in the lungs. However, endothelial cells also express ACE2 [12-14], andthese cells also express other important clinical outcomes that are often seen in COVID-19 patients, including hypertension and thrombosis. The endothelium of the kidney, one of the major human organs, has been suggestedas the virus' potential target (disease, pulmonary embolism, cerebrovascular and neurologic diseases). Young COVID-19 patients have shown signs of Kawasaki disease, which supports our notion that SARS-CoV-2 caused a systemic vasculitis .

## THE PATHOPHYSIOLOGY OF COVID-19

Spike has been discovered as a co-receptor for coronavirus entry. Spike is a surface glycoprotein (peplomer) that SARS-CoV-2 uses to enter host cells [3]. Therefore, the density of ACE2 in each tissue may be connected to howsickly that tissue is [4]. Other receptors have been suggested to aid SARS-CoV-2 entry into human cells, includingtransmembrane serine protease 2 (TMPRSS2), sialic acid receptors [4], and extracellular matrix metalloproteinaseinducer (CD147, also known as basigin) [6]. Furthermore, catepsins B and L have been shown to be significant entry factors in the pathogenesis of COVID-19.

## A RENAL ILLNESS COVID-19 REPORT

More than 20% of COVID-19 patients who are critically ill or die have been shown to have acute kidney injury (AKI), a statistic that is consistent across studies from China [15], Italy, and the United States It is important to note that AKI, proteinuria, and hematuria have all been independently associated to a higher risk of dying in COVID-19 individuals . Furthermore, a meta-analysis of 1389 COVID-19 patients revealed that those with a severe COVID-19 condition had a much-increased risk of having underlying chronic renal disease (3.3 percent vs. percent; odds ratio 3.03, 95 percent confidence interval: 1.09-8.47). A single-cell analysis research has shownthe

expression of ACE2 and TMPRSS2 in human renal endothelial cells and most recently, electron microscopy has demonstrated the presence of viral particles in the endothelial cells of the glomerular capillary loops of a COVID-19 patient. According to immunohistochemistry tests, renal endothelial cells do not express ACE2. Additionally, endothelial damage was a common finding in the renal histological examinations of 26 COVID-19 patients [16] in the absence of interstitial inflammatory infiltrates.

## DIABETES AND HIV-19

Diabetes mellitus is a prevalent co-morbidity in COVID-19 patients and is linked to a poorer outcome . In fact, 20% of patients who had pneumonia episodes with uncertain etiologies and a history of exposure to the Huanan seafood market before January 1, 2020, also had diabetes [24]. Similar findings were obtained in the 1099 COVID- 19 patients investigated by Guan and colleagues: 7.4% of patients developed diabetes, and for those with severe illness (vs. 5.7%), this number rose to 16.2% . Additionally, compared to 13.7 percent, 35.8 percent of patients who suffered the composite outcome of ICU admission, mechanical ventilation, and mortality had diabetes. According to Italian statistics, more than two-thirds of COVID-19 patients who did not survive had diabetes. In summary, diabetes is a prevalent co-morbidity, a risk factor, and an independent prognostic factor in COVID-19 patients. Furthermore proving that diabetes has negative impacts on COVID-19 patients are two meta- analyses.[17]

## THROMBOSIS AND COVID-19

Patients with COVID19 often exhibit coagulation problems, organ failure, and coagulopathy, which are associatedwith higher mortality [18]. Important data were obtained from examinations of samples collected from COVID- 19 patients upon admission and during their hospital stay for the prothrombin time (PT), activated partial thromboplastin time (APTT), antithrombin activity (AT), fibrinogen, fibrin degradation product (FDP), and D- dimer . Non-survivors had prolonged PT and significantly higher D-dimer and FDP levels than survivors at admission. In the latter stages of their hospitalisation, non-survivors also had their fibrinogen and AT levels dramatically decreased and dropped, which is compatible with a clinical diagnosis of disseminated intravascular coagulation (DIC). Particularly, among 191 COVID-19 patients investigated at two hospitals in Wuhan, D-dimerlevels > 1 g/L at admission suggested an 18-fold increase in the odds of dying before discharge. Be aware that DICcaused by a systemic infection displays an early, overly inflammatory response that is only linked to endothelial damage [19]. Antibiotics could be used for treatment of secondary infections [20,21,22] as Covid-19 is immune suppressive disease (the disease which decrease the immunity[23]

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