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Review

Silent Invasion: COVID-19's Hidden Damage to Human Organs

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Abstract

This review considers that many organs other than the lungs are affected by COVID-19. Early research largely concentrated on lung damage, although new information indicates that the virus damages at least ten primary body systems: cardiovascular, neurological, renal, hepatic, endocrine, hematological, gastrointestinal, immune and mental health. The author considers clinical trials, pathology, imaging results and biomarker investigations published by mid-2022. Common abnormalities include endothelial dysfunction, chronic inflammation, cytokine storms and microvascular damage, all of which contribute to the pathogenesis of the disease in the entire body. In this article, mild infections may lead to severe conditions including but not limited to myocarditis, brain fog, kidney disorders or immune system burnout. It also examines existing and emerging treatments-antifibrotics, immune modulators and vascular-targeted therapies-and demands a standardized approach to monitoring and management of patients on a long-term basis. This review highlighting that COVID 19 is a long-term systemic disease that requires a multidisciplinary approach.

Keywords: COVID-19; multi-organ damage; long COVID; systemic disease; post-acute sequelae; pathophysiology

1. Introduction

The COVID-19 pandemic has impacted much more than infectious diseases since its onset in late 2019. Initial disease prevention campaigns focused on preventing acute respiratory diseases. Having seen the consequences of SARS-CoV-2, we understand that the virus may target numerous body parts and leave permanent effects on the body. Official statistics indicate more than 777 million confirmed cases and at least 7 million deaths worldwide as of June 2025. However, there exists a backwater crisis, that is, long-term injury to a number of organs in survivors.

Existing research indicates that individuals who experience mild or even moderate illness can also develop difficulties with their cardiovascular, neurological, renal, hepatic and immune systems in the future. This damage is the result of several factors: viral persistence, endothelial injury, immune dysregulation, microvascular thrombosis and extended inflammation. Despite our current substantial body of research, there is still a lot of research organized in a manner that makes it organ- or symptom-specific which leaves individual practitioners with partial counsel.

The seriousness of post-COVID complications has been confirmed by recent cohort and imaging studies, particularly in long COVID, also known as post-acute sequelae of SARS-CoV-2 infection (PASC). Even several months after the infection, patients complain of long-term fatigue, shortness of breath, memory and cognitive issues ("brain fog") and cardiac problems. In some cases organ damage becomes visible only with subsequent testing, indicating a dissonance between the perceived sense of the patients and the objective level of health. Meta-analyses and systematic reviews

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concentrate on a single outcome including myocardial inflammation or pulmonary fibrosis but an integrated picture of multiple-organ disease remains lacking.

This review addresses this gap. In discussing the clinical, pathological and mechanistic evidence, it provides a generalized view of the impact of COVID-19 on ten essential systems: the respiratory, cardiovascular, neurological, renal, hepatic, gastrointestinal, endocrine, hematological, immune and psychological systems. The common denominators are endothelial dysfunction, cytokine storm and oxidative stress.

The most recent longitudinal studies, biomarker studies and treatment trials were also included in this review. It does not enumerate the damages separately but rather the relationship between the various damages. It discusses possible treatment options including antifibrotic drugs, immunomodulatory agents and vasculature-based treatments which could delay and reverse the loss of organ function.

The important conclusions are as follows:

The systemic basis is also supported by clinical, pathological and mechanistic data indicating that COVID-19 is an illness of 10 major physiological systems without organ specificity. Long-term organ damage is common and may persist even after symptoms are resolved.

The combination of cross-disciplinary data demonstrates shared pathways, including endothelial dysfunction, cytokine storms and oxidative stress.

Emerging therapies, such as antifibrotic agents, immunomodulators and vascular-directed treatments, may be useful in reducing or restricting organ damage. An integrated surveillance, collaboration and intervention approach is required for early detection, risk stratification and focused therapy. To achieve better results in the treatment of patients and the sustainable development of health systems in the future, it is paramount to consider COVID-19 a systemic disease.

2. Respiratory System

Viral Pneumonia- The most frequent lung issue developed due to SARS-CoV-2 infection is viral pneumonia which is usually caused by COVID-19. The alveolar epithelial cells of the lungs are invaded by the virus which leads to localized inflammation and fluid buildup. This inflammatory exudate increases the difficulty of oxygen getting to the blood and carbon dioxide getting out. Symptoms include fever, chronic cough, chest pain and progressive inability to breathe. In many cases, bilateral peripheral ground-glass opacities and patchy consolidations recorded by high-resolution CT scans are helpful in distinguishing between COVID-19 pneumonia and usual bacterial pneumonia. Disease development may be rapid, especially in the elderly or other individuals with underlying medical conditions. In extreme cases, diffuse alveolar damage can cause life-threatening respiratory failure and necessitate interventions to provide support (Xu & Shi, 2023).

ARDS Acute Respiratory Distress Syndrome (ARDS) The acute respiratory distress syndrome (ARDS) is a serious and often fatal condition to which a large proportion of severe COVID-19 patients develop. ARDS initially presents with pulmonary inflammation, elevated alveolar-capillary membrane permeability and massive pulmonary edema, resulting in poor lung compliance and impaired oxygenation. The combination of direct viral lung injury and the so-called cytokine storm (hyperinflammatory systemic reaction) aggravates lung damage. Clinically, ARDS is manifested by the onset of dyspnea, critical hypoxemia that cannot be eliminated by supplementary oxygen and diffuse pulmonary infiltrate on imaging tests of the chest. Despite the lowered COVID-19 ARDS mortality rates due to advances in critical care, the ailment is associated with a high mortality probability. A characteristic of COVID-19 ARDS that has not been previously described with ARDS due to other causes is what is termed silent hypoxemia: acute hypoxemia with low oxygen levels that occurs in the absence of much or any breathlessness. This may slow diagnosis and therapy by medical staff (Rekalova, 2023).

Low oxygen levels (Hypoxia) Hypoxia is one of the indicators of severe COVID-19. The level of oxygen in the blood decreases when the alveoli are destroyed and fluid accumulates within them. Hypoxia may cause mild fatigue and severe organ dysfunction and may result in the so-called silent

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hypoxia which is when the levels of oxygen are low and shows minimal or no signs of breathlessness. This can be caused by a decrease in respiratory drive or the neurological effect of the virus and predisposes patients to acute deterioration. Chronic/extreme hypoxia also imposes pressure on the heart, brain, kidneys and other important organs increasing the risk of multi-organ failure. Therefore, pulse oximetry has become important for the detection and tracking of hypoxia in hospitals and at home during the pandemic (Razik et al., 2021).

The Pulmonary Fibrosis – Pulmonary fibrosis is another recognized and frequently emerging long-term sequela in patients who survive ICU-treated severe COVID-19 pneumonia or ARDS with the need to be placed on a ventilator. It manifests through the unpathologic regeneration of damaged lung tissue, substituting the alveoli of normal, flexible tissue with rigid, fibrotic scars. This scarring causes a decrease in lung elasticity and gas exchange as well as chronic symptoms, including exertional dyspnea, persistent chronic cough and reduced exercise tolerance. Patients with a history of severe hypoxemia, a lasting inflammatory response or mechanical ventilation are at risk. Even though partial recovery is possible, a small group of patients end up with permanent disability. The public health and clinical effects of post-COVID pulmonary fibrosis remain unexplored and antifibrotic treatment, rehabilitation and early treatment of long-term disability are actively being researched (Ali, 2022).

Organizing Pneumonia - Organizing pneumonia occurs after a person recovers from COVID-19 infection. This is observed during the subacute (intermediate between acute illness and complete recovery) or recovery stages. Abnormally thickening of the small airways and air sacs in the lower part of the lung occurs and takes the form of a scar. This prevents normal exchange of gases and may lead to cough, difficulty in breathing, low grade fever or feeling un-energetic even after you get out of the initial illness. It appears as patchy areas of density or consolidation of lung tissue on radiography. Lung function tests demonstrated a pattern of inability to move freely. In some cases, they improve by themselves or after taking steroid medication whereas in others they remain poor and recur. The most effective method of treating COVID-19-related organizing pneumonia remains unknown to doctors and they are currently trying to find the means of identify people at higher risk and help them (Alanazi et al., 2021).

Bronchial inflammation is also caused by SARS-CoV-2 which damages the bronchial tube lining, a major air passage transporting air from the throat to the lungs. The resulting injury is inflammation which may persist for weeks following acute infection. The symptoms include a dry stubborn cough, wheezing or a tight chest. The infection destroys the lining of the bronchi and results in an immune response that inflames the tubes and releases excess mucus, making them extremely receptive. Such bronchitis contrasts with regular bacterial bronchitis due to COVID-19 related inflammation that may take months to resolve which is one of the reasons why many individuals with COVID-19 experience cough, some call the so-called long COVID. A person of any age might have this kind of bronchitis and it is even possible to experience it in individuals with initially mild disease only. Existing research has examined the possibility of such chronic inflammation causing lasting airway hyperactivity or creating a pathway for asthma development among individuals with high susceptibility (Zhou, 2024).

Thromboembolism in Lungs - Thromboembolism in Lungs is one of the most dangerous complications associated with COVID-d-19 and it is characterized by the blockage of a circulating artery in the lungs by a blood clot. The combination of infection and the inflammatory response of the body develops a so-called hypercoagulable condition, where blood clots more easily. Endothelial damage, triggering of the coagulation cascade and abundant prothrombotic cytokines are involved. The disease can present acutely, typically accompanied by chest pain, dyspnea, tachycardia or collapse and is fatal unless promptly managed. PE is usually prevalent among diseases in hospitals, especially in an intensive care unit, although it also occurs in those who have been released or who were not seriously ill. Many high-risk patients are now advised to undergo preventive anticoagulation; however, doctors should weigh the threat of clotting against the threat of bleeding. Future research should determine the optimum duration and dose of prophylactic anticoagulation during both acute and chronic management of COVID-19 (Malas, 2020; Poor, 2021; Alexia, 2020).

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Long COVID-related breathlessness–Persistent breathlessness is one of the most frequently experienced and incapacitating long-term consequences of long COVID, that is, long-term effects that persist weeks or months after acute illness. This may occur even in individuals who do not require hospitalization or additional oxygen. The reason for this is complicated. Chronic lung inflammation, airway bronchoconstriction, partial deconditioning, microvascular alterations and autonomic issues may all contribute to this condition. Effective imaging and lung function usually indicate minimal harm; therefore, the experience of air deficiency is believed to be caused by other factors that are not apparent or quantifiable on examinations of the lungs. Out-of-breathness can strongly restrict day-to-day life, initiate anxiety or partake in depression. Scientists are experimenting with rehabilitation regimes, breathing and medicine to relieve symptoms but have not yet found an effective treatment (Xu & Shi, 2023).

Reduced diffusion capacity of the lung (impaired lung diffusion capacity) - The ability of the lung to absorb oxygen into the blood during breathing is known as diffusion capacity of the lung (also called DLCO - diffusing capacity of the lung). The reduced diffusion capacity of the lung is observed in a significantly high percentage of patients, even months after recovering from acute COVID-19. This implies that there is a reduced ability of oxygen to enter the blood from the alveoli. This may be due to chronic inflammation, destruction of the small blood vessels known as microvessels and premature laying down of connective tissue known as fibrosis of the alveolar-capillary membrane. These individuals do not often observe noticeable symptoms, although some report constant difficulties in breathing and exercising. The severity and duration of such impairment appear to be correlated with the severity of the initial illness; however, even those who had only mild COVID-19 may show unusual results. Researchers are also monitoring whether the reduced diffusion capacity improves with time or whether survivors of this virus can suffer lasting damage. It is not yet known what chronic health conditions this compromised lung diffusion may lead to. Further studies are required using a long-term approach (Qin, Wang & Wang, 2023).

Lung damage due to cytokine storm- A significant mechanism for causing severe and even fatal COVID-19 in patients is the so-called cytokine storm, an excessive immune response in which the body produces excessive amounts of inflammatory cytokines. Within the lungs, this storm leads to tissue destruction, blood vessel leakage and large-scale infiltration of immune cells. These changes soon develop into severe acute respiratory failure. Critically ill COVID-19 patients can develop acute deterioration of breathing, unsolvable hypoxemia and shock. Immunomodulating agents such as corticosteroids and cytokine inhibitors can aid those populations that respond, although there remains some dispute among physicians over who and when. Overloading immunosuppression may cause infections with other agents. There is still a problem of determining the optimal point between immune response suppression and secondary infection avoidance in COVID-19 treatment (Razik et al., 2021).

3. Cardiovascular System

Many patients admitted to hospitals with COVID-19 exhibit indications of myocardial injury, represented by high levels of cardiac troponin in the blood. Researchers have reported that approximately every third patient with hospitalization has this complication. Myocardial injury refers to injury to the heart muscle either immediately or gradually which may be due to direct viral attack, extensive system inflammation, absence of oxygen or dysfunction of the microvasculature. In patients with heart disease, the condition is associated with poor outcomes and increased mortality. Healthcare teams should closely monitor arrhythmias and heart failure when troponin levels increase in patients with COVID-19. It may be difficult to distinguish between real acute coronary syndromes and non-specific damage to the heart due to severe illness (Yancy, 2020; Lala, 2020; Yan, 2020).

Another interesting complication of COVID-19 is Myocarditis or inflammation of the heart muscle. Both pure viral infection of the heart and an exaggerated immune response are destructive. In severe cases, sudden cardiac death may occur, whereas mild cases may present with chest pain, palpitations or heart failure. Tests such as cardiac MRI and endomyocardial biopsy can also confirm

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inflammation but doctors do not frequently perform these tests during the acute illness period. The actual occurrence of myocarditis is not known because it can coincide with other heart conditions and because it is difficult to diagnose myocarditis within a short time. Inflammation can generate chronic cardiac dysfunction and necessitate chronic treatment and monitoring of heart failure or arrhythmia (Xu & Shi, 2023).

Cardiac Arrhythmias - COVID-19 has been connected to a lot of arrhythmias which are abnormality rhythm of the heart. These consist of tachyarrhythmia, including atrial fibrillation and bradyarrhythmia, including sinus arrest. In individuals with or without a history of heart disease, arrhythmias may occur due to direct heart involvement, systemic inflammation, hypoxia or electrolyte imbalances. Some of these arrhythmias are reversible, while others are dangerous and require urgent medical care. Severe arrhythmias may also occur even in patients with mild or no symptoms which is why cardiac rhythm should be monitored carefully during and after infection. The very long-term consequences of arrhythmias associated with COVID-19 are yet to be determined (Nguyen, Nedzlek, & Corcoran, 2022).

Acute Coronary syndrome (ACS) - COVID-19 has the potential to increase the likelihood of developing acute coronary syndrome (ACS) such as heart attacks. The coronary arteries are lined by unstable atherosclerotic plaques which are prone to rupture leading to the formation of clots because of the virus and the subsequent inflammatory process that follows. It is also possible that the hypercoagulable condition associated with COVID-19 encourages clotting of the coronary vessels. ACS has symptoms such as chest pain, difficulty in breathing, alterations in the EKG and requires immediate treatment. Therapeutic approaches for treating ACS in COVID-19 patients are challenging because their symptoms are the same as those of a viral infection and special precautions against infection are required in the cardiac treatment unit. It is crucial to detect and treat the condition in time as lower levels of delay increase the possibility of complications (Razik et al., 2021).

Heart Failure: Heart failure is an existing risk as well as a developed risk factor for COVID-19. In COVID-19, heart failure may begin when the heart muscle is damaged directly, when the heart muscle becomes inflamed (myocarditis), when the body does not receive oxygen (hypoxia) and when overwhelming inflammation suppresses the heart. All these problems can increase the load on the heart, decrease heart performance, cause fluid accumulation and decrease blood flow to major organs. Others experiencing stable heart failure prior to COVID-19 may deteriorate (worsen) in the course of or following infection and others who had not previously experienced heart failure may develop it. This complication is associated with high rates of intensive care admission and mortality. Medical workers must achieve a balance in fluids, oxygen, COVID-19 and heart failure treatments (Rekalova, 2023).

Thromboembolism: COVID-19 induces vulnerability to thromboembolism in veins and arteries. Due to the activation of clotting pathways and the creation of robust inflammation by the virus, the infection produces a hypercoagulable condition (one that is likely to form clots). This causes blood clots in the coronary arteries and may result in a heart attack, as well as clotting of the veins which may result in deep vein thrombosis (DVT) and pulmonary embolism (PE). Depending on the location of the clot, patients can experience chest pain, limb swelling and unexpected breathlessness. Anticoagulation has become common practice in hospitals where thromboembolism has afflicted a large number of people with severe COVID-19. Nevertheless, the efficacy of prophylactic anticoagulation in non-hospitalized or mild cases of infection and the optimal duration of therapy remain unknown (Razik et al., 2021).

Worsening of pre-existing hypertension and the development of new-onset hypertension may occur through the destruction of the Renin-Angiotensin-Aldosterone System (RAAS), in which hypertension is regulated and vascular tone and fluid equilibrium are attained. Through its ACE2 receptors, the virus binds to the RAAS and increases the level of Angiotensin II. Increased angiotensin II causes blood vessels to narrow, resulting in increased blood pressure. Hypertensive patients are more likely to develop serious consequences of COVID-19 infection and are not always able to normalize their blood pressure during the course of an illness and after its occurrence. This has huge

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implications as uncontrolled hypertension increases the chances of developing cardiovascular events and organ injuries when faced with COVID-19. Further studies are required to determine the long-term consequences of COVID-19 on blood pressure and optimal treatment practices (Razik et al., 2021).

Endothelial Dysfunction: COVID-19 damages the inner lining of blood vessels, also called the vascular endothelium. The virus may infect them directly or cause inflammation that destroys them. This harm results in endothelial dysfunction, impaired dilation, elevated inflammation and a prothrombotic (clot-prone) environment. This condition increases the risk of thrombotic events and multi-organ injury. It can also attribute COVID-19 to cardiovascular,renal and neurological complications. Based on this perception, anti-inflammatory and anticoagulation-focused treatments have emerged (Rekalova, 2023).

Stress Cardiomyopathy (Takotsubo Syndrome): Stress cardiomyopathy or takotsubo syndrome has also been reported in patients with COVID-19. This condition leads to weakening of the left ventricle of the heart which balloons up temporarily and is normally associated with physical or emotional stress. The patients typically experience chest ache with alterations in EGs similar to those experienced in heart attack with the absence of blocked coronary arteries. There is no precise reason known but it is believed to be due to high levels of stress hormones and extreme activity of the sympathetic nervous system leading to stress due to the illness or pandemic. The recovery of heart function in the majority of patients occurs within weeks and in rare cases, acute heart failure or arrhythmias can occur. Those with other health issues are more prone to be attacked (Zhou, 2024).

Long COVID Heart Symptoms- Other individuals who develop COVID-19 experience heart issues even after many months. This condition is known as long COVID. These issues entail pain in the chest, rapid heartbeats and weakness or fatigue upon exercising. Non-serious heart damage may not be detected during tests but the symptoms are factual. Potential triggers include autonomic nervous system impairment, persistent low-grade inflammation, vascular changes in small blood vessels and the effects of previous myocarditis or cardiac damage. COVID-19 survivors should have regular cardiac checkups because these complications are associated with the potential deterioration of the quality of life and the ability to perform basic tasks. Scientists are attempting to discover more effective treatment methods (Xu & Shi, 2023).

4. Nervous System

Delirium is characterized by abrupt disorientation and reduced awareness which alternately appear and disappear. It is commonly found in older adults and in patients who are infected with COVID-19 and are hospitalized, especially in intensive care. The pathogen can enter the brain and cause a powerful systemic response. It may reduce oxygen, imbalance blood chemicals and result in extremely high levels of inflammation which contributes to the risk of delirium. Delirium is associated with prolonged hospital stays, increased complications and mortality. In other instances, delirium persists until patients go home, thus emphasizing the importance of early diagnosis and effective treatment (Liao, 2021).

Brain Fog - Patients often suffer from brain fog months after COVID-19. Brain fog implies an inability to concentrate, memory loss and slow thinking. It can occur even in individuals with mild breathing difficulties. Scientists are not certain as to the reason it occurs; however, it may be a condition of prolonged inflammation in the brain, poor blood-brain barrier, damaged tiny blood vessels or harm due to the immune system. Brain fog makes work, school and daily life very difficult. Treatments are supportive, as there is still no particular medicine to address this post-COVID issue (Nemati, 2021).

Stroke - COVID-19 increases the chance of an ischemic stroke, particularly in people who are seriously sick or have heart disease or blood vessel conditions. The reasons for this include excessive inflammation, a prothrombotic state, impaired blood vessels and direct action of the virus on vessel cells in the brain. Stroke can occur among younger adults and can be more aggressive than normal strokes. Signs such as sudden weakness, speech difficulties, loss of sight and loss of consciousness

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can be observed. Prompt diagnosis and treatment are critical; however, it is difficult to manage stroke patients in COVID-19 units due to infection precautions and the like. To date, no studies have been conducted on long-term outcomes (Hess, 2020).

Anosmia (loss of smell) - Initially, one of the COVID-19 indicators was sudden loss of smell (anosmia). It mostly precedes a cough or fever. This could be a result of inflammation or direct damage to the nerve and support cells in the nose by the virus. Unlike the loss of smell due to other upper-respiratory infections that tend to have a backup congestion of the nose, COVID-19 anosmia lacks this backup congestion. A large number of individuals recover their sense of smell within a short duration, whereas others fail to do so, making the consumption of food and nutrition desirable. Early identification of anosmia can help isolate and curb its spread (Gendrin, 2020).

Ageusia (taste loss) - COVID-19 frequently accompanies the loss of taste (ageusia) with loss of smell. This is because a person may lose taste receptors on the tongue or taste sensory transmission nerves. Ageusia may be seen in the initial stages of the infection and may extend for several weeks. The vast majority of patients recover but some describe prolonged or changed taste that may decrease appetite and lead to weight loss and unpleasant meals. Taste loss persisting with anosmia is also considered a primary indicator of COVID-19 among other respiratory diseases (Salzano, 2020).

Brain inflammation Encephalitis: Encephalitis is inflammation of the brain. It is a severe but uncommon malady that may develop as a COVID-19 complication. Symptoms include headache, fever, confusion, seizures, altered consciousness and severe cases leading to coma. The virus may have directly attacked the brain; however, it is also possible that an immune and general inflammatory process was involved. The virus in the brain can only be detected via its tissue samples but it is difficult to find which forces doctors to depend on brain scans and cerebrospinal fluid to determine its presence. COVID-19-related encephalitis can make a person seriously ill and sometimes even lethal. Survivors are susceptible to sustained issues, including cognitive and engine weaknesses; hence, early identification and management of care are important (Kamal, 2022).

Peripheral Neuropathy- Peripheral neuropathy is another complication reported in COVID-19. It manifests itself in the form of pain, tingling, numbness and weakness of limbs. It occurs as a result of damaged or malfunctioning peripheral nerves. The etiology is likely to be direct viral, immune overreaction or that caused by long-term immobility, malnutrition or serious disease. The symptoms may be as simple as a change in sensation or as severe as weakness or neuropathic pain. Cases resolve over time with the help of rehabilitation but some continue to have long-lived deficits that influence movement and quality of life. The pandemic has prompted scientists to continue learning more about the causes, risk factors and optimal treatment of COVID-related peripheral neuropathy (Córdova-Martínez, 2022).

Guillain-Barre Syndrome (GBS) - Another known autoimmune disease associated with COVID-19 is Guillain-Barre Syndrome (GBS). It leads to acute muscle weakness which begins in the lower extremities and then involves the upper extremities, face and lung muscles. In severe cases, patients require mechanical ventilation. This condition occurs when a viral infection is followed by an attack of the immune system on the myelin sheath of the peripheral nerves. Cases of COVID-19-related GBS have been reported worldwide and may occur during acute disease or in the weeks following acute disease. The majority of patients do recover, albeit weeks or months later and with some deficits. Early treatment with intravenous immunoglobulin or plasmapheresis may improve the condition (Foschi, 2020).

Neuropsychiatric Symptoms - Issues linked to neuropsychiatric disorders of anxiety, depression and post-traumatic stress disorder (PTSD) have soared due to the pandemic. The causes of tide may be inflammation, brain chemical alteration or even stress due to sickness or the overall consequences of the pandemic. Recovering COVID-19 patients and people who have been affected by the pandemic socially and economically are both going through mental struggles. The symptoms may be either short-term or chronic and interfere with sleep, mood and functioning. Mental health is a critical component of the COVID-19 recovery process and requires both care and psychological assistance (Hafeez,2021).

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Long-term Cognitive Dysfunction - The emergence of persistent cognitive (cognitive) problems has contributed to the phenomenon of long COVID. Constant issues with attention, memory, executive role and processing speed may also plague individuals whose original illness was not severe. Daily activities become more difficult and recovery from work is slowed down because of such deficits. The mechanisms are probably multifactorial, including inflammation, microvascular damage, consequences of critical illness and perhaps direct viral injury. The most up-to-date support involves cognitive rehabilitation, exercise and other health condition management; however, an actual treatment does not exist. Additional research should be conducted to determine the causes of these risks, outcome prediction and effective treatment (Alwan, 2023).

Acute Kidney Injury (AKI) - Acute kidney injury (AKI) is a frequent, bad outcome in inpatient covid-19 patients, particularly severe cases. AKI occurs when the kidneys lose their function abruptly, resulting in an increase in the amount of waste products in the bloodstream, impairing the fluid and electrolyte balance of the body and, in some cases, multi-organ failure. AKI caused by COVID-19 may occur in a variety of ways: direct attack of the virus on the cells of the kidney, profound systemic inflammation (cytokine storm), hypoxia due to respiratory difficulty and secondary manifestations such as sepsis or shock. Patients with a history of chronic kidney disease, diabetes, hypertension or mechanical ventilation are at a greater risk of developing AKI. A shorter AKI has been associated with longer hospital stays, increased risk of requiring dialysis and increased mortality (Razik et al., 2021).

Direct Viral Infection of Kidney Cells- There increasing evidence that SARS-CoV-2 can directly infect kidney cells. Coronavirus infects tubular epithelial cells as it infiltrates kidney tissue through ACE2 receptors. This direct infection can kill and harm cells, leading to short-term or even permanent issues. This has been substantiated by recent reports showing the presence of viral particles or RNA within kidney biopsy and autopsy specimens. This infection impairs regular filtration and reabsorption, initiates local inflammatory processes and may induce proteinuria or AKI. Its long-term effects remain to be studied; however, it indicates that the kidney is one of the direct targets of COVID-19 (Xu & Shi, 2023).

Proteinuria (Protein in Urine) - One frequent laboratory finding in COVID-19 is proteinuria (presence of unnatural levels of protein in urine). Proteinuria commonly occurs early in the course of the illness and appears to be more prominent as the disease progresses. This signifies that there has been damage to the glomerular filtration barrier or renal tubules, thus permitting the leakage of proteins from the blood into the urine. Proteinuria may be short-lived and indicative of acute damage or persistent, indicating potentially severe or chronic destruction. Proteinuria can present with AKI but also without kidney failure. Increasing levels of proteinuria are associated with adverse outcomes and may indicate that a person is at high risk of future kidney issues (Razik et al., 2021).

Hematuria (Blood in Urine) - Hematuria, urine with blood, has been described in a small number of COVID-19 patients and may indicate either direct renal damage to the urine-producing structures of the kidney (glomeruli) or to the urinary tract. It may manifest as AKI, proteinuria or alone or it may be invisible with no visible blood. However, it may be macroscopic, indicating severe hemorrhage. Although it may be temporary, it is indicative of serious inflammation or even glomerulonephritis. Hematuria is an indicator of renal involvement in COVID-19 and is possibly associated with severe disease outcomes and prognosis (Song, 2020; Zeng, 2020).

Thrombotic Microangiopathy (TMA)-COVID-19 increases the risk of blood clots in the body and occasionally, these may develop in the small vessels of the kidneys. TMA is caused by thrombi blocking the kidney microvasculature, causing tissue ischemia, damage and loss of function. It is the outcome of endothelial damage, activation of clotting mechanisms and broad inflammation. TMA may result in proteinuria, hematuria, AKI and it may result in acute or chronic kidney impairment. Understanding TMA has led to interest in anticoagulation therapy and monitoring of clotting and kidney markers in COVID-19 patients (Agarwal, 2021).

Cytokine-mediated Inflammation - Severe COVID-19 in some people results in an altogether overactive immune reaction to the virus, producing excessive amounts of pro-inflammatory

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cytokines into the blood (a cytokine storm). This unregulated process impairs kidney tissues and results in swelling, declining filtration and deteriorating kidney function. Both acute and long-term kidney injury and damage can be explained by the involvement of cytokine-mediated inflammation. The management of the cytokine storm has become an important component of the treatment of severe COVID-19, since inflammation can be decreased by using medications that reduce kidney (and multi-organ) damage (Pai, 2020).

Due to COVID-19, richness of the body (volume depletion) may occur because of fever, vomiting or diarrhea. Assuming that this deficit is not compensated, blood pressure will fall (hypotension) which reduces the blood supply to the kidneys. The kidneys receive less blood and thus do not eliminate waste effectively and may fail to maintain the proper fluid and electrolyte balance, increasing the chances of acute kidney injury. This occurs irrespective of direct virus-related damage to the kidneys and reveals why the fluid balance and blood pressure of patients with COVID-19, in particular, older patients with pre-existing kidney failure, require close monitoring (Xu & Shi, 2023).

CKD insult Continued deterioration - Individuals with a history of chronic kidney disease (CKD) have a high possibility of infection COVID-19 causing exacerbation of the disease and even the development of end-stage renal disease. Weak kidney system At the best of times, kidney systems are easily overwhelmed in response to a viral attack on the kidneys and systemic inflammatory response of the body in addition to any acute hemodynamic issues. Many patients with CKD who contract COVID-19 are hospitalized and some require acute dialysis. Another reason why COVID-19 increases CKD risk is the increased possibility of secondary complications following the disease, including infections and cardiovascular issues which further aggravate the situation (Mustafa, 2022).

Kidney Fibrosis - The fibrosis of the kidneys may occur as long-lasting inflammation with COVID-19, in particular, the severe or prolonged form of the illness. Healthy kidney tissue is changed to scar tissue which complicates the filtering of blood and balancing of fluids. The risk is increased in individuals who experience acute kidney injury, persistent inflammation or long hospital stays. Some studies on pulmonary fibrosis indicate that it can be prevented with early intervention thereby preventing fibrosis altogether. Currently, research is being conducted to determine whether it is the same as the kidneys (Qin, Wang & Wang, 2023).

High risk of death in kidney patients - Kidney problems put a person at a very high risk of dying because of COVID-19, particularly in individuals who are also undergoing dialysis. Patients with kidney disease have a greater risk of developing serious diseases because their immune system is less effective, they tend to have additional diseases such as diabetes and hypertension and they have an increased risk of fluid overload, electrolyte appetite and infection. Research has demonstrated that acute or chronic kidney disease among hospitalized COVID-19 patients leads to significantly higher mortality rates than in kidney-unimpaired patients and maintenance and intensive treatment of this population group are required (Dong, 2020).

5. Liver

Hepatic Dysfunction-Liver Enzyme Elevation - High levels of liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are seen in most humans infected with COVID-19. These enzymes increase with liver cell damage. This liver difficulty may be temporary and mild and may even be more pronounced in people with severe or critical illness. These may be due to direct viral hepatocyte effects, immune-mediated hepatocyte inflammation or the general effects of systemic disease. Major elevations should be associated with closer attention as they may indicate more significant involvement of the liver, although most of them are temporary. This has shown that hepatic dysfunction in COVID-19 is associated with poor outcomes in hospitalized patients, focusing on local people (Zhang, 2020; Bodewes, 2020; Francis, 2020).

Further Damage of Pre-existing Liver Disease - Severe COVID-19 is more likely to occur in the presence of pre-existing liver diseases, including hepatitis B/C infection, fatty liver disease or cirrhosis. The virus can rapidly develop liver decompensation, with jaundice, coagulopathy, encephalopathy or ascites. Existing liver disease enables COVID-19 to lead to further cases of viral

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hepatitis, acute-on-chronic liver failure and a poorer overall prognosis. Advanced liver disease also causes immunosuppression which increases the risk of infection and a lack of liver reserve increases the danger of any additional stress on the body upon the organ. Proper results can be achieved by timely care and early diagnosis of high-risk patients (Kameswari, Ezhilarasan, & Lakshmi, 2020).

Cholestasis (Obstruction of the Bile) - COVID-19 may also lead to cholestasis, a condition in which bile fails to be transported to the duodenum. This usually causes jaundice, dark urine, itching (pruritus) and increased levels of bilirubin and alkaline phosphatase. Cholestasis can be secondary to direct viral infection of the bile duct epithelium or immune- or drug-related liver injury. It is prevalent in severely ill patients, particularly those experiencing multi-organ-down syndrome. Cholestasis that is prolonged or severe may delay the recovery process and lead to secondary infections or liver damage. (Kameswari, Ezhilarasan, & Lakshmi, 2020)

Direct Viral Injury to Hepatocytes- ACE2 is highly expressed in liver cells and bile duct epithelial cells which allows SARS-CoV-2 to directly enter the liver cells and bile duct epithelial cells through the ACE2 receptors. Cytopathic changes, hepatocyte apoptosis, necrosis and inflammation may result from viral infection. This direct injury, occurring together with the systemic inflammatory response of the body, can describe the entire spectrum of liver disease in COVID-19, from mild elevation of enzymes to rare cases of fulminant hepatitis. This is supported by post-mortem investigations that have shown the presence of viral RNA or proteins in the liver. To implement specific treatment methods, it is necessary to understand the role of direct viral infection and secondary damage (Xu & Shi, 2023).

COVID-19 can harm the liver. Hypoxic hepatitis (shock liver) is a type of liver damage. In very severe instances, such as lung failure and poor oxygen levels, liver cells lack sufficient oxygen. This results in a severe increase in liver enzymes even up to acute liver failure. It has a high mortality rate and most cases occur in people who are undergoing systemic shock, sepsis or multi-organ dysfunction. Physicians can assist in raising oxygenation levels and stabilizing blood pressure (Zhou, 2020).

Drug-induced liver injury (DILI) is another concern. Consecutively consumed medicines that are likely to hurt the liver are numerous in COVID-19 sick patients. Several medications can result in liver problems, including antivirals, antibiotics and corticosteroids, particularly when they are taken in excess or when they are combined. Symptoms include hepatitis, severe liver enzyme elevation, jaundice and acute liver failure. Individuals with existing liver issues or those who are on multiple medications when visiting a hospital are at a higher risk. One should pay attention to close observation of liver tests and doctors need to distinguish DILI and SARS-CoV-2 or other liver damage (Borobia, 2021; Lou, 2023).

Severe COVID-19 can also cause a cytokine storm. An onslaught of immune messages, called cytokines, triggers inflammation in most organs. The liver may be attacked even in the absence of a direct viral attack. Liver enzymes, coagulopathy and, in some cases acute liver failure were indicated in blood tests. The extent of liver injury tends to be equal to the extent of systemic inflammation and respiratory failure. Immunomodulator drugs or corticosteroids relax the immune system and restrict liver damage to ameliorate the outcomes (Velikova, 2021; Mitrev, 2020).

Non-alcoholic fatty liver disease (NAFLD) predisposes individuals to severe COVID-19 and liver complications. NAFLD is characterized by an overabundance of liver fat, prolonged swelling and immune issues. Such factors can predispose the body to an increased response to SARS-CoV-2 infection. Patients with NAFLD are prone to acute liver damage, hospital admission and progression to progressive disease or acute-on-chronic liver failure. Metabolic problems and long-term liver health are also aggravated by COVID-19. Tight management and monitoring of metabolic risk factors are advised (Yoo, 2021; Singh, 2021; Hariyanto, 2023).

The portal vein can also be clotted with COVID-19. It is the major blood vessel that receives liver blood flow from the abdominal organs. Portal vein thrombosis (PVT) occurs when a clot appears in the portal vein. Such an obstruction may lead to portal hypertension, bowel injury or hepatic failure. Symptoms include abdominal pain, liver enlargement, ascites and gastrointestinal bleeding. Risk

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factors include profound illness, extent of bed rest and other pre-existing liver ailments. It is difficult to diagnose due to the similarity of symptoms to the complications of other COVID-19 cases and life-saving intervention is the early usage of anticoagulants (Luciano, 2021).

Some patients experience long-term liver complications even after survivorship. It has been observed that high liver enzyme levels remained persistent and severe and inflammation and fibrosis or steatosis occurred. These phenomena can be due to residual viral activity, lasting immune imbalances, toxicity to medicines or unknown liver maladies. Frequent follow-up and liver testing deserve attention, especially in patients with significant liver involvement when ill, particularly in cases of pre-existing liver disease. They have an increased risk of developing chronic liver disease (Mohammed, 2024; Stasi, 2025).

6. Gastrointestinal System

Diarrhea is considered one of the most widespread gastrointestinal symptoms in COVID-19 patients. It occurs when the coronavirus damages the enterocytes which are the type of cells that line the interior intestines and have ACE2 receptors and inflammation of the intestines. At any point during the illness, diarrhea may occur, sometimes even prior to respiratory symptoms or as an individual symptom. It tends to last only a short time, although it may be serious and lead to dehydration, electrolyte imbalances and malnutrition, particularly among elderly individuals or those with chronic diseases. The detection of GI symptoms such as diarrhea allows doctors to diagnose the disease at an early stage and limit infection, as viral RNA can be detected in the stool and has the potential to spread via the fecal-oral route (Baumgart, 2020; Zheng, 2020; Turova, 2021).

Nausea and vomiting are common GI symptoms of COVID 19, particularly in the early stages of the disease. They can be triggered by direct viral infection of the gastrointestinal tract, systemic inflammation or drug side effects. Although generally mild, chronic or serious nausea and vomiting may cause dehydration, restriction of oral intake and make treatment difficult, particularly in older adults or patients with GI disorders. Preventing complications and helping the recovery, it is essential to identify and provide, in the first place, supportive care, including fluid and electrolyte replacement (Andrews, 2020; Kumar, 2020; Tilg, 2020).

Abdominal Pain: a small proportion of patients with COVID-19 complain of abdominal pain and cramping which may vary from mild tenderness to excruciating pain, possibly similar to other acute conditions of the abdomen. The cause of pain can be localized inflammation of the intestines, invasion of the GI mucosa by a virus or another complication (mesenteric ischemia: reduced blood flow to the intestines). Pain may be one of the initial symptoms in severe cases, often causing a delay in diagnosis in individuals who do not have respiratory symptoms or those with mild symptoms. Abdominal pain should be considered a symptom of COVID-19 because it must be evaluated immediately to distinguish it from conditions such as appendicitis and gallbladder disease (Lovis, 2022; Hayashi, 2021).

Loss of Appetite (anorexia) Loss of Appetite or anorexia is prominent in both mild and severe cases of COVID-19. It has systemic inflammation, direct GI involvement and general body effects of viral illness as its driving factors. Anorexia can be among the first symptoms that result in a decrease of food consumption, weight loss without intentions and poor recuperation in both hospitalized and elderly individuals. Never-ending anorexia will lead the patient to malnutrition and muscle wasting which will exacerbate his/her condition. Individuals at higher risk may require supportive nutritional care and appetite stimulants (Martin, 2020; Nacinovich, 2022).

Gastrointestinal bleeding is less frequent but a grave issue in COVID-19, particularly in individuals who are in a critical condition or are undergoing the treatment of blood clots through the application of anticoagulant medicine. Bleeding may manifest as blood in the stool or vomit and may occur secondary to pre-existing ulcers, new erosions, stress-induced mucosal disease or direct damage to the mucosa by viruses. This is more dangerous for patients with underlying GI pathology or coagulopathy. GI bleeding during COVID-19 could be complicated by infection control issues and efforts to avoid blood clots due to anticoagulants, with the associated risk of excessive bleeding. Early

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detection, nurturing support and endoscopic assessment (in non-harmful conditions) are the main determinants of better results (Aneis, 2024; Mohamed, 2021).

Disruption of the liverGut axis - COVID-19 may damage the liverGut axis which is the body process that links the bacteria in the gut and the liver. The virus alters the composition of the intestinal microbiota, damages the intestinal lining and increases intestinal permeability. The toxins released by the bacteria along with the inflammatory messengers then find their way into the bloodstream and are directed to the liver where they cause systemic and hepatic inflammation. The result of this damage to the gut is the exacerbation of the existing liver disease and more serious symptoms in the stomach and bowel. The liver-gut axis is among the focuses of research in severe COVID-19 due to the possible spread of these effects to other organs (Chen, 2021; Gasbarrini, 2020).

Intestinal Ischemia - Another infrequent though severe intestinal perception of COVID-19 is intestinal ischemia. It occurs when there is a decrease in blood supply to the intestines usually due to the formation of blood clots in the mesenteric vessels that occur as a result of the hypercoagulable state caused by the virus. Intestinal ischemia causes people to experience severe abdominal pain, vomiting and, in a more developed form, dislodging some part of the intestines which in some cases requires emergency surgery. Late identification exposes patients to the possible danger of multi-organ failure and death. Timely and prompt treatment is crucial, particularly for critically ill or high-risk patients who exhibit abrupt stomach discomfort (Gosta, 2022; Williams, 2023).

Disturbed Gut Microbiota - COVID-19 has the ability to alter the composition of the intestinal microorganisms. The number of beneficial bacteria decreased, whereas the number of harmful microbes increased. Scientists have attributed this change to increased inflammation of the system and reduced immune system responses. A bad gut can increase the severity of the effects of COVID-19, duration of the illness and its consequences. Research suggests that positive alterations in the balance of gut microbes, achieved either with probiotics or dietary modification could contribute to recovery and reduce long-term complications; however, additional clinical trials are required (Batista, 2021).

Long persistence of virus SARS- CoV 2 fecal shedding SARS-CoV 2 RNA in stool samples has been identified weeks after the respiratory symptoms dissipate indicating the persistence of the virus in the intestine. Increased duration of viral shedding becomes a concern for absorbing fecal-oral transmission of the virus in congested or hygienically poor conditions. This also implies that the GI tract may serve as a reservoir for reinfection or persistence or symptoms in some individuals. Care centers should monitor this route and increase efforts for infection control (Xu & Shi, 2023).

Post-COVID Functional GI Disorders - There is another post-COVID effect after acute covid-19 modalities, where some people develop a long-term impairment of GI which is like irritable bowel syndrome (IBS) - abdominal pain, irregular bowel movements, bloating and discomfort. Such symptoms could take months to develop and are caused by the presence of persistent inflammation, altered gut microbiome, immune disturbances and stress associated with the disease. The intervention is aimed at alleviating symptoms, dietary adjustments and transferring patients to the gastroenterology service if necessary (Santoso, 2024).

7. Immune and Endocrine Systems

Cytokine storm - Among the most dangerous complications of severe COVID-19 is a cytokine storm. It occurs when the immune system is overactive, releasing excessive pro-inflammatory cytokines into the blood. This hyperactivity results in an extensive inflammatory process, leakiness of small blood vessels and rapid destruction of multiple organ systems, including the lungs, heart, liver and kidneys. Acute respiratory distress syndrome (ARDS), shock and multi-organ failure among critically ill patients are motivated by storms. Supportive treatments and, in some restricted situations, immunomodulatory treatments (corticosteroids or cytokine traps) are used to manage the reaction. Timely diagnosis and therapy play an important role in enhancing the survival of patients who have experienced a cytokine storm (Machado, 2021).

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Lymphopenia (deficient lymphocytes) - Moderate/severe COVID-19 is characterized by lymphopenia (or significant reduction in the number of lymphocytes (in particular T cells and B cells). The direct destruction of lymphocytes cannot be ruled out as a result of SARS-CoV-2 or that it alters the outcome of bone marrow activity by provoking massive systemic inflammation. Lymphopenia is associated with more adverse courses of disease and the inability to clear the virus combined with an increased risk of secondary infections. A persistent deficit may also impair the ability to develop a robust antibody response and may determine the natural immune status and efficacy of vaccines in convalescent patients (Xu & Shi, 2023).

Hyperinflammation Lengthy, severe immune stimulation is an immune reaction in a small group of COVID-19 patients which results in the extreme production of cytokines and other inflammatory signals. This condition can directly produce tissue-damaging effects and increase the possibility of developing ARDS, myocarditis (heart inflammation), acute kidney failure and neurological complications. Hyperinflammation is as severe as the worst clinical outcomes and the biomarkers are C-reactive protein, ferritin and interleukin-6 which tend to be very high. Treatment tends to include immunosuppressive and regulatory treatment in selected individuals and the challenge regarding attacking inflammation is that it is related to reducing the immune response (Sitorus, 2020).

Camphor Activation Syndrome (MAS) - The Macrophage Activation Syndrome (MAS) is an uncommon and severe hyperinflammatory disorder exhibited in a few COVID-19 infected people. It is also characterized by unregulated hyperactivation and hyperproliferation of macrophages which are a type of immune cell, causing dysfunction of multiple organs, a cytokine storm, coagulation abnormalities and cytopenia. Clinically, MAS may be difficult to differentiate from other causes of systemic inflammation; however, it characteristically demands an early and vigorous initiation of high-dose corticosteroids and immunosuppressive or biologic cytokine-targeting therapy. Unless timely intervention is established, the risk of mortality is high (Ombrello, 2021).

Immune Exhaustion: The failure of immune cells to serve their purpose, particularly T lymphocytes, caused by long-term or severe infection. Continuous viral replication and fortifies the immune fortifies immune activation in COVID-19 cause immune cells to enter an unwanted functional null or exhausted state, damaging the immune system's ability to clear the virus, resulting in persistent infection or slowed recovery. Immune exhaustion is also prevalent in COVID-19 patients and includes immune exhaustion indicators including augmented expression of inhibitory receptors in patients with severe disease. The duration of immune memory and the risk of reinfection or inadequate response to a vaccine may also be associated with immune deficiency (Xu & Shi, 2023).

Autoimmune reactions may occur due to COVID-19. It may initiate new diseases or aggravate pre-existing diseases. Examples include Guillain-Barré syndrome (GBS), a rapidly progressive autoimmune neuropathy that causes sudden onset of weakness and paralysis, systemic lupus erythematosus (SLE) and other rheumatic diseases. Scientists believe that the issue is due to molecular mimicry, when the proteins in the virus resemble those within the body and the overreactive immune system can attack the tissues in the body. These issues may emerge during acute infection or weeks later. The best outcomes are usually achieved with rapid and cautious immunomodulatory treatment (Zhou, 2024).

Delayed antibody production occurs in some COVID-19 patients, particularly those with cancer organ transplants or those receiving certain medications. This slow (weak) humoral immunity may allow the virus to persist, manifesting longer or more severe symptoms and increasing the chances of reinfection. Vaccine effectiveness can also be undermined by slower antibody reactions. An early indicator of delay will allow doctors to plan additional protection and post-exposure measures for high-risk patients (Jain, 2021).

Natural Killer (NK) cells are cells that are prospectively used to contrast the onslaught of the virus in the body as the initial line of defense, functioning in the innate immune system. Severe Acute Respiratory Syndrome Coronavirus 2 (COVID-19) disease is associated with low cell counts and inactivation of NK cells. This creates a disadvantage because it makes it difficult to maintain low

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levels of viral load; thus, the viral load becomes high and the disease becomes severe. Activation of NK cells is now considered a potential biomarker and intervention point; however, it is not easy to revert to normal NK activity in severe COVID-19 (Razik et al., 2021).

Immunological dysregulation is observed in many patients, long after the initial infection, particularly in patients with long COVID. They experience persistent low-level inflammation, perturbed cytokine profiles, a shift in T or B cell effectivity and the presence of autoantibodies. Fatigue, muscle pain and brain fog are long-term symptoms generated by these changes. The duration of these immune issues has not yet been discovered and it is also unclear whether they can completely recover; however, their awareness signifies future observation and research of effective long-lasting COVID implications and treatment (Aguilar, 2024).

Post-infectious complications of COVID-19 such as Multisystem Inflammatory Syndrome in Children (MIS-C) and adults (MIS-A) are severe yet uncommon. They emerge weeks following COVID-19 infection and may resemble either Kawasaki disease or toxic shock syndrome. They are characterized by fever, inflammation and difficulty in various organ systems (heart, gastrointestinal tract, kidneys and skin). It must be carefully monitored and diagnosed early and treated with immunomodulatory agents, such as intravenous immunoglobulin and corticosteroids, to avoid organ effects and enhance survival. Lab work is ongoing to ameliorate the causes, risk factors and most optimal treatment of this problem (Xu & Shi, 2023).

8. Mental Health

Due to the COVID-19 pandemic, anxiety disorders have increased significantly worldwide. Fear of infection, uncertainty about the future, impairment of everyday life and prolonged social isolation have worsened the anxiety of many people. This increase cut across all age groups but was particularly powerful among individuals with existing mental health issues, first-line healthcare workers and low-income households. Anxiety was frequently expressed through constant worrying, panic and preoccupation with family and health issues. Since the pandemic is recurring, evolving and fluctuating the restrictions and guidance provided by governments regarding public health measures have made the symptoms of anxiety difficult to manage and, in some cases, have exacerbated them (Mulling, 2022).

Depression also increased significantly during the pandemic. Social distancing, lack of support, grief, loss of employment and health concerns were some of the driving forces. Others reported persistent morbidity, despair, loss of interest in everyday events and alterations in eating or sleeping. Isolation combined with the loss of routine, in many cases, exacerbated pre-existing depression or caused new depression, even in those who had never had a mental health issue prior. Worldwide levels of depression increased significantly, demonstrating the necessity of simplified mental health care access and enhanced social support in the present and post-pandemic contexts (Chankurtaran, 2021).

Post-traumatic stress disorder (PTSD) was observed more frequently among COVID-19 survivors, medical professionals and individuals who suffered huge losses. They were characterized by intrusive memory, flashbacks, feelings of numbness, avoidance and increased feelings of anxiety or alertness. Being confined in a hospital because of a terrible disease, exposure to death and the observation or loss of close friends and family members puts tremendous emotional stress on patients. Moral injury and burnout occurred in medical personnel due to the high death rate of patients, critical condition of supplies and severe working environments. Chronic PTSD may have detrimental implications on everyday performance and quality of life; thus, PTSD patients in these communities require trauma-informed care and perpetual mental wellness counselling (Hu, J., 2022).

More people also dealt with sleep issues and insomnia became more common during the pandemic. Among these were stress and anxiety about health or finances, immediate consequences of illness or recovery and drastic changes in routines and sleep schedules. Sleep is also adversely affected by increased social isolation, more time spent on screens and less physical activity. Sleep problems were mentioned by both the general population and survivors of COVID-19 and for some

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individuals, this issue persisted for months after the acute illness (Kalra et al., 2020). Sleep aggravates anxiety and depression symptoms, as well as overall health and people remain stuck in a vicious cycle that requires specific assistance.

Even people with mild COVID-19 and many of its survivors insist on telling how they continue to experience issues with their thinking which are commonly referred to as brain fog. They reported the inability to concentrate, memory loss, reduced clarity of thoughts and an impression that their thoughts were more sluggish. Brain fog may take weeks or even months after the acute period of illness and is a principal post-acute symptom of long COVID. The exact reasons remain to be investigated by scientists; however, they believe that it can be associated with persistent inflammation, brain injury with injuries to the small blood vessels in the brain or the effects of the virus directly on the nervous system. These issues may cause complications in daily performance, reduce work productivity and diminish quality of life which is why it is valuable to recognize and support survivors (Tsai, 2024).

The pandemic and associated restriction measures (lockdown, social distancing, restrictions on gatherings) made most people lonely and many withdrew into their social lives. Older adults, other individuals living alone and individuals with few social networks were the most affected. Reduced interactions with other people raise the chances of developing depression, anxiety and minor thinking and memory decline. Staying at home alone for a long time and making reconnections difficult after lifting the restrictions were also challenges (Gariepy, 2023).

During the pandemic, more people in certain populations consumed substances. An increasing number of people relied on alcohol, tobacco or drugs to cope with stressful or depressive situations generated by lockdowns or social restrictions. There were better rates of alcohol, tobacco and drug use which were associated with increased addiction, poor mental and physical health and increased emergency room episodes. This trend demonstrates the necessity of making mental health and addiction-related services accessible, primarily when a crisis occurs (Dubertret, 2020).

Suicidal ideation and behaviors also increased during the pandemic, especially among young adults, individuals with prior mental conditions and individuals experiencing extreme financial or social pressure. All these groups are at risk because of restrictions on care, social loneliness, grief and fear of the future. Therefore, phone calls, counseling and therapy are important first steps (Yang, 2024).

Healthcare providers on the frontlines experienced a long spell of excessive stress. They had to treat a high number of patients, deal with the risk of infection on a daily basis, work long shifts and encounter a lot of pain and loss. This leads to high burnout or emotional exhaustion, depersonalization and a lack of a sense of achievement in patients. Besides the personal well-being of these professionals, burnout can negatively impact patient safety and the functioning of healthcare systems. To assist, hospitals and health agencies should provide powerful institutional backup, simpler access to mental health interventions and acknowledgment of the pressure experienced by healthcare workers (Kinross, 2020).

The pandemic increased the rates of obsessive practices and health anxiety. The global concern over hygiene and prevention of illness prompted a large portion of the population to wash their hands frequently, clean regularly or watch for symptoms or headlines. Others went to an extreme with such steps and they may evolve into the most significant clinically authorized obsessive-compulsive disorders. Ambiguity regarding the virus and the unending stream of media reports coupled with changing health instructions nourished an overarching concern, causing elevated health anxiety or constant worry about being infected or infecting others with COVID-19. It may interfere with daily activities and aggravate pre-existing anxiety or obsessive-compulsive conditions. This raises concerns for mental health professionals who claim to encounter more clients with such problems; therefore, early intervention and assistance are important (Abramowitz, 2021).

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9. Conclusion

This review investigates the multi-organ consequences of COVID-19, demonstrates how much more than the respiratory system is impacted by SARS-CoV-2. Based on clinical, pathological and biomarker analyses in the first half of 2025, we reported long-term damage in ten physiological systems: cardiovascular, neurological, renal, hepatic, immune and psychological systems.

This review is unique in the way the outcomes of various disciplines are combined and attention is reflected on similar processes which are endothelial dysfunction, chronic inflammation and microvascular damage. These overlapping circles provide an understanding of how the same viral infection may lead to several chronic diseases, such as heart inflammation, brain fog, kidney damage and immune malfunction, even in those with initially mild symptoms.

In summary, this article provides a coherent framework through which clinicians and researchers can comprehend, diagnose and treat post-acute COVID-19 complications. It also indicates promising agents, including antifibrotics and immunomodulators which may have the potential to minimize or reverse organ injury.

In conclusion, COVID-19 cannot be simply regarded as a respiratory disease; it should also be considered a more systemic disease that leaves long-term effects. To consider effective clinical care approaches to public health and research priorities after the pandemic, a more comprehensive understanding of its silent multi-organ effects is required.

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