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Review Article

Post COVID-19 Effects on Different Body Systems: A Literature Review

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Introduction

In December 2019, there was an outbreak of novel coronavirus 2019 in Wuhan, then spread to other countries. The outbreak of a new coronavirus, termed officially by the world health organization (WHO) coronavirus disease 19 (COVID-19) and the international committee on taxonomy of viruses, has suggested SARS-COV-2 as the name of the virus that causes covid-19 (1-3). It has large numbers of deaths, especially since its first identifications in Wuhan, China. Because of this continuous increase in numbers, the virus has become a focus of many scientists and researchers, so through these studies done on the cases of COVID-19, it is discovered that it isn't just attacking the lungs. Still, it's causing harm to many-body systems, especially in more advanced cases of COVID-19. This is expected for the COVID-19 virus because we also harm many-body systems through our experience in dealing with previous Corona Viruses (SARS AND MERS).

Post COVID-19 Immunological Effects

The patient immune system is involved in COVID-19 infection pathogenesis (4). To date, there are not enough studies about explaining the immune response during and after COVID-19 infection (5). Studies showed multiple immunological effects appeared during and after the convalescent stage. Zhou et al. (6) said the highest IgM level appeared in one patient's sera and then turned into IgG by two weeks of infection and exhibit nucleocapsid protein-specific antibody response.

Krammer and colleagues (7) showed that patients exhibit anti-S antibodies post COVID-19 symptoms. Zhou et al.(6) also reported that the convalescent sera prevents SARS-CoV2 from entering its target cells. Recently, there are reports about the use of convalescent blood plasma to treat COVID-19 patients. This suggests the presence of a humoral immune response and its existence after the cure. Ling Ni et al. (8) proved in their study that there is a correlation between the neutralizing antibodies titers and the SARS-CoV-specific T cells. And this confirms the presence of protective immunity against it.

There are reports saying that the virus persists in the patients' sputum and feces after discharge from the hospital (9). Even more, other reports said that the virus was detected in the patients' sputum, and others noted in the patients' feces after negative PCR (10). Up till now, there's no

immunological or any scientific evidence suggesting the possibility of COVID-19 reinfection. Even those patients who tested negative and discharged and then returned with positive tests aren't thought to be reinfected. The reasons for getting these positive retests are

•Technical errors during sampling or transport

• Detoxication process

• Silent carrier state

• Also, those patients are young and tested with commercially available kits and then retested with more hypersensitive tests. So, we can say that the infection gives future patient protection against the virus (11).

Post COVID-19 Neurological Effects

SARs-COV-2 would possibly leave permanent neurotoxic effects on the brain in the long run. For instance, the Spanish flu outbreak of 1918-19 was associated with an increase in the prevalence of post-encephalitic Parkinsonism. It is currently unclear whether the SARS-COV-2 infection will immediately lead to mental health or neurodegenerative effects after the acute respiratory tract infection has passed (12-13).

Sub-acute signs that occurred 3–10 days after the development of COVID-19 symptoms, Guillain-Barre syndrome (14), and Miller-Fisher syndrome (15) cases have been reported. Also, clinically striking are cases of Kawasaki-like multisystem inflammatory syndromes now being recognized in children. COVID-19 survivors are at risk for developing long-term neurological consequences, either by aggravating a preexisting neurological disorder or initiating a new disorder. Findings that support this concern show that one-third of patients have evidence of cognitive impairment and motor deficits at the time of discharge. This is particularly relevant because overall, COVID-19 clinically affects the elderly most severely (16). There is a significant overlap in the age ranges when people usually develop neurodegenerative disease or cerebrovascular disease and the age of risk of most common COVID-19 infection. This overlap means that there is an urgent need for surveillance and care. COVID-19 is connected to a severe innate immune response and continued rise of systemic cytokine levels. Importantly, this innate immune response has been suggested to drive and predict mortality and severity (17).

Related inflammatory mediators and cytokines found to be elevated include C-reactive protein, interleukin-1β, interleukin-2, interleukin-2 receptor, interleukin-4, interleukin-10, interleukin-18, granulocyte colony-stimulating factor, interferon-γ, CXCL10, macrophage inflammatory protein 1-α, monocyte chemoattractant protein 1, and tumor necrosis factor-α (16-18). Concomitantly, most patients show signs of T cell decline with a decrease in lymphocyte counts. Because systemic inflammation promotes cognitive reduction and neurodegenerative disease, COVID-19 survivors will experience neurodegeneration in the following years (19, 20) through cytokine levels. We can predict the subsequent occurrence of hippocampal atrophy in patients that developed severe sepsis (21).

This evidence suggests that COVID-19 survivors are at high risk for the development of neurological disease in general and Alzheimer's disease in particular. Neurologists and psychiatrists should be alerted to a possible increase in such cases in patients surviving COVID- 19. Prospective studies are needed to investigate potential correlations between acute and subacute COVID-19 infections and long-term neurological sequelae in this patient cohort.

Few cases of Acute Disseminated Encephalomyelitis (ADEM) were related to MERS. The first case of "COVID-19 associated disseminated encephalomyelitis" was reported in a 40 years woman (22). This individual had COVID-19 symptoms followed 11 days later by dysphagia, dysarthria, a gaze preference, and facial weakness. A chest x-ray showed pneumonia, and an NPRT-PCR was positive for SARS-CoV-2. Head CT showed multiple areas of patchy hypo attenuation, and an MRI showed areas of increased FLAIR and T2 signal in the subcortical and deep white matter that were felt to be consistent with demyelination. Her CSF was normal.

A second reported case was in a 54 years woman who developed neurological deterioration (GCS 12) and seizures and had x-ray lesions consistent with COVID-19 and a positive NP RT-PCR for SARS-CoV-2 (23). Her MRI showed multiple periventricular T2 hyper-intense, non-enhancing lesions in the white matter of the cerebrum, brainstem, and spinal cord consistent with multifocal demyelination. Her CSF studies were unremarkable, including a negative CSF RT-PCR for SARS CoV-2. After she was treated with high dose dexamethasone, her symptoms gradually relieved. A single case of acute flaccid myelitis has also been described in COVID-19 (24). This patient experienced a flaccid areflexic lower limb paralysis and upper limb weakness, urinary and bowel incontinence, and a T10 sensory level. Unfortunately, neither CSF studies nor spine imaging was available, so the mechanism became unknown.

The most convincing example of ADEM-like pathology related to COVID-19 was in a 71 years patient who developed symptoms immediately after coronary bypass graft surgery that developed to respiratory failure and a hyper-inflammatory state. The examination of postmortem cases showed disseminated hemorrhagic lesions, brain swelling, and subcortical white matter pathology with perivenular myelin injury and necrotic blood vessels, and perivascular inflammation. The lesions had characters of both acute disseminated encephalomyelitis and acute hemorrhagic leukoencephalitis (25).

The rarity of post-infectious potentially immune-mediated cases following COVID-19 other than GBS and its variants, and the general scarcity of details, makes their status unclear. The patients of ADEM-like illnesses are hard to be differentiated from some of the patients with associated MRI white matter lesions and acute encephalopathy. Still, they can be distinguished from patients of encephalitis by the absence of CSF pleocytosis. GBS is a common neurological disease even in the absence of COVID-19, and identifying the magnitude of the COVID-19 risk and association will require better epidemiological data. However, the 5 cases of GBS present in a population of 1000-1200 COVID-19 patients seen over one month by Toscano et al. in Northern Italy suggest an incidence that is higher than expected in the general population (~1/100,000 person-years) (26). The mechanism of pathogenesis will need to be identified, and the efficacy of conventional therapies including IVIG and plasma exchange evaluated

Post COVID-19 Psychosocial Effects

Survivors of COVID-19 may have some psychosocial disorders as depression (27), post-traumatic stress (28), and anxiety (29); due to isolation for long times and the fear all the time about surviving, others fear stigmatization (30). Family members of COVID-19 survivors can also be psychologically affected by depression, anxiety, and stress (31). So psychosocial rehabilitation should be done by social workers or rehabilitation psychologists for patients with depression, anxiety, or PTSD. Also Education on the importance of participation in family and social activities should be included (32).

Post COVID-19 Cardiopulmonary Effects

Studies showed that because of inflammation and subsequent fibrosis of the myocardium, there is a risk of developing the subclinical myocardial disease and/or arrhythmia in COVID19 survivors, so we put into consideration the use of cardioprotective therapy as B-blockers, mineralocorticoid antagonists, and statins that are taken on the long run (33). Autopsy samples became available from the Toronto SARS epidemic, which demonstrated that SARS-CoV RNA in 35% of autopsied expired patients' hearts, which reveals the virus has cardiotropism and can infect the myocardium (34). The 7 of 20 patients who died of SARS-related coronavirus had SARS-COVID genome present in the heart that study by Oudit et al. (34) in this small case series, all patients who have SARS-COVID genome in there samples had increased myocardial inflammation, including macrophage infiltration, which suggests a direct effect from viral infiltration and the associated inflammation.

COVID-19 may lead to cardiac arrest by multiple mechanisms as myocarditis, thromboembolism, vascular inflammation, cardiac arrhythmia, and even acute coronary syndrome caused by fear of this pandemic. Social distance may also cause cardiac arrest because of substance abuse (35). According to a cohort study based on CMR and blood markers, there are numerous cardiac affections by the virus that may persist even after recovery, regardless of the severity of COVID-19 infection, time of diagnosis, or previously associated conditions. Using the LGE technique found that cardiac affection in the form of pericardial involvement by effusion with subsequent fibrosis and/or edema, myocardial edema and/or fibrosis associated with bad outcomes depending on the measurement of native T1 & T2 and also reduction of ejection fraction (36).

On lungs, SARS-CoV-2 has multiple effects that may occur as endothelial injury, pulmonary microthrombi, and vascular leak by several mechanisms such as mitochondrial inflammation infiltration, DNA damage, and oxidative stress. The risk of pulmonary vascular injury that leads to subsequent pulmonary hypertension that needs hospitalization or mortality risk would increase (37). Research-based on SARS and influenza showed that pulmonary injury in the form of pleural thickening, bronchiectasis, pneumatocele, pulmonary fibrosis, nodules, and the bullous formation and respiratory function as ventilation and diffusion dysfunction take several years to return to the normal (38).

COVID-19 Effects on the Digestive System

There is no doubt that COVID-19 will be represented in the history of medicine, especially concerning acute respiratory symptoms, with as many infectious and fatal outcomes as there are. But the involvement of the virus' digestive system is noticed too and cannot be pass unnoticed, it is not the size of the respiratory system, but its involvement cannot be ignored. According to a recent report, it is found that SARS-CoV-2 RNA was present in a stool sample on day 7 of illness (39). Therefore, the COVID-19 patient will be contagious until the 7th day of infection through faecoral route even if he was asymptomatic. Human pathogenic coronaviruses (severe acute respiratory syndrome coronavirus [SARS-CoV] and SARS-CoV-2) bind to their target cells through angiotensin-converting enzyme 2 (ACE2) (6, 40, 41), which is expressed by epithelial cells of the lung, intestine, kidney, and blood vessels (40). In the light of that, SARS-CoV-2 infects the epithelial cells of the gastrointestinal glands of the stomach, duodenum, and rectum, and, to a much lesser extent, of the esophagus, which led to the suggestion that infectious virions are secreted by gastrointestinal cells infected with the virus (42).

Patients infected with SARS-CoV-2 may have a low incidence of G.I. symptoms diarrhea in the range of 1%–3.8% (17, 43-45)and high incidence of G.I. symptoms with diarrhea and nausea in 10.1% and vomiting in 3.6% (46). Patients with these symptoms must be taken into account as a possible infection source even if these patients do not have respiratory symptoms (42). It was also noticed that SARS-CoV-2 was present in the saliva of most infected patients, which suggesting that salivary glands can also be infected by the virus (47).

Not only gastrointestinal tract is suspected to be infected by the virus, but the liver is also infected in up to 60% of infected patients, so liver damage is suggested particularly with the increase of the levels of the transaminases, hypoproteinaemia, and prolonged prothrombin time (48), and this elevation is accompanied by direct damage to the intrahepatic bile ducts (49), which indicate the liver is at least temporarily damaged. Also, liver damage is more common in patients who have severe COVID-19 disease (50).

Currently, we have no information about whether people with chronic liver diseases are at increased risk of getting COVID-19 or having severe COVID-19. But according to available knowledge and clinical expertise, people of any age who have severe underlying medical conditions, including people with liver disease, are more susceptible to severe illness from COVID-19, mainly if the underlying medical conditions are not well controlled (50). As

we mentioned before, SARS-CoV-2 use ACE2 to enter its target cells, which is expressed in key metabolic organs and tissues, including pancreatic beta cells, adipose tissue, the small intestine, and the kidneys (40, 51), so it is expected that COVID-19 will impact these cells, and cause pleiotropic alterations of glucose metabolism that could complicate the pathophysiology of preexisting diabetes or lead to new mechanisms of disease (52). This leads to complications of diabetes with a viral infection such as ketosis or ketoacidosis and induced diabetic ketoacidosis (DKA), increased ketosis increased length of hospital stay, and mortality (53, 54).

The interactions and effects of SARS-CoV2 on the esophagus, stomach, bile ducts, and pancreas have not been reported at present but may be detected in the future (55).

COVID-19 Effects on the Renal System

The kidneys are the body's filters that screen out waste products, toxins, and extra water from the body. SARS-CoV2 cause the formation of tiny clots in the bloodstream, which lead to clog the smallest blood vessels in the kidney and impair its function, plus the virus itself, infects the cells of the kidney. As we mentioned before, kidney cells have receptors that enable SARS-CoV-2 to attach to them, invade, and make copies of it, damaging those tissues. Kidney damage is more common in patients who have severe COVID-19 disease, even those who had no underlying kidney problems before they were infected with the coronavirus (56). According to early reports, about 30% of hospitalized patients with COVID-19 in China and New York had moderate or severe kidney injury. Reports from doctors in New York are saying the percentage could be higher (56). According to a prospective cohort study (57), 27% had hematuria at hospital admission, and 44% of COVID-19 patients had proteinuria, while 5% of patients developed acute kidney injury during in-hospital. Patients with kidney disease were more susceptible to death. Autopsy studies are becoming available that point to the possibility of a direct cytopathic effect of SARS-CoV-2 on renal cells (58, 59).

After the death of a 93-year-old woman with COVID-19, it is found that there are viral particles with typical features of coronavirus, indicating SARS-CoV-2 in podocytes through the analysis of kidney tissue by transmission electron microscopy taken 12-h postmortem. This patient was the first case in Europe, which had SARS-CoV-2 in the kidney. COVID-19 patients with injured glomerular epithelium may have very harmful compl8cations, particularly in the acute setting and diabetes, such as capillary barrier dysfunction and proteinuria, hematuria, altered coagulation, and worsening of edema, among multiple factors, so the evaluation of kidney function should be taken into account timely in every patient at risk. Sperati says that patients with COVID-19 related kidney damage should follow up with their doctors to ensure kidney function is returning to normal. Lasting kidney damage might require dialysis or other therapies, even after recovery from COVID-19 (56).

Dialysis Patients are more susceptible to the COVID-19; this fact was confirmed in an analysis of data from a German registry. About 2% of the dialysis patients had tested positive for the virus by the end of May. The ERA–EDTA COVID-19 database was established in March. Data on 1073 patients with COVID-19 and kidney failure from 26 countries had been entered in the ERACODA database since June 1. At 28-day follow-up, 21% of kidney transplant patients had died, as had 25% of dialysis patients (60).

Hematological Effects of COVID-19

Early studies suggest that there is a distinct coagulation disorder associated with COVID-19. Based on these studies, autopsy studies were done on COVID-19 patients, and their results demonstrated fibrin thrombi within distended small vessels and capillaries and extensive extracellular fibrin deposition (61). So we must understand the rate of bleeding and thrombotic manifestations related to COVID-19 coagulopathy and the clinical benefit of abnormal coagulation testing to

predict risk for thrombosis, bleeding, and severity of illness. According to recent studies, there is an increase in circulating D dimer (62-64) and a prolonged prothrombin time (P.T.) (17, 65). Both of them were associated with higher mortality, and regarding to laboratory findings, disseminated intravascular coagulation (DIC) developed in >70% of patients who succumb to the infection (65) and thrombotic rates in excess of 20% to 30%. Still, the use of prophylactic anticoagulation was not consistent between studies (66, 67).

Researches from the Tufts University School of Medicine in the U.S. demonstrated that the female hormone estrogen also increases the chance of blood clots during pregnancy and in women taking birth control pills or hormone replacement therapy, but they need more research to understand the effect of COVID-19 on coagulation to know if the virus aggravates the risk of blood clots and strokes associated with oral contraceptive pills, other estrogen therapies, and pregnancy-associated risks (68). Based on the high venous thromboembolism (VTE) rates detected in the studies, there are some investigators recommended experimental escalations to use anticoagulants as prophylaxis to prevent COVID-19 complications (67). A recent report investigated the association between mortality and the administration of low molecular weight heparin (LMWH) for at least seven days.

The use of anticoagulant therapy resulted in lower mortality in patients with sepsis-induced coagulopathy score ≥4 (LMWH: 40.0% vs. No-LMWH: 64.2%, p = 0.029), lower mortality in patients with D-dimer over six-fold the upper limit of normal (LMWH: 32.8% vs. No-LMWH: 52.4%, P = 0.017), but there was no overall benefit for patients on LMWH (LMWH: 30.3% vs. No-LMWH: 29.7%, respectively, p = 0.910) (69). Expert opinion guidance statement didn't assert standard prophylactic dose or escalated anticoagulation was optimal to prevent thrombotic events (70). Bikdeli's consensus group's recommendations advocated extended prophylaxis with LMWH or direct oral anticoagulants (DOACs) as reasonable after hospital discharge, with individualized risk stratification for thrombotic and hemorrhagic risk (71).

Effects of COVID-19 on Genitalia and Sexuality

Most of the COVID-19 patients are males, and also affect their genitalia more than females. Yet, females are more protected against COVID-19 (72). The effect of COVID-19 infection on the genital organs is still unclear (73). ACE2 receptors are represented in the genitalia of males and females (74, 75). These receptors are claimed to be the host for the COVID-19 virus. So, it is expected to injure the testicular cells. Fever, one of the COVID-19 infection symptoms, is claimed to cause orchitis like that with mumps and testicular degeneration (76).

Xu et al. showed in their study that the autopsy, taken from six patients, died with SARSCoV=1, proves the presence of orchitis in those patients. The most common theory for explaining this case is that fever triggers an immune response in the form of secondary immune orchitis (77). Xu et al. also showed in a previous study that the SARS virus affects Leydig cells, causes damage to the blood-testis barrier and the seminiferous epithelium (78). Ma et al. showed in their study the occurrence of an increase in L.H. hormone and decrease in testosterone/L.H. and FSH/LH ratios. This leads to hypogonadism (79).

Effects of COVID-19 on Smell and Taste Sensation

As It's a challenge to obtain viral culture from olfactory epithelium and the limitation of cases presented by sensorineural viral anosmia, but it's observed that there is an association between COVID-19 and anosmia & sometimes dysgeusia, so SARS-CoV2 is supposed to be neuroinvasive and neurotropic (80). There is no evidence of chronic olfactory affection as it needs study on a large population over the years, so it remains under researches (81). As the virus affects the olfactory epithelium, not the olfactory sensory neurons because of the presence of angiotensin-converting enzyme 2 receptor and transmembrane serine

protease2 on the epithelium, so olfactory bulb remains normal in size with MRI (82).

Effects of COVID-19 on Bone and Joints

Not only a fever, dry cough, and shortness of breath are COVID-19 common symptoms, but also myalgia and arthralgia have also been reported in 14.8 % of patients. Arthralgia means joint pain, but arthritis is a disease that causes joint inflammation, which can, in turn, cause joint pain and stiffness, symptoms such as joint stiffness, aches, muscle pain and inflammation, such as swelling, tenderness, and redness around the joint may accompany the arthralgia (83). People with osteoporosis and those taking medications to treat osteoporosis must know that having osteoporosis does not increase the risk of either contracting coronavirus or having severe complications. However, staying fracture-free now is critical for anyone with osteoporosis because healthcare systems are over-stretched, with general recommendations urging people to avoid hospitals and doctor's offices unless necessary (84).

Post COVID-19 Pain

The arising COVID-19 spread out is expected to lead to prompt and chronic mental health disorders. During and after SARS and MERS spread, infected persons often suffer psychological problems, anxiety or depressive, psychiatric disorders, and chronic fatigue (85, 86). We should be considered chronic ache in the biopsychosocial pattern, which includes symptoms as a complicated reaction between biological, psychosocial, and social triggers (87, 88). Predisposing factors as hereditary factors, past ache experience, and injuries might be physical or emotional, or biological triggers in persons with a weak stress response system (89-93). The COVID-19 pandemic possess multiple attributes that could cause chronic pain practically with anxiety over numerous months

Recommendations

Although, until now, we do not have enough evidence on short term and long term effects of COVID-19 if, by any chance, we can prevent that damage, we should do. We think that an MRI brain and heart at the time of presentation and time of discharge would explain how the COVID-19 be destructive in a person, moreover, follow up with a cardiologist, pulmonologist, and neurologist is mandatory for a while. Multidisciplinary teams during the COVID-19 pandemic should be included in any hospital to save more lives.

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