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Neurological and systemic manifestations in Long covid: Underestimated sequel of covid's pandora!

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Abstract

COVID-19 pandemic is phase of decline worldwide and in short time declared as pandemic is over. Globally, more than half cases are now facing either one vague or nonspecific and or organic symptoms in post covid phase. Long covid symptoms has significant impact on quality of life in those cases suffered from disease in recent past and lingering to almost two years since infection. Importantly, not all cases of COVID-19 were shown long covid symptoms. Most common long covid symptoms as joint pain, fatigability, chest discomfort, shortness of breath, hair loss, chest pain, weight gain, anxiety/depression & memory impairment. Neurological, gastrointestinal, renal, endocrine & rheumatological systems were badly affected in long covid. Brain fog, renal dysfunction, and diabetes mellitus were frequently documented as systemic manifestations of long covid. Pathophysiology resulting into long covid manifestations is still not completely validated. Researchers have reported 'immune dysregulation', 'autoimmunity', 'antigenic mimicry' & 'coagulation abnormalities' are probable pathophysiological mechanism for long covid. Some of the long covid effects shown complete reversibility including post covid lung fibrosis. Reboot system to restore immune dysregulation and recovery in long covid is real concern. Protocolised approach is required for timely workup of cases with long covid symptoms. Effect of vaccination in preventing long covid is not known and impact of evolution of new strains COVID-19 in future on ongoing long covid is real concern.

Keywords: Long covid; Brain fog; Renal; Diabetes mellitus; COVID-19; Antigenic mimicry

1. Introduction

COVID-19 is caused by the infection of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), a singled-stranded RNA coronavirus. Coronavirus disease 2019 (COVID-19) has escalated into an unprecedented global pandemic since the first case was identified in December 2019. [1] Though a majority of patients recovered from COVID-19 infections, over 70%-of-survivors were reported to have impairments in one or more organs 4 months after initial symptoms. [2] They are termed "long haulers" [3], or patients living with "Chronic COVID syndrome", "post-COVID-19 syndrome", or "postacute-COVID19 [4,5]. Extensive symptoms have been reported by convalescent patients, such as chronic cough, chest tightness, shortness of breath, cognitive dysfunction, and extreme fatigue, in Long COVID-19 Syndrome [6].

Postacute-COVID-19 is defined as ongoing symptomatic COVID-19 for people who still have symptoms 4 and 12 weeks after the onset of acute symptoms, while post-COVID-19-syndrome is for people who still have symptoms for more than 12 weeks after onset of acute symptoms according to the UK NICE guidelines [6]. In a systematic review and meta-analysis looking into the long-term effect of COVID-19, the five most common symptoms are fatigue (58%), headache (44%), attention disorder (27%), hair loss (25%), and dyspnea (24%) [7,8]. These symptoms may take months to

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resolve, even among non-hospitalized persons with mild illness course in the acute phase [9,10]. Long-term effects of COVID-19 infection on respiratory, cardiovascular, neurological, and metabolic system of recovered COVID-19 patients. [figure 1] [11]

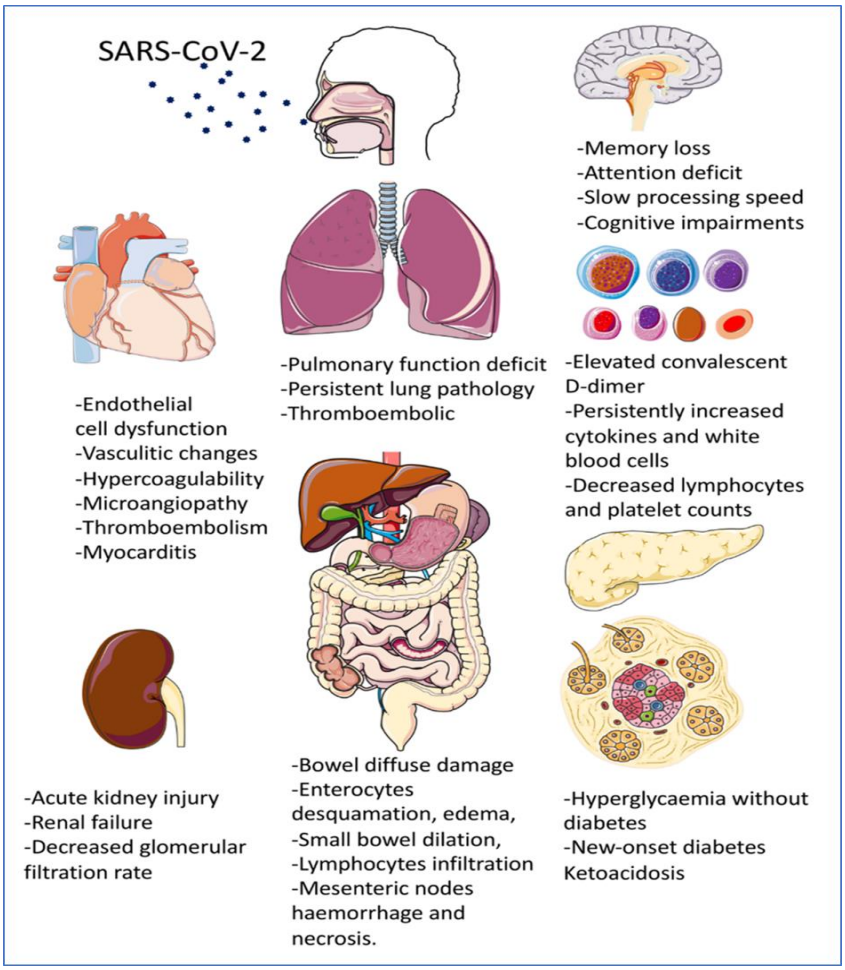

Figure 1 Summary of multi-system clinical presentations of Long COVID-19 Syndrome

2. Pathophysiological mechanisms for Long covid:

The exact mechanism behind the persistence of symptoms has to be identified. Reason for the persistence of symptoms can be the sequelae of organ damage, varying extent of injury (organ damage) and varying time required for the recovery of each organ system, persistence of chronic inflammation (convalescent phase) or immune response/auto antibody generation, rare persistence of virus in the body, nonspecific effect of hospitalization, sequelae of critical illness, post-intensive care syndrome, complications related to corona infection or complications related to co morbidities or adverse effects of medications used.[12,13] SARS-CoV-2 uses the angiotensin-converting-enzyme 2 (ACE2) receptor to enter the cell through binding with spike-like protein (S-protein) [14,15], though some other receptors may also be involved. ACE2, therefore, plays a vital role in the pathogenesis of COVID-19. ACE2 is widely expressed in different body tissues, including the lung, heart, liver, kidney, and gastrointestinal system [16]. Thus, multi-organ injuries are observed in COVID-19, such as acute respiratory distress syndrome, acute myocardial injury, acute kidney injury, and acute liver injury. Survivors of severe COVID-19 are also found to be with multi-organ impairments after discharge [17]. Severity of illness increases with more lung parenchymal involvement as documented with chest imaging in proportion with inflammatory markers such as IL-6, CRP, Ferritin and LDH. [18-22] Authors have documented persistent smouldering infection as underlying mechanism for long covid. [23] Second hypothesis is that mast cell activation syndrome could possibly contribute to long covid symptomatology. [24-25] The third hypothesis put forth that sustained dysregulated immune system activation with subsequent chronic low-grade inflammation could lead to pathological consequences like autoimmunity leading to organ dysfunction. [26-27] or antigenic cross reactivity or mimicry. [28-31] Authors have documented long-lasting functional alterations of T-cells, with persistence of cytotoxic profile with decrease in dendritic cells revealed 7 months post-infection. [32-33] Residual excessive

inflammation after infection & auto-antibodies against immunomodulatory proteins or against tissues are other proposed mechanism for long covid. [34-35] Other possible hypothesis is persistence of SARS-CoV-2 in the lower gastro-intestinal tract was revealed in asymptomatic subjects several months after COVID-19, suggesting that residual viral proteins within tissues could possibly be the basis for a persisting immune reaction.[36]

2.1. Long covid symptom & possible mechanisms for gastrointestinal system dysfunction

The incidence of post-COVID-19 related gastrointestinal symptoms is between 3% and 79%, in various reports [37,38]. The gastrointestinal system is rich in ACE2 and furin expression, a serine protease which cleaves the S-spike protein into S1 and S2, leading to easier attachment of the virion to the ACE receptors and the cell membrane [39,40]. Viral shedding is observed in fecal samples at least 5 weeks after symptoms onset, supporting the viral proliferation and fecal-oral transmission hypothesis [41-43]. This results in diffuse damage of the bowel, leading to enterocytes desquamation, edema, small bowel dilation, lymphocytes infiltration, and mesenteric nodes hemorrhage and necrosis. High fecal calprotectin levels were found in patients with persistent diarrhea but without the history of prior inflammatory bowel disease, indicating an underlying intestinal inflammatory process [44]. Studies have suggested the possibility of persistent gastrointestinal dysfunction in various ways: plasma cells and lymphocytic infiltrations into lamina propria of intestinal tissues [45], intestinal dysbiosis [46], and high cytokines level were detected in stool samples [47]. The clinical presentation is similar to irritable bowel syndrome, and the symptoms develop after the resolution of acute COVID-19 infection [48].

Mild impairments in various gastrointestinal organs are observed in low-risk young patients with Long COVID-19 Syndrome ($p < 0.05$) [49]. These include the liver (10%), pancreas (17%), and spleen (6%). The detailed mechanism is still under investigation. Multi-organ injury to hepatobiliary systems may be related to drug-induced liver injury, systemic inflammatory reactions, hypoxia-reperfusion hepatic injury, and possible direct viral injury by SARS-CoV-2 [50]. Typical liver injury pattern can be mixed hepatitic and cholestatic in nature: with elevation of aspartate transferase (AST) and alanine transferase (ALT), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP) [51,52]. A 2-month follow-up study showed that the liver enzymes (ALT, AST, GGT, and ALP) remained persistently elevated 14 days after discharge, while the liver functions in the majority of survivors normalized 2 months after hospital discharge [52]. However, patients with liver cirrhosis suffer more mortality compared with those without cirrhosis according to a study involved with 745 patients, suggesting COVID-19 has a significant impact on chronic liver condition [53].

Though pancreatic injury has been observed in COVID-19 survivors [54], the rise of serum amylase and lipase may be due to other causes [55]. Hyperamylasemia may be related to salivary gland inflammations [56], chronic alcoholism, and COVID-19 associated diarrhoea [57].

2.2. Long covid symptom & possible mechanisms for renal system dysfunction

Renal impairments have been commonly observed in COVID-19 patients due to the high abundance of ACE2 expression in the kidneys [58]. A systematic review and meta-analysis showed that acute kidney injury (AKI) was observed in half of the non-survivors and less than 1% in survivors [59]. This is consistent with another study which showed that 1.4% of patients experienced renal failure in longer term follow-up of Long COVID-19 Syndrome [60]. Previous studies showed that both chronic and acute kidney injury were associated with significant risks of mortality and morbidity [61,62]. COVID-19 associated estimated glomerular filtration rate (eGFR) declines more rapidly than those without COVID-19 infections [63].

Among discharged survivors with AKI previously, one in three would still depend on renal replacement therapy (RRT) at discharge, and one in six remains RRT dependent 60 days after hospital admission [64]. Longer follow-up duration shows more promising results for restoration of renal functions in survivors with previous AKI: over 90% achieve variable degrees of renal recovery, with over 60% achieving complete recovery [65]. In the management of COVID-19 associated kidney injury, early recognition of kidney involvement and the use of preventive and therapeutic measures to limit AKI and subsequent progression to more severe stages are crucial to reduce morbidity and mortality [66].

Several hypotheses have been postulated to explain the pathophysiology of COVID-19-induced AKI. These include sepsis, renal infarction, respiratory failure-induced AKI, and direct viral invasion of host cells [67]. Around 60% patients experienced sepsis, and 20% experienced septic shock, in the earliest study in Wuhan, China [68]. Kidney infarctions were observed in another study [69]. There is close association between respiratory failure and AKI, showing that nearly 90% patients receiving mechanical ventilations developed AKI [70,71].

2.3. Long covid symptom & possible mechanisms for Endocrine system dysfunction

Patients with Long COVID-19 Syndrome may also present with endocrinopathy, such as diabetes mellitus (DM). As SARS-CoV-2 binds to ACE2 receptors, which are also expressed in pancreatic β-islet cells, it is plausible that the virus may disturb glucose metabolism by intruding the cells. Several decades ago, virus-associated DM was reported on enteroviruses, such as coxsackievirus B [72], rotavirus [73], mumps virus [74], and cytomegalovirus [75]. β-islet cells infected with the viruses triggers phagocytosis by macrophages, thereby triggering autoimmune responses because of the exposure of the antigen from damaged islet cells, which leads to type I DM (T1DM). This autoimmunity promotes anti-viral T-cell memory, which reacts with new infections. As a result, antibodies are likely to target the islet cells, hence aggravating the T1DM. There are also some precedents of SARS coronavirus binding to ACE2 receptors and damaging islets cells [76].

Hyperglycemia without DM and new-onset DM are both associated with a poor course of COVID-19 after excluding risk factors, such as obesity and corticosteroid administration [77]. Newly diagnosed DM cases associated with COVID-19 are mostly T1DM. [78,79]. These patients have favorable outcomes when the infections do not cause hypoxemia, but the outcome is poor for patients with severe COVID-19 [80,81].

In spite of the association between T1DM and the COVID-19, there is still insufficient correlation analysis. A report also shows the association of insulin resistance with poor outcome of COVID-19 by using TyG index, suggesting the possibility of new-onset T2DM because of the infection [82]. Owing to the limited number of reported cases, there may be sampling bias causing unexpected findings or conclusions.

2.4. Long covid symptom & possible mechanisms for haematological System dysfunction

A study showed that over 30% survivors had elevated d-dimer and 9.5% had elevated C-reactive protein levels with a median follow-up of 54 days after discharge [83]. Elevated convalescent D-dimer were more common with hospitalized COVID-19 survivors whose age was over 50 years ($p < 0.001$) [84]. The C-reactive proteins returned to normal in more than 90% of survivors at a median of 80.5 days (range 44–155) after initial diagnosis [84].

Cytokines are also shown to be persistently increased 3 months after initial infection [22]. Interleukin-4 (IL-4) and IL-6 are increased in all survivors. In a systematic review and meta-analysis involving 21 studies, increased white blood cells count, decreased lymphocytes and platelet counts, elevated biomarkers of inflammations (IL-6, IL-10), evidence of cardiac muscle injury (cardiac troponin level), liver and renal functions, coagulation profiles, and serum ferritin were strongly associated with progression of COVID-19 [85,86]. Research data on the trend of these biomarkers in COVID-19 survivors is lacking. More research is required to select the most prognostic biomarkers for monitoring clinical progression of Long COVID-19 patients.

Since COVID-19 may induce endothelitis and systemic inflammations after recovery [87,88], anticoagulants in high-risk patients should be considered. Cardiovascular risk factors should be controlled with the usual guidelines. Venous thromboembolism risk assessment is recommended and the use of thromboprophylaxis with rivaroxaban, betrixaban, or low-molecular weight heparin for high-risk patients are acceptable [89,90]. Close monitoring of D-dimer levels with IMPROVE D-dimer score is recommended as a part of thromboembolism risk. Age-adjusted D-dimer cut-off level should be further explored to rule out venous thromboembolism in both patients with active COVID-19 infection and survivors [91].

2.5. Long covid symptom & possible mechanisms for Neurological System dysfunction

Long COVID-19 Syndrome is associated with mood changes, cognitive difficulties, headache, fatigue, dizziness, memory loss, confusion, and attention deficit [92]. Previous studies on coronavirus SARS-CoV-1 and MERS-CoV show survivors may live with neurological symptoms, such as memory loss, attention deficit, and slow processing speed [93]. A significant proportion of COVID-19 survivors complains of memory loss more than 100 days after hospital discharge [94]. Anatomically, the cognitive impairments and memory loss may be associated with ischemic damages to cerebral white matter [95]. The underlying reason for COVID-19-related neurological damage may be related to blood vessel damage, impaired oxygen supply, viral infiltration into the central nervous system, and inflammatory cytokines-mediated cellular damage [96.97]. Hypoxic injury, microbleedings, and neuronal inflammations in different areas of the brain have been observed in various reports [98.99]. One of the notable sites is the brainstem. The brainstem contains numerous distinct nuclei and subparts that regulate various physiological process: respiratory, cardiovascular, gastrointestinal, and neurological. As neurons do not readily regenerate, brainstem dysfunction may be long-lasting, contributing to Long COVID-19 Syndrome [100]. Radiologically, hypometabolism in various brain areas have been observed in post-COVID-19 patients, indicating underlying SARS-COV-2 related neurotropism [101].

Mitochondrial swellings secondary to hypoxic damage are being observed in Long COVID-19 patients [102]. Neurons with high metabolic demand of oxygen, thus, become dysfunctional, leading to impairments of cognitive functions. This has been similarly observed in other pandemics [103]. The hypometabolism in parahippocampal gyrus, thalamus, and some white matter may be a secondary result of hypoxic damage to these areas, leading to memory loss and cognitive dysfunctions [104].

The multifocal neurological damages in COVID-19 patients result from indirect T-cell and microglia damage in the brain, similar to strokes and neuroinflammatory diseases [105]. Cytokines IL-4 and IL-6 are also shown to be persistently elevated in individuals reported with neurological symptoms [106]. Protein markers related to neuronal dysfunction are increased compared with historic control level. These markers include: amyloid beta, neurofilament light, neurogranin, total tau, and p-T181-tau. The increased markers may be accountable for some psychopathologies, such as anxiety and depression, in COVID-19 survivors [107]. Over 40% of survivors without prior psychiatric conditions live with depression within 90 days of recovery from severe COVID-19 associated respiratory failure [108].

The prevalence of psychological distress is high in the initial phase after discharge [109]. A study of 126 survivors of COVID-19 patients showed 31.0%, 22.2%, and 38.1% of them suffered stress, anxiety, and depression, respectively, suggesting the rate of psychological distress is high in early convalescence [109]. Thus, timely evaluations and treatment are required. Severely ill patients who receive complicated procedures, such as intubations or those who experienced severe complications, may be considered as high-risk of post-traumatic stress disorder [110]. Though evidences about the association between long COVID and post-traumatic stress disorder (PTSD) is lacking, assessing PTSD for these survivors is still recommended [110].

Lastly, we recommend to assess inflammatory markers assessment in all cases suspected with any neurological symptoms during follow up in post covid care setting especially those having any systemic long covid manifestations [111-116]. These markers have played significant role in assessment of cases form entry point to follow up and sequential change will guide to predict early chances of Neurological symptoms in selected symptomatic cases of recovered COVID-19 cases [114-120].

3. Conclusion

Long covid in known complication of Covid-19 disease irrespective of severity and hospitalization. Long covid can be predicted during hospital discharge in selected cases with inflammatory and coagulation pattern abnormalities. Long covid should be actively evaluated in those cases with aggressive interventions in indoor units and comorbidities. Importantly long covid pulmonary manifestation as lung fibrosis is reversible and now considered as post covid sequelae.

Long covid is underestimated, improperly evaluated and half-heartedly treated during follow-up due to lack of suspicion especially in geriatric cases. All treated cases need prompt evaluation, more awareness regarding its manifestations and its impact on quality of life is must to have successful treatment outcome. Systemic manifestations are really considered as sequel of Covid's Pandora and miserable pathological process of ongoing viral pandemic.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest to declared.

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