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(RESEARCH ARTICLE)

Long COVID symptoms, pathophysiology and possible mechanisms: Still, we are learning!

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Abstract

Long COVID is an unpredicted sequel of COVID-19 disease documented nearly in half cases globally. Long COVID is multisystem syndrome with nonspecific symptoms and organic signs of unidentified pathology occurs after COVID-19 disease. Long COVID symptoms has been documented in some cases irrespective of disease severity or hospitalization. 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 (ten in number) as joint pain, fatigability, chest discomfort, shortness of breath, hair loss, chest pain, weight gain, anxiety/depression & memory impairment. Pathophysiology resulting into long COVID manifestations is still not completely validated. Researchers have reported 'immune dysregulation' and '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. Long COVID symptoms cases are more health conscious and usually follows pattern of doctor shopping due to underestimation by family physicians either due to lack of suspicion or lack of knowledge regarding treatment protocol.

Keywords: Long COVID; Joint pain; Fatigability; Chest discomfort; Shortness of breath; Hair loss; Chest pain; Weight gain; Anxiety/depression; Memory impairment

1. Introduction

"Long COVID" is a term used to describe presence of various symptoms, even weeks or months after acquiring SARS-CoV-2 infection irrespective of the viral status. It is also called "post-COVID syndrome". It can be continuous or relapsing and remitting in nature. There can be the persistence of one or more symptoms of acute COVID, or appearance of new symptoms. Majority of people with post-COVID syndrome are PCR negative, indicating microbiological recovery. In other words, post COVID syndrome is the time lag between the microbiological recovery and clinical recovery. Majority of those with long COVID show biochemical and radiological recovery [1,2]. Long COVID can be divided into two stages-post acute COVID where symptoms extend beyond 3 weeks, but less than 12 weeks, and chronic COVID where symptoms extend beyond 12 weeks [3].

Thus, among people infected with SARS-CoV-2 the presence of one or more symptoms (continuous or relapsing and remitting; new or same symptoms of acute COVID) even after the expected period of clinical recovery, irrespective of the underlying mechanism, is defined as post COVID syndrome or Long COVID. It is estimated that 31%-69% of COVID-19 survivors will experience long COVID symptoms after initial recovery from SARS-CoV-2 infection [4,5]. Generally,

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initial symptoms of long COVID symptoms include fatigue (29%), muscle pain, palpitations, cognitive impairment (28%), dyspnoea (21%), anxiety (27%), chest pain, and arthralgia (18%). Correspondingly, a recent meta-analysis of 36 studies identified fatigue, cognitive impairment, joint pain, anxiety, and depression as primary clinical symptoms of long COVID [6]. A massive international survey found fatigue, malaise and cognitive impairment as the most prevalence symptoms experienced among individuals with reported long COVID [7]. Approximately 30% of non-hospitalized COVID-19 patients reported lingering symptoms 2 months after initial infections. Similarly, less than 1% of COVID survivors achieved complete recovery at 80 days after infection [8].

The lingering symptoms of long COVID reflect chronical damages of multi-systemic organs. Such health conditions post a significant burden on the quality of life among COVID survivors. [9-11] Post pandemic systemic affections and longer symptomatic phases labelled as "Long COVID" is documented in currently ongoing pandemic, and it has been also described in Russian flu, where many affected patients had crippling and long manifestations [12].

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 [13,14].

2.1. Dysregulated inflammation in ongoing COVID and its impact on long COVID

COVID-19 pneumonia is heterogeneous disease with variable effect on lung parenchyma, airways and vasculature leading to long term effects on lung functions. Pathophysiological mechanism is immune activation, inflammatory, thrombogenic and direct viral affection to lungs and extrapulmonary tissues [15]. COVID-19 pandemic has been studied over last two and half years for its highly pathogenic nature of corona virus. Main hurdle for acquiring immunological memory after natural infection is genetic change in spike proteins of nucleocapsid of novel corona virus after certain interval resulting into different waves documented in different geographical settings. Dysregulation in the immune system can lead to an unappropriated local and systemic immune responses and subsequently the rapid spread of the virus, leading to severe COVID-19 disease.

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 [16-19]. Authors have documented persistent smouldering infection as underlying mechanism for long COVID. [20] Second hypothesis is that mast cell activation syndrome could possibly contribute to long COVID symptomatology [21-22]. 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. [23-24] or antigenic cross reactivity or mimicry. [25-28] 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 [29-30].

Residual excessive inflammation after infection & auto-antibodies against immunomodulatory proteins or against tissues are other proposed mechanism for long COVID. [31-32] 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 [33].

2.2. Coagulation abnormalities in ongoing COVID and its impact on long COVID

Acute COVID-19 infection is also characterized by dysregulated, circulating inflammatory biomarkers, hyperactivated platelets, damaged erythrocytes and substantial deposition of microclots in the lungs [34-36]. In those discharged with elevated biomarkers, 30.1% and 9.5% had persistently elevated D-dimer and C reactive protein, respectively. 38% of chest radiographs remained abnormal with 9% deteriorating [37]. In the largest global study to-date on this issue, a survey of 3,762 Long COVID/PASC patients, from 56 countries found nearly half still could not work full-time 6 months post-infection, due mainly to fatigue, post-exertional malaise, and cognitive dysfunction [38].

Data is available regarding vascular changes and thrombotic microangiopathy, diffuse intravascular coagulation and large-vessel thrombosis are major reasons for a poor COVID-19 prognosis [39]. These comorbidities are linked to multisystem organ failure, as well as pulmonary vascular endothelialitis. The presence of endotheliopathy in particular,

is likely to be associated with critical illness and death [40]. It is also suggested that endothelial dysfunction contributes to COVID-19 -associated vascular inflammation, COVID-19 -associated coagulopathy, and pulmonary fibrinous microthrombi in the alveolar capillaries. In some instances, patients present with a significant elevation in D-dimer/fibrinogen degradation products. D-dimer and fibrinogen degradation products may indicate the failing attempt of the fibrinolytic system to remove fibrin and necrotic tissue from the lung parenchyma (and also from the circulation), but being consumed or overwhelmed in the process [41-42].

Other possible mechanism is dysregulation of the balance in fibrin-forming and fibrin-dissolving (plasmin generation) pathways and simultaneous presence of persistent anomalous (amyloid) microclots and a pathological fibrinolytic system is a major aspect of COVID-19 pathogenesis. The plasmin and antiplasmin balance may be central to this phenomenon. An important element of the fibrinolytic system is the conversion of circulating zymogen plasminogen to its active form plasmin. Endogenous activators of plasminogen are the tissue-type plasminogen activator (tPA) and urokinase-type plasminogen activator (uPA). The catalytic activity of tPA is largely dependent on the presence of fibrin, as both tPA and its substrate plasminogen bind to the lysine residues on fibrin, using it as a cofactor for plasmin generation. Plasmin is the effector protease of the fibrinolytic system, well known for its involvement in fibrin degradation and clot removal. Plasmin is also recognized as a potent modulator of immunological processes by directly interacting with various cell types including cells of the vasculature (endothelial cells, smooth muscle cells) In fact, the removal of misfolded proteins and the maintenance of tissue homeostasis seem to be major physiological functions of plasmin resulting into acute or lingering overload of anomalous (amyloid) fibrinogen microclots in circulation [43-45].

3. Does long COVID manifestations can be predicted during hospitalization?

COVID-19 pneumonia is heterogeneous disease with variable effect on lung parenchyma, airways and vasculature leading to long term effects on lung functions. Pathophysiological mechanism is immune activation, inflammatory, thrombogenic and direct viral affection to lungs and extrapulmonary tissues [46]. COVID-19 pandemic has been studied over last two and half years for its highly pathogenic nature of corona virus. Main hurdle for acquiring immunological memory after natural infection is genetic change in spike proteins of nucleocapsid of novel corona virus after certain interval resulting into different waves documented in different geographical settings. Dysregulation in the immune system can lead to an unappropriated local and systemic immune responses and subsequently the rapid spread of the virus, leading to severe COVID-19 disease.

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Residual excessive inflammation after infection & auto-antibodies against immunomodulatory proteins or against tissues are other proposed mechanism for long COVID [58-59]. 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 [60].

3.1. Symptomatology of long COVID

It is estimated that 31%-69% of COVID-19 survivors will experience long COVID symptoms after initial recovery from SARS-CoV-2 infection [61,62]. Generally, initial symptoms of long COVID symptoms include fatigue (29%), muscle pain, palpitations, cognitive impairment (28%), dyspnoea (21%), anxiety (27%), chest pain, and arthralgia (18%). Correspondingly, a recent meta-analysis of 36 studies identified fatigue, cognitive impairment, joint pain, anxiety, and depression as primary clinical symptoms of long COVID [63]. A massive international survey found fatigue, malaise and cognitive impairment as the most prevalence symptoms experienced among individuals with reported long COVID. [64] Depiction of the clinical course of long COVID shown in figure 1 [65].

3.2. Possible mechanisms for fatigue

Fatigue is more profound than being overtired; it is unrelenting exhaustion and a constant state of weariness that reduces a person's energy, motivation, and concentration. Chronic fatigue following viral infection may be the result of miscommunication in the inflammatory response pathways [66]; however, a cross-sectional analytical study found no association between pro-inflammatory markers and long-term fatigue in COVID-19 patients with persisting fatigue [67]. It is likely that a range of central, peripheral, and psychological factors play a role in the development of post-COVID-19 fatigue. A narrative review explains that congestion of the glymphatic system and the subsequent toxic build-up within the central nervous system (CNS), caused by an increased resistance to cerebrospinal fluid drainage through the cribriform plate as a result of olfactory neuron damage, may contribute to post-COVID-19 fatigue [68].

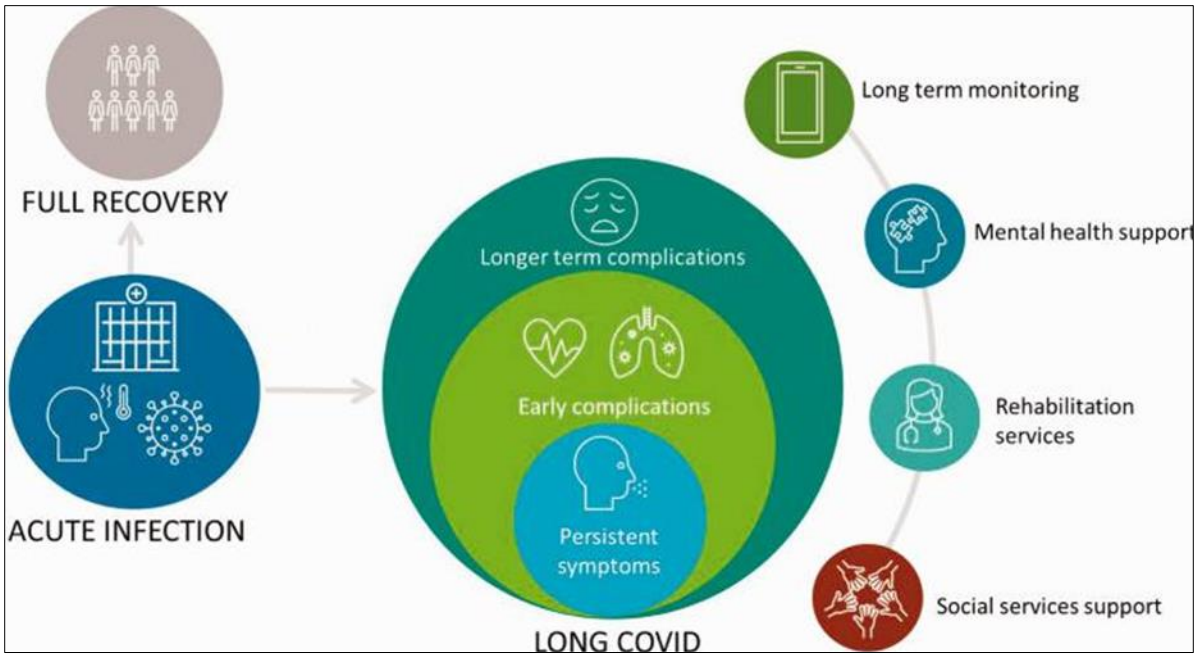

Figure 1 Depiction of the clinical course of long COVID

Hypometabolism in the frontal lobe and cerebellum has also been implicated in COVID-19 patients with fatigue and is likely caused by systemic inflammation and cell mediated immune mechanisms, rather than direct viral neuro-invasion. [69,70] It is unknown whether this finding continues into long COVID.

Negative psychological and social factors associated with the COVID-19 pandemic have also been linked to chronic fatigue [71,72]. Lastly, peripheral factors such as direct SARS-CoV-2 infection of skeletal muscle, resulting in damage, weakness, and inflammation to muscle fibers and neuromuscular junctions may contribute to fatigue [73-76]. Overall, it is probable that several factors and mechanisms play a role in the development of post-COVID-19 fatigue.

Post-COVID-19 fatigue has been compared with myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), with many overlaps between the two [77]. Symptoms common to both ME/CFS and long COVID include fatigue, neurological/pain, neurocognitive/psychiatric, neuroendocrine, autonomic, and immune symptoms, with both ME/CFS and long COVID patients having long symptom durations, reduced daily activity, and post-exertional malaise [77]. ME/CFS remains enigmatic, therefore, research into long COVID may assist in developing understanding of ME/CFS and vice versa.

3.3. Possible mechanisms for Dyspnea

As COVID-19 is principally a respiratory illness, acute illness can cause substantial damage to the lungs and respiratory tract via SARS-CoV-2 replication inside endothelial cells, resulting in endothelial damage and an intense immune and inflammatory reaction [77,78]. Those who overcome the acute infection may develop long term lung abnormalities, leading to dyspnea [79]; however, most individuals who develop long-term breathing difficulties post-COVID-19 have no signs of permanent or long-lasting lung damage [80]. It is likely that only those at high risk of developing breathing difficulties, including older people, those who endure acute respiratory distress syndrome, those who have extended

hospital stays, and those with pre-existing lung abnormalities, are prone to develop fibrotic-like changes to lung tissue [81]. The fibrotic state observed in some patients with ongoing dyspnea may be provoked by cytokines such as interleukin-6, which is raised in COVID-19 [82] and is involved in the formation of pulmonary fibrosis [83]. Pulmonary vascular thromboembolisms have been observed in patients with COVID-19 [84] and may have detrimental consequences in patients with long COVID.

3.4. Possible mechanisms for Cardiovascular abnormalities

Cardiac injury and elevated cardiac troponin levels are associated with a significantly increased risk of mortality in patients admitted to hospital with acute COVID-19 infection [85,86]. Persisting cardiovascular abnormalities may be burdensome for people with long COVID. ACE2 receptors are highly expressed in the heart, [87] providing a direct route of infection for SARS-CoV-2. Studies have shown that sarcomere disruption and fragmentation, enucleation, transcriptional changes, and an intense local immune response occurs in cardiomyocytes infected by SARS-CoV-2 [88,89]. Pathological responses to acute cardiac injury and viral myocarditis, such as endothelial damage and microthrombosis, can lead to the development of coagulopathy, [90] while chronic hypoxia and an increase in pulmonary arterial pressure and ventricular strain may further precipitate the incidence of cardiac injury in people who have had COVID-19 [91]. Furthermore, sustained immune activation can lead to fibrotic changes [92] and displacement of desmosomal proteins, [93] which could be arrhythmogenic. Viral infection has previously been shown to precede POTS (postural orthostatic tachycardia syndrome) [94] and, with the ACE2 receptor expressed on neurons, viral infection by SARS-CoV-2 may have direct negative consequences on the autonomic nervous system [95]. A complex combination of infection, an autonomic nervous system induced pro-inflammatory response, and a level of autoimmunity may all contribute to the establishment of autonomic dysfunction and POTS [96].

3.5. Possible mechanisms for cognitive dysfunction

Studies have explored cognitive function and deficits in patients with COVID-19 and suggest that the virus can cause septic encephalopathy, non-immunological effects such as hypotension, hypoxia, and vascular thrombosis, and immunological effects such as adaptive autoimmunity, microglial activation, and a maladaptive cytokine profile [97]. Additionally, patients admitted to hospital with COVID-19 have presented with a range of complaints including encephalopathy, cognitive impairment, cerebrovascular events/disease, seizures, hypoxic brain injuries, corticospinal tract signs, dysexecutive syndrome, an altered mental status, and psychiatric conditions. [98,99] These findings reveal that neurological symptoms associated with COVID-19 are common, diverse, and could pose substantial problems for rehabilitation and ongoing care following recovery from COVID-19. It is unknown who is most affected by cognitive complaints induced by COVID-19 and how long they persist; however, patient experiences and published summaries of long COVID have described “brain fog” to be a common and debilitating symptom [100-102].

Coronaviruses including SARS-CoV-2 can infect the central nervous system (CNS) via hematogenous or neuronal retrograde neuro-invasive routes [103]. The entry mechanism and subsequent CNS infection may explain the high incidence of neuro-inflammation seen in patients with COVID-19, and may result in damaging long term effects, with associations of viral infections and chronic neuro-inflammation with neurodegenerative and psychiatric disorders already elucidated [104,105]. SARS-CoV-2 may also affect the permeability of the blood-brain barrier, which would enable peripheral cytokines and other blood derived substances to enter the CNS and further drive neuro-inflammation.[106] Thrombo-inflammatory pathways may be the cause of the increased prevalence of stroke in COVID-19, [107] while “brain fog” may evolve from PTSD or deconditioning following critical illness and invasive treatment.[108] Evidence suggests that a direct viral encephalitis, systemic inflammation, peripheral organ dysfunction, and cerebrovascular changes may contribute to the development of long term sequalae following COVID-19 [109].

3.6. Possible mechanisms for Olfactory and gustatory dysfunction

Abnormalities of smell and taste have been reported to persist following recovery from COVID-19. Non-neuronal expression of the ACE2 receptor may enable entry of the SARS-CoV-2 virus into olfactory support cells, stem cells, and perivascular cells. This local infection could cause an inflammatory response which subsequently reduces the function of olfactory sensory neurons. Additionally, by damaging the support cells responsible for local water and ionic balance, SARS-CoV-2 may indirectly reduce signaling from sensory neurons to the brain, [110] resulting in a loss of sense of smell.

ACE2 receptors are also expressed on the mucous membrane of the oral cavity, particularly on the tongue, [111] therefore SARS-CoV-2 has a direct route of entry into oral tissue, which may result in cellular injury and dysfunction. Moreover, SARS-CoV-2 may bind to sialic acid receptors,[112] causing an increase in gustatory threshold and resulting in degradation of gustatory particles before they can be detected [113]. Another possible mechanism of gustatory

dysfunction in COVID-19 and long COVID concerns the functional link between taste and smell, whereby gustatory perception is reduced because of antecedent olfactory sensory dysfunction [114].

3.7. Other commonly reported long COVID manifestations:

COVID-19 infection can result in multi-organ impairment in individuals with low or high risk for severe acute disease [115]. Studies show the presence of acute kidney injury in discharged patients who have recovered from COVID-19 [116-118]. Although the long-term effects of COVID-19 on the kidneys are not fully elucidated, a study assessing kidney function in patients with COVID-19 found that 35% had decreased kidney function at 6 months post-discharge [119]. Acutely, pancreatitis triggered by SARS-CoV-2 has been seen in people with COVID-19, while serum amylase and lipase levels have been observed to be higher in people with severe illness compared with mild cases, and computed tomography images have shown pancreatic injury [120]. Kidney injury may occur through several mechanisms associated with COVID-19, including sepsis [121] and lung injury leading to hemodynamic changes and hypoxemia [122]. The ACE2 receptor is highly expressed in the pancreas, [123] perhaps to a greater level than in the lungs [120]; however, it is unclear whether pancreatic damage is a direct result of viral infection within the pancreas, or caused by the systemic inflammatory response seen during COVID-19 [124]. The spleen also expresses ACE2 receptors [119] and may be directly attacked by the virus, rather than the intense systemic inflammation being the primary cause of splenic damage [125]. Chronic systemic inflammation is frequently observed long after the clearance of acute COVID-19 infection, therefore, it is likely that this elevated inflammatory state causes long term complications in multiple organs in people with long COVID.

Lastly, we recommend to assess inflammatory markers assessment in all cases suspected with any respiratory and systemic symptoms during follow up in post covid care setting especially those having any systemic long covid manifestations [126-138]. 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 long covid symptoms and post covid sequel in selected symptomatic cases of recovered COVID-19 cases [130-144].

4. 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. Vaccination will prevent long COVID in majority and decrease severity of illness in survivors.

Compliance with ethical standards

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Venkatesh Hospital and Critical care center Latur Maharashtra India.

Disclosure of conflict of interest

No conflict of interest.

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