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Post Covid Complications: Aftermath of the covid pandemic on health.

Post Covid Complications: Aftermath of the covid pandemic on health.

Anwesha Bhattacharjee, Pathoja Maity, Souradipto Choudhuri, Suvroma Gupta*

[1]Department of Biotechnology, Haldia Institute of Technology, West Bengal, India

*Corresponding Author's Email: suvroma.gupta@gmail.com

ABSTRACT

The most recent pandemic human race has faced is the covid pandemic. It is caused by the Severe Acute Respiratory Syndrome Coronavirus 2 or better known as *SARS-CoV-2* virus. The death toll of the pandemic is estimated to be 6,636,721 as on November 28, 2022. The pandemic, originally started at the beginning of 2020, has left its mark on various aspects of our life. SARS-CoV-2 also had undergone several mutations during the period, making it more challenging to find a cure. Though some vaccines have been discovered during the time and are currently in use, no cure so far has been developed. This article sheds light on the mutation of the virus and emphasises on several post covid complications reported after the pandemic.

Keywords: COVID-19, SARS-COV-2, Complications.

RESUMEN

La pandemia más reciente a la que se ha enfrentado la raza humana es la pandemia del covid. Es causada por el Síndrome Respiratorio Agudo Severo Coronavirus 2 o mejor conocido como virus SARS-CoV-2. El número de muertos por la pandemia se estima en 6.636.721 al 28 de noviembre de 2022. La pandemia, que comenzó originalmente a principios de 2020, ha dejado su huella en varios aspectos de nuestra vida. El SARS-CoV-2 también había sufrido varias mutaciones durante el período, lo que hacía más difícil encontrar una cura. Aunque se han descubierto algunas vacunas durante el tiempo y están actualmente en uso, hasta ahora no se ha desarrollado ninguna cura. Este artículo arroja luz sobre la mutación del virus y enfatiza varias complicaciones post covid reportadas después de la pandemia.

Palabras clave: COVID-19, SARS-COV-2, Complicaciones.

INTRODUCTION

There have been many pandemics in the past. But the most recent pandemics humans have faced is the covid pandemic. Starting at a wholesale market of Wuhan province in china (Yang et al., 2020), the disease spreaded around the globe in no time. On February 11th, 2020, WHO declared it as a pandemic. Several researchers started their work for finding a cure or a vaccine to the mentioned disease. Several vaccines have been developed by several

researchers and many of them are currently in use (Bhattacharjee et al., 2022). Though during the pandemic, frequent mutation of the virus pose a significant hindrance to the development of vaccines. Also after the pandemic, several other health issues were reported as discussed below.

1. Mutations

There have been several mutations in the spike proteins of SARS-CoV-2. The mutations led to delta and omicron variants. The detailed discussion about the mutation can be found below.

1.1. Alpha Variant (B.1.1.7)

The first variant of concern i.e. the alpha strain first emerged in the United Kingdom in the month of September, 2020. This particular variant can cause around 50% more transmission than earlier strains of Covid that includes increasing disease severity and has spread in almost 192 locations worldwide. This strain was the major driving force of causing a third wave in Canada, during the first months of 2021. There are certain key mutations found in the spike protein that enables viral entry into the human cells, which also marks the strain distinctively from the original Wuhan strain. The important mutations that are found are N501Y mutation - This specific mutation makes the virus more infectious , where the binding of the spike protein to the cellular receptors is ameliorated. D614G mutation - This another type of mutation is also found to significantly improve viral replication. Though there is another mutation i.e. P681H mutation which has also shown voluntary functions in transmission but its functions are still under research. According to the WHO, another distinct mutation of alpha variant has been found i.e. the E484K mutation which helps the virus to slither away from the body's immune cells which results in eluding from the neutralising antibodies earlier generated in the body by previous events (Duong, 2021; From Alpha to Omicron: Everything You Need to Know about SARS-CoV-2 Variants of Concern, 2021).

1.2. Beta Variant (B.1.35)

The next more dreadful variant i.e. the Beta variant of SARS-CoV-2 was first detected in South Africa and was termed as a variant of concern in December, 2020. The infectivity of this variant eventually led to a surge in the number of hospitalisations and deaths. There are 21 types of mutation found in Beta variant, out of which 9 spike protein mutations are more prevalent. Apart from mutation in N501Y, the key mutations found are E484K, K417N that aids in evading the virus from the neutralising effect of the antibodies, orf1b deletion in the receptor binding domain (RBD) and L18F, D80A, D215G, Δ242–244, R264I, A701V found in the N terminal domain. This variant has been considered to be 50% more transmissible than the previous variants. The main concern has become the reduced neutralising effect of the antibodies on this variant which will result in re-infecting in the body which has already recovered from the covid-19 (From Alpha to Omicron: Everything You Need to Know about SARS-CoV-2 Variants of Concern, 2021; Yadav et al., 2022).

1.3. Gamma Variant (P.1)

The designated variant of concern was first detected in Brazil in November, 2020 which in January, 2021 started to spread rapidly and became the principal lineage associated with the second wave of infection. This strain has been found to be 1.7-2.4 times more infectious than the local variants present in the country. There are 21

lineage defining mutations found in this variant, out of which 10 spike protein mutations are considered to be predominant. In these predominant mutations found, there are three types of mutations present namely, K417T, D614G, E484K and N501Y found in the RBD region which is eventually related to surge in transmission, a higher viral load and susceptibility for immune evasion, and getting reinfected with SARS-CoV-2 (Duong, 2021; From Alpha to Omicron: Everything You Need to Know about SARS-CoV-2 Variants of Concern, 2021; Nonaka et al., 2021).

1.4. Delta Variant (B.1.617.2)

The distinctly transmissible Delta Variant was first detected in India in May 2021 which later has been detected in around 176 locations worldwide. This specific variant is found to induce 60% more transmission than the alpha variant and has become the dominant variant by overtaking other variants in many countries. Including mutation in D614G, there are certain additional mutations found in this particular variant but are not found in others such as, L452R mutation- induces surge in transmissibility as well as passes the virus from getting destructed by the immune defence, T478K mutation- Helps in the viral entry by escaping immune system recognition of the body, P681R mutation- Amplifies the ability to activate the severe ailments present in the body. There have been another emergent variant found namely, delta plus variant first identified in Nepal, which has been assessed to carry an additional mutation i.e. K417N plays a significant role in viral transmission (Duong, 2021; From Alpha to Omicron: Everything You Need to Know about SARS-CoV-2 Variants of Concern, 2021).

1.5. Omicron Variant (B.1.1.529)

This rapid spread of this variant was detected in November,2021 in numerous countries. There are a large number of mutations found in Omicron variants including certain mutations of concern like- N501Y, D614G, K417N and T478K mutations, though these mentioned mutations are common in other variants and some other mutations have also been found but are still under research (From Alpha to Omicron: Everything You Need to Know about SARS-CoV-2 Variants of Concern, 2021).

1.6. Spike Protein Mutations

As we know, starting from the first wave there were a lot of changes in the structure of the spike protein of SARS-CoV-2. The mutations in the spike protein led to the development of different variants like delta variant and omicron. It was found that there was a certain acceleration in the composition of amino acids as compared to the delta variant like: arginine, lysine, glutamic acid, aspartic acid. So the sites of mutation in the spike protein of delta variant are T19R, G142D, Δ156-157, R158G, Δ213-214, L452R, T478K, D614G, P681R, D950N which made it more deadlier (Kumar et al., 2021).

2. Impact of COVID-19

2.1. Impact of COVID-19 on fertility rate

The biases in the severity of COVID-19 has shown to be more on gender rather than just age. According to the meta-analysis conducted worldwide over a huge proportion of the population after being transmitted with the disease, the odds of males being affected resulting in critical conditions and greater mortality rates were more in comparison to the females being affected.

TMPRSS2 gene expression plays a crucial role in the infectiveness capacity of COVID-19. In an in-vitro study based on females, the hormone 17β-estradiol primarily produced by the ovaries during the life cycle of reproduction impacts by alleviating the expression of TMPRSS2 gene and therefore reducing the infectivity of SARS-CoV-2 (Zarei et al., 2021). Also according to research studies, curtailed gene expression of ACE2 and TMPRSS2 seen in endometrium leading to lower susceptibility to SARS-CoV-2 infection.

In case of males infected severely, the presence of SARS-CoV-2 in the testis has resulted in causing damage to inflammation induced blood testis barrier (Markiewicz-Gospodarek et al., 2021). The level of testosterone is found to be lower in proportion to the elevated level of Luteinizing hormone in infected males especially such substantial alteration of these levels has been noticed in severe cases of COVID-19 infection (Collins et al., 2022). In the meta-data based studies, the critical and fatality rates of men are greater in number to those of women. The number of cases of premenopausal women getting affected with SARS-CoV-2 is even though more but not highly fatal in comparison to the cases of men getting affected. Although, in many cases after recovery the levels of sex hormones were seen to be normal in comparison to alteration of levels that occurred during the onset of infection (Zarei et al., 2021). Due to the presence of vast data globally for the widespread of COVID-19, it is challenging to understand the relationship of COVID-19 and human fertility but investigations are currently going on to reach a legitimate conclusion (Collins et al., 2022).

2.2. Impact of COVID-19 on endocrine system

The presence of the ACE2 receptor and TMPRSS2 is known to play a significant role towards SARS-CoV-2 in gaining cellular access. Therefore,in humans ACE2 and TMPRSS2 mRNA are expressed in some endocrine glands such as thyroid gland, pancreas, testis, ovaries.

The retrospective studies have revealed that patients infected with COVID-19 exhibited reduction in thyroid levels. Patients who developed pneumonia from the infection of COVID-19 exhibited lower levels of TSH and total T3 level but no notable alterations occurred in the total T4 level. Evenmore, some results of patients who passed away due to severe SARS-CoV-2 infection showed excruciating destruction of thyroid glands. Although patients who recovered from COVID-19 were observed to have no differences in the thyroid hormone levels after some time. According to the current evidence, there are various factors stimulated due to COVID-19 infection that might be responsible for causing thyroid dysfunction but long term research is still lacking to reach a conclusive deduction (Zarei et al., 2021; Clarke et al., 2021).

The onset of SARS-CoV-2 infection in the human body has consequently influenced the extremity of diabetes mellitus. Patients with no history of diabetes have been observed to develop hyperglycemia after COVID-19 infection. Even several cases of ketosis, sudden emergence of hyperglycemia and detection of diabetes eventuated where specifically infected COVID-19 patients diagnosed with type-1 and type-2 diabetes mellitus were observed with higher risk of mortality. The intensity of abrupt diagnosis of diabetes after onset of COVID-19 poses vulnerable threats to human health and thus current investigations have been undertaken to assess the complex relationship of diabetes and COVID-19. Expression of ACE2 and TMPRSS2 gene present in vasculature of pancreas causes SARS-CoV-2 cell entry mediation. According to research studies, huge proportion of patients with Type 2 diabetes as well as infected with COVID-19 showed a higher tendency of getting detected with ketoacidosis which is a condition occurring when the secretion of pancreatic insulin is insufficient in order to meet the glycemic needs although ketoacidosis is normally seen to occur in case of Type 1 diabetes. Major proportion of patients admitted with severe SARS-CoV-2 infection as well as diagnosed hyperglycemia retrieved to euglycemic condition after disease recovery and proper medical care and other proportion dealt with persistent hyperglycemic levels. Therefore, Type 2 diabetes induced elderly people pose a higher risk of getting infected with SARS-CoV-2 associated with hyperglycemia and ketoacidosis and also to patients who haven't been treated previously with insulin. The occurrence of hyperglycemia with or without prior diabetes can cause an elevation in mortality rates as it impairs immune response which can occur by reducing the activity of macrophages and polymorphonuclear leukocytes thus impacts on excessive cytokine response and produces a vigorous pro-inflammatory effect. Due to elevated levels of blood sugar, pancreatic tissues may get damaged in the body (Clarke et al., 2021; Abramczyk et al., 2022). This link between COVID-19 and diabetes needs further investigation for clinical relevance and also long term follow up from recovered patients is required.

2.3. Impact of COVID-19 on nervous system

A multitude of neurological complications have been recorded in patients after transmission of SARS-CoV-2 in the body. This pathogen is known mainly for adversely affecting the respiratory system but some neurological conditions such as confusion, stroke, neuromuscular disorders, neuro-ophthalmological disorders, neurosensory hearing loss and dysautonomia are prevalently manifested during and after infection. During long Covid, illnesses are persistent such as impaired concentration, headache, sensory disturbances, depression, and even psychosis for a long period of months and patients affected also include the younger generation. The incomprehensive mechanisms induce immune dysfunctions which consist of neuroinflammation and antineural autoimmune dysregulation, coagulopathy, neuronal injury and also causes viral entry into the central nervous system. Assessing the neural manifestations such as neuroinflammation and neuronal injury indicates aggravating or triggering further deterioration in neurodegenerative diseases such as parkinson's diseases or Alzheimer's (Spudich & Nath, 2022). Thus, it is clear through observations that severity of COVID-19 induces a direct effect on consequent neurological diagnosis and also increases the perils related to cerebrovascular events such as ischaemic stroke and intracranial haemorrhage. The emergence of complex mechanisms related to causing neurological conditions after transmission of SARS-CoV-

2 persistently affecting nervous system become a part of long covid symptoms leading to major reason of public health concerns (Zarei et al., 2021).

2.4. Impact of COVID-19 on mental health

An apprehensive issue has been raised regarding mental health concerns since the emergence of COVID-19. The outbreak and spread of any contagious diseases globally are shaped based on the psychological orientation and response of the population towards it as well as alter the intensity of psychological distress during and after the outbreak. Incalculable cases of major mental health complications as well as psychological distresses such as anxiety, depression and post traumatic stress disorder have surfaced in this period which has become alarming and are amplifying significantly. Intimidating manifestations of suicidal thoughts and behaviours including health workers are aggravating extensively posing multitude public health concerns (WHO, 2022; Cullen et al., 2020). Widespread of the pandemic led to the major psychological reactions including maladaptive behaviours, emotional distress and defensive responses. The vulnerability of the mental disorders increases in patients prone to psychological problems. Accordingly, the susceptibility of getting severely infected with COVID-19 increases in patients with pre-existing mental issues and substance use disorders stimulating various risks associated with testing and treatment as well as induces negative impact physically and psychologically derived from pandemic. Therefore, psychological components play a salient role in association with public health concerns and also annotates the different coping mechanisms of people towards the menaces related to pandemic and also countless losses associated with it (Cullen et al., 2020).

2.5. Impact of COVID-19 on cancer patients

Manifestation of complications derived from transmission of COVID-19 has been observed in patients with comorbidities including cancer patients. The severity of cases is seen to be more in patients with pre-existing malignancy in contrast to others with no malignancy. Some other factors including age, gender, smoking status, cancer status also stimulates the risk factors of malignant patients after transmission of SARS-CoV-2 infection (Sha et al., 2020). The prevalence of infection has been observed to be more in patients with haematologic malignancies rather than in patients with solid tumours. Based on investigations, the susceptibility of COVID-19 has been seen to be more in patients with prostate cancer and lesser in case of lung, urothelial, bone cancer. The fatality rates occurring in patients differed on the basis of cancer type where thyroid cancer, acute leukaemia, male genital and breast cancer have led to be in association with the highest mortality rate from COVID-19 infection (Fillmore et al., 2020). Although, the prognosis and treatment of lung cancer patients severely infected with COVID-19 has been thoroughly challenging. Therefore, the risk factor and mortality rate is more in cancer patients rather than the general population and such patients also suffer unbearable prognosis. Evenmore, the psychological status of cancer patients more notably in women patients were also shattered due to rapid transmission of COVID-19 infection, greater fatality rates in subpopulation, insufficient antineoplastic treatments on regular basis as well as quarantine measures leading to occurrence of various mental health issues (Sha et al., 2020). Thus, cancer patients with Covid

positive condition showed more tendency of hospitalisation, ICU admissions, and respiratory support after transmission of SARS-CoV-2 in comparison to the general population (Fillmore et al., 2020).

2.6. Impact of COVID-19 on Mucormycosis

A vast range of microbial infections i.e. opportunistic bacterial and fungal infections have been observed to play a noteworthy role in patients after onset of COVID-19 infections. Worldwide, multitude cases of co-infection of mucormycosis in patients with already transmitted COVID-19 have been recorded (Singh et al., 2021). To reduce the mortality rates in critical patients, comprehensive corticosteroid treatments have been administered in patients which induced a fatalistic effect on immunological as well as physiological factors leading to causing of consequent fungal diseases. An exemplary environment on the basis of low oxygen level (hypoxia), acidic medium (metabolic acidosis, diabetic ketoacidosis [DKA]), high iron levels (ferritins), high glucose levels (diabetes, new-onset hyperglycemia, steroid induced), decreased function of White blood cells in phagocytic activity resulted from immunosuppression(mediated by SARS-CoV-2 or steroids or any comorbidities) facilitated the impregnation of mucorales spores in infected COVID-19 patients coupled with several other factors such as hospitalisation or mechanical ventilator support for a prolonged period (Singh et al., 2021). Mucormycosis is an uncommon, fatal fungal infection caused by various genera and species mainly known as angioinvasive disease injuring victims with compromised immunity. It may involve and affect different parts of the body depending on the underlying conditions (Singh et al., 2021; Hoenigl et al., 2022). Some vital conditions related to the treatment of mucormycosis involve keeping underlying maladies in check, surgical operation of necrotic infected tissue and specialised antifungal therapy. The higher mortality rates due to mucormycosis with SARS-CoV-2 infection even after meticulous therapy and treatment dispensed with lamenting results.

The currently present therapeutic actions are not sufficient for negating the fatality as well as mortality issues related to COVID-19 associated mucormycosis. The known antifungal drugs and treatments are resulting to be effective against Mucormycosis and therefore new antifungal therapeutics are under development although rapid and vigorous treatment mechanisms are difficult to administer in affected individuals with short term clinical studies (Hoenigl et al., 2022).

3. After Effects Of Covid On Several Organs

3.1. Effect on lungs

As we know, around 67 million people were infected with 1.5 million deaths during the COVID-19 outbreak at the end of November 2020. Hypertension and diabetes are some of the primary risk factors for COVID-19. Although even after recovering from COVID-19 many people still suffer from certain post-COVID complications starting from fatigue to severe Progressive fibrotic lung disease (Carfì et al., 2020). Pulmonary fibrosis causes scarring of lungs and has a detrimental effect on the lungs tissue. It was found that a cytokine storm might be a probable cause for causing an abnormal immune mechanism leading to pulmonary fibrosis (George et al., 2020). In a cytokine storm, numerous inflammatory cytokines are produced at a greater rate. As a result positive feedback occurs on other immune cells due to excessive cytokine production allowing more immune cells being recruited at the site of

injury leading to organ failure. As we know that ACE-2 is a key receptor for SARS-CoV-2 infections. So an injury in the lungs causes an activation and proliferation of type 2 alveolar epithelial cells (or AEC_2) in order to envelope the exposed surfaces of alveoli and thus activating the provisional matrix (Chambers, 2008). But in the normal repair mechanism the provisional matrix disperses very slowly and then the lungs once again repossess its normal structure and activity. Now when the Type 1 alveolar epithelial cells are damaged the AEC_2 undergoes apoptosis and then it gets differentiated into AEC_1 to reinstate the alveolar epithelium (Hadjicharalambous & Lindsay, 2020) ultimately causing abnormal tissue repair followed by collagen deposition, fibroblast activation etc. It was found that myofibroblasts and fibroblasts are the key cells responsible for idiopathic pulmonary fibrosis. So the EGF, PDGF, IL-1 and TGF-β help in differentiating and proliferating the fibroblasts into myofibroblasts which maintains the fibrotic process. Now the myofibroblasts produce IL-8, IL-6, IL-1 and monocyte chemo-attractive protein 1 which ultimately triggers the inflammatory response. So the ECM made by myofibroblasts is more in a disordered fashion as compared to that of fibroblasts. Enormous accumulation of ECM components like fibronectin, collagen etc causes thicker alveolar walls which ultimately hampers the gaseous exchange. Figure 1 gives a pictorial description of the process.

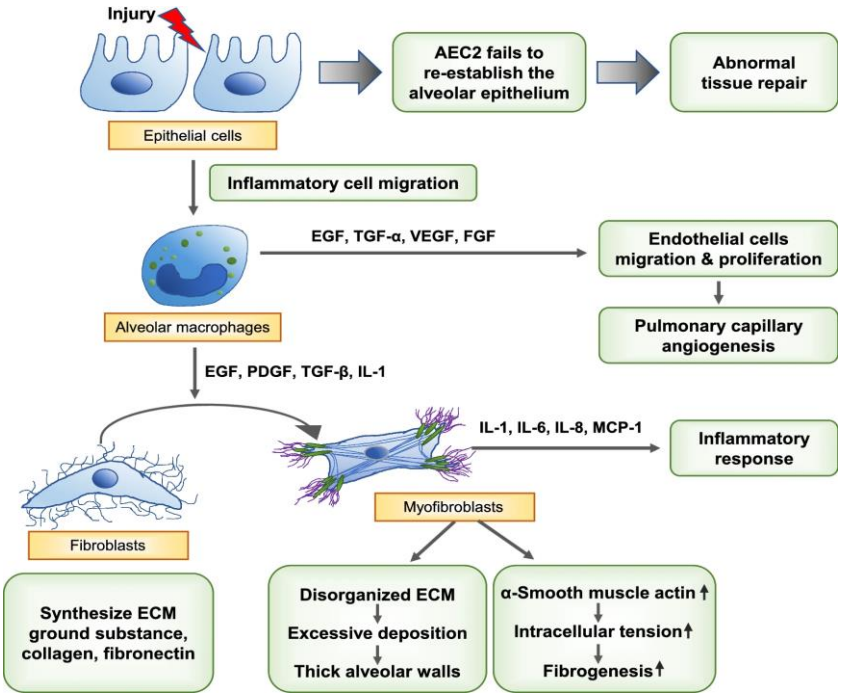

Figure 1. Summary of pulmonary fibrosis mechanisms

3.2. Effect on Heart

It was found that patients who were suffering from (SARS-CoV-2) infections were prone to several cardiovascular complications like Postural orthostatic tachycardia syndrome or (POTS). Patients suffering from POTS suffer from chest pain, palpitations, dyspnea etc. In POTS the heart rate increases more than 30 beats/min without significant hypertension. Normally the autonomic nervous system (ANS) controls the blood pressure and heart rate in our body. But the people who are suffering from POTS, their body is not able to maintain a steady and stable blood

pressure. Now there are different types of POTS like Hypovolemic POTS (this happens when the blood volume is reduced), Hyperadrenergic POTS (This occurs due to over activeness of the sympathetic nervous system), Neuropathic POTS (This happens when there is a loss of nerve supply). The ultimate cause behind POTS is still not known. But one such mechanism might be possible that the coronavirus provokes maximum production of antibodies that reacts with the G-protein coupled receptors, autonomic nerve fibres, autonomic ganglia and several cardiovascular receptors ultimately leading to the malfunctioning of the autonomic nervous system as mentioned by the researchers (Blitshteyn & Whitelaw, 2021; Shouman et al., 2021; Wallukat et al., 2021; Guilmot et al., 2020). Now by activating the muscarinic and adrenergic receptors the autoantibodies finally lead to venous pooling and malfunctioning of the PNS, dysregulation of autonomic nervous system and tachycardia ultimately causing POTS (Blitshteyn & Whitelaw, 2021) (Shouman et al., 2021; Wallukat et al., 2021; Guilmot et al., 2020; Ståhlberg et al., 2021). It was also found that several cardiovascular damage is occurring due to the dysregulation of renin-angiotensin-aldosterone system, hyperinflammation etc.

3.3. Effect on liver

As we know, for the intracellular penetration of SARS-CoV-2 virus the angiotensin-2 conversion enzyme (ACE-2) plays a very important role. Many tissues like heart, liver, lungs express ACE-2. So the liver acts as a potential target organ for SARS-CoV-2 virus. It was found that the destruction of the virus penetrating the ACE-2 pathway in liver tissue causes an immune reaction like liver lesion and fibrosis due to anti-COVID drug therapy. It was found that many patients suffered from Drug-induced liver injury (DILI) which was caused due to the administration of certain drugs during the treatment of COVID-19. It was a significant cause for the acute liver failure with a high mortality rate. According to some evidences, it was found that the combination of ritonavir and lopinavir overdose activated the stress pathway of the endoplasmic reticulum in the liver and through the caspase cascade system it induces hepatocyte apoptosis ultimately leading to severe liver damage . It was also found that the antiviral drug given during COVID-19 treatment was widely metabolised by hepatocytes through cytochrome P_{450} (CYP3A4 system), which is an inhibitor. So the excess production of toxic intermediate ritonavir metabolised by CPY3A4 leads to severe liver damage (Cao et al., 2010; Guevara et al., 1993). So a COVID-19 patient who is subjected to different medications are at a higher risk of liver damage due to the side effects of different drugs administered during the treatment.

3.4. Effect on kidney

COVID-19 has affected the kidney in several ways. Even the drugs that were administered during the treatment had a severe effect on kidney .It was found that the kidney transplant patients were much more prone to COVID-19 due to the usage of immunosuppressive medications, shortened perseverance of antibody titers following active infection, a pro-coagulated state caused due to chronic kidney disease (CKD) leading to urgent need for hospitalisation. There are lots of direct and indirect effects of coronavirus which might sustain even after the recovery from COVID-19 and could lead to chronic kidney disease(CKD), sepsis and acute kidney injury (AKI). There were several pathophysiological mechanisms like endothelial damage, tubular injury, podocyte injury etc experienced during the post COVID-19 period. Podocyte injury leads to the malfunctioning of the glomerulus. It

causes collapsing glomerulopathy which is a very common disease found among most of the COVID-19 patients (Wu et al., 2020). It occurs due to the upregulation of $APOL_1$ gene due to viral infection ultimately leading to the dysregulation of podocytes and glomeruli (Nichols et al., 2015; Friedman & Pollak, 2016).

3.5. Effect on eyes

Even after recovering from COVID-19 many patients are still suffering from several eye problems like conjunctivitis, retinal artery occlusion, retinal haemorrhage, retinal vein occlusion. According to some evidence, it was found that people who contracted COVID-19 disease experienced severe inflammation throughout their body ultimately causing blood clots to form. When these clots move through the veins and arteries it blocks the flow of oxygen ultimately causing the cells to die.

During eye stroke (or retinal artery occlusion) the blood clots blocks the flow of oxygen in the arteries of the retina ultimately leading to the death of the cells. Even sometimes when the blood clots prevents the nutrients from getting inside the retina as a result the retinal tissue begins to swell and ultimately causes death of the cells. This leads to 'Cotton Wool' spots in the eyes.

4. Conclusion

Covid pandemic has left the world shaken. Though researchers have found several vaccines to the disease, a cure is yet to be found. Covid is itself like a pneumonia fever and was first miscategorised as pneumonia. But as stated in the article, it has severe effects on human health. It has its effect on various systems and organs of the human body. Also the trend of several other diseases have gone upwards after the pandemic. Hence these can be categorised as the long term effect of covid pandemic. Adequate research in this field can strengthen the molecular interaction and disease interaction in the human body.

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