LONG-TERM IMMUNOLOGICAL CONSEQUENCES OF COVID-19 ON HEALTH

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ABSTRACT
SARS-CoV-2 [severe acute respiratory syndrome coronavirus] that initially came to notice in December 2019 is the agent responsible for COVID-19 is still spreading rapidly worldwide and it is presently a potent danger to the world and also to the economy. Patients with COVID-19 are still at risk of Acute Respiratory Distress Syndrome (ARDS), respiratory failure, and death. Those patients whose aged more than sixty years with comorbidities, children, and healthcare workers are highly vulnerable to this virus patient shows various symptoms most commonly cough, fever, difficulty in breathing, fatigue, sore throat. The infection could be categorized into three stages: mild infection, the pulmonary stage, and the inflammatory stage. As the COVID-19 pandemic continues, it has been clear that infection caused due to SARS-CoV-2 might be responsible for the unpredicted long-term health consequences. In addition to this, it has acute respiratory manifestations, adversely SARS-CoV-2 also affects the other organ systems. However, there is limited to the management of COVID-19 related conditions of the extrapulmonary systems. After recovery, patients remain at risk for lung disease, heart disease, and mental ailment. There may be long-term consequences of adverse effects they observed in the course of COVID-19 and during its treatment. This review provided information about the extrapulmonary manifestations of COVID-19 that may impair the urinary, cardiovascular, gastrointestinal, hematological, hematopoietic, neurological, or reproductive systems. Also, the main purpose of this article is to describe the current concern of the extra pulmonary complications that were caused due to COVID-19 and also to improve the management and diagnosis of these patients.

Keywords: COVID-19, Treatment, Extra-pulmonary manifestation, Management, SARS-CoV-2

INTRODUCTION
The spread of coronavirus disease 2019 (COVID-19) was mainly caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) lately, it has become a global pandemic and public health problem in all countries [1-3]. Coronavirus infections are responsible for severe and life-threatening acute respiratory syndromes in humans. As per the report of 16 June 2020, more than 7900000 confirmed cases come out and approximately 434796 total deaths for COVID-19 have been reported globally. The evolution of vaccines is an early stage, also the increasing number of infected patients is rapidly worldwide. In addition to the common acute respiratory symptoms (such as fever, cough, and dyspnea), also COVID-19 patients have shown signs and symptoms of injury to other organ systems, which may make difficult medical management. COVID-19 affects the upper airways (sinuses, nose, throat) and lower airways (windpipe, lungs). The virus enters into host cells through the enzyme angiotensin-converting enzyme 2 (ACE2) due to this lungs are the most affected organ by COVID-19, which is most prone in type-II alveolar cells of the lungs [4]. The virus makes use of a particular surface glycoprotein called a “spike” (peplomer) that connects to ACE2 and enters into the host cell [4]. The density of ACE2 in each tissue match up with the seriousness of the disease in that tissue and some have suggested decreasing ACE2 activity might be protective, [5] though another view is that increasing ACE2 using angiotensin II receptor blocker medications could be protective [6]. As the alveolar disease progression, respiratory failure might evolve and death may occur [7].

SARS-CoV-2 and the immune system
SARS-CoV-2 belonging to the coronavirus family caused two epidemics at the beginning of the 21st century; one was named SARS-CoV-2, and the second is Middle East Respiratory Syndrome (MERS).

Coronavirus is large enveloped viruses with a positive-sense RNA genome. The spike (S) has two S1 and S2 domains, which are responsible for invasion, attachment, and entry into human cells. The receptor-binding domain in S1 interconnects with angiotensin-converting enzyme 2 on the human host cell surface, which is a similarity entry mechanism to SARS-CoV; however, the S2 domain is responsible for virus-cell membrane fusion and viral entry with higher affinity [8]. Higher manner of the ACE2 receptor in adults as compared to children may be the reason for higher infection in adults as compared to children [9, 10]. As increased the extent of enzymes in the liver, heart, and kidneys of COVID-19 patients with pneumonia; this could also responsible for the multiple organ failure in some of the patients [11]. Therefore, these organs constitute the main target for the virus.

Fig. 1: Structure of coronavirus (self-modified)

Symptoms
Large number of patients suffers from common cold and flu when infected with this virus, although some maybe asymptomatic. About 80% of patient will show mild symptoms are shown in about 80% of the patient with this. It is observed that adults having greatest immunity to fight against this infection but the disadvantage is they rapidly spread the infection. In 99% of patients shows fever with seriously high temperature, while remaining patients will
experience fatigue, dry cough. In One-third of patient dry cough, difficulty in breathing observed. From day 1 to day 17 various symptoms of COVID-19 given below they goes from bad to worse.

Day 1: In the first day fever together with, muscle pain, dry cough, fatigue are observed. In some patients nausea and diarrhea observed.

Day 5: Few Patients experienced difficulty in breathing particularly in elderly patients having earlier health condition.

Day 7: On the day 7th patients need to be admitted in the hospital.

Day 8: Patients develop ARDS is develop by patients in this the fluid fills up into lungs and this is mostly fatal. Usually happens in severe cases.

Day 10: Worsening of the symptom if the disease is in progression and at this movement the patient is shifted to ICU. Percent of death is very less.

Day 17: After two-and-a-half weeks patients who recover are discharged from hospital. Although, it is strenuous to find out symptoms in early stages of infection. Commonly observed after 5-6 d.

**Covid-19 death rate due to medical conditions**

Information made by Centers for Disease Control and Prevention (CDC) and other studies shows that death rate is more in old age people and the people who do not having the good medical conditions. People with serious illness, such as diabetes, heart, lungs disease, have a greater risk. Death rate is 1% of those who have no history of medical conditions. Death rate is 10.5% for those who suffer from cardiovascular diseases, death rate is 7.3% for those who suffer from diabetes, death rate is 6.3% for people who suffer from Chronic respiratory disease (such as asthma and chronic obstructive pulmonary disease), death rate is 6.0% for people suffering from hypertension and death rate is 5.6% for people suffering from cancer.

**Treatments**

Allopathic treatment and management comprise oxygen therapy, drug treatment, and intravenous fluid infusion using life support in hazardous cases. Coronavirus may demonstrate comparable proteins for virus reproduction in the human immunodeficiency virus (HIV). Therefore, HIV protease inhibitors and nucleoside derivatives may be efficacious to treat COVID-19 like combination therapy of lopinavir. A minute ago, an Italian patient of COVID-19 was treated by making a combination of lopinavir (200 mg) and ritonavir (50 mg) twice a daytime, in Sawai Man Singh (SMS) Hospital, Jaipur India. Equally well, the patient has also presented a combination of oseltamivir and chloroquine drug. The patient was recovered and the test was found negative for COVID-19. Some drugs are in clinical trial and outcomes are still expected. The best methodology to fight with viruses is vaccination. Consequently, scientists are trying to cultivate a vaccine for this virus and undoubtedly may be available later some time. Existing clinical indication does not support preventing angiotensin-converting enzyme inhibitors or angiotensin receptor blockers in patients with COVID-19 [12, 13].

**Anti-viral drugs**

Drugs that come under this category typically follow either of the next three mechanisms the ion channel blocking agent, virus-viral replication inhibition, and serine protease inhibition. In the market existing antiviral drugs typically target the four primary groups of viruses: herpes, hepatitis, human immunodeficiency virus (HIV), and flu. Previous outbreak incidents of viral infections like SARS-CoV and MERS-CoV as well as hemorrhagic fever viruses like Ebola were managed with this category of drugs only [14, 15].

**Anti-malarial drugs**

It comes under three groups grounded on their antifolate compound, mode of action aryl amino-alcohol compound, and artemisinin. Maximum of these drugs are eradicated progressively from the body enduring for long periods after consumption. The disadvantage of these drugs is that antimalarial drug resistance cultivates for any drugs below this category [16, 17].

**Anti-HIV drugs**

These drugs are categorized into diverse groups grounded on their targets retro-transcription, reverse transcription, viral-cell fusion, proteolytic processing, and combination of proviral DNA into the host genome and co-receptors interactions. Drugs that come under these groups have been permitted by the FDA (Food and Drug Administration) and are nowadays publicly used for the treatment of HIV.

**Anti-inflammatory drugs**

Enormous inflammatory response is detected in COVID-19. Anti-inflammatory drugs, particularly JAK-STAT inhibitors are used in contradiction of rheumatoid arthritis, may be operational against raising levels of cytokines, and valuable in preventing viral infection. Rendering to the latest study, an inflammatory drug, baricitinib is when applied in combination with means of anti-viral drugs like Remdesivir, it increases the effectiveness of the drug to decrease viral infection [18, 19].

**Monoclonal antibodies**

The virus is known to occur in the host cells by binding the S protein to ACE2 receptors. By emerging neutralizing antibodies in contradiction of the sense organs, there is a high probability of decreasing the harshness of the disease. Presently, only a few of drugs have been sanctioned for usage against SARS-CoV-2 [20].

**Other clinically used drugs**

Even earlier the announcement of COVID-19 as a pandemic by WHO, there was an enormous lack of disease precise drugs. Due to promptly increasing virus, it is important to make available appropriate treatment for the affected patients [21].

**Ribavirin**

Ribavirin is one of the broad-spectrum drugs whose therapeutic potential was revealed in 1972. This antiviral drug is used to treat hepatitis C. It is typically used with interferon α (IFN). This medicine is, approved by the USFDA, it plays for the active site of RdRp [22].

**Sofosbuvir**

This drug is also FDA approved drug in contradiction of NS5B and target as a nucleotide polymerase inhibitor applied for the treatment of hepatitis C. It was applied in combination with interferon or RBV. This drug was earlier applied for the treatment of the Zika virus [23].

**Lopinavir/Ritonavir**

Lopinavir/Ritonavir is a protease inhibitor, which objects the HIV. It was acknowledged by 1998 and approved by the USFDA by 2000. This drug blocks the formation of viral proteins by disturbing the proteolytic treatment by mimicking its structure as a peptide cleaved by HIV protease. Ritonavir along with another flu drug, like oseltamivir was described to result in the whole recovery after displaying signs of COVID-19 associated pneumonia [24].

**Remdesivir (anti-viral peptide)**

This specific drug is an adenosine nucleotide analog, which was applied to treat in contradiction of SARS-CoV, MERS-CoV, and Ebola [94]. It is a favorable and potential drug that roots premature cessation by entering the nascent viral RNA. Currently, it is undertaking clinical trials for Ebola treatment. An additional, recent study has to show that Remdesivir scored 0.77 μM at half-maximal concentration beside COVID-19 and obstructed viral infection [25].

**Chloroquine**

This drug, which comes under an anti-malarial drug, has exhibited potential in the treatment of avian influenza A. Chloroquine also has revealed the potential to possess anti-viral and immune-modulating properties. Chloroquine showed 1.13 μM at the half-maximal concentration in contradiction of SARS-CoV-2 and obstructed viral infection with an increase in endosomal pH mandatory for viral fusion [26].
**Vitamin D**

The vitamin D supplementation for treating of COVID-19 is used to boost your immune function. The vitamin D deficiency affects the respiratory immune function that increases the chances of COVID-19 could potentially protect against COVID-19 infection. Thus improvement in immunity through better nutrition is good factor. Thus the nutrients used as vitamin D they shows a significant role in immune function.

**Table 1: Approved allopathy drugs used for the treatment of the COVID-19**

| S. No. | Drug name                  | Disease treated                  | References |
|-------|-----------------------------|----------------------------------|------------|
| 1.    | Remdesivir                 | MERS, SARS COVID-19              | [68]       |
| 2.    | Flavivir                    | COVID-19                         | [69]       |
| 3.    | Lopinavir                   | MERS, SARS COVID-19              | [70]       |
| 4.    | Influenza drug              | MERS                             | [71]       |
| 5.    | Acyclovir                   | MERS, SARS and COVID-19          | [72]       |
| 6.    | Reverse transcriptase inhibitor | SARS                      | [72]       |
| 7.    | Tocilizumab                 | COVID-19                         | [72]       |
| 8.    | Oseltamivir                 | COVID-19                         | [72]       |
| 9.    | Azadivine                   | COVID-19                         | [72]       |
| 10.   | Carfilzomib                 | COVID-19                         | [72]       |
| 11.   | Indinavir                   | COVID-19 and SARS               | [73]       |
| 12.   | Baricitinib                 | COVID-19                         | [74]       |
| 13.   | Chloroquine                 | Coronavirus OC 43 SARS           | [75, 76]   |
| 14.   | Alcohol Inhalation therapy  | COVID-19                         | [77]       |
| 15.   | Ritonavir+Lopinavir         | MERS, SARS                       | [72]       |
| 16.   | RNA dependent RNA Polymerase | Murine Coronavirus, SARS | [78]       |
| 17.   | Papain like protease       | MERS and SARS                    | [79]       |
| 18.   | Fusion inhibitors i.e. Lamivudine adefovir dipivoxil etc. | SARS | [80]       |
| 19.   | Protease Inhibitors: saquinavir, ritonavir, indinavir, lopinavir, etc | SARS | [81]       |
| 20.   | Nucleotide reverse transcriptase inhibitors: tenofovir disoproxil fumarate | SARS | [82]       |

**Longterm immunological consequences**

**LUNGS**

One of the most common possible consequences of SARS-CoV-2 involved severe acute inflammation including scarring or fibrosis, which may be irreparable and lead to long-term health problems. In the lungs may cause pulmonary fibrosis, leaving patients breathless with a reduced lung capacity. In one study, 94% of hospitalized patients showed residual abnormalities on CT scans at the time of discharge following severe COVID-19. These studies were slight but indicate that it will be necessary to follow by COVID-19 survivors to determine the lung function abnormalities are persistent, and how they should be managed.

**Acute kidney injury and renal failure**

The kidneys are most commonly affected extrapulmonary organs in a patient infected with SARS-CoV-2 especially, in those patients who are critically ill [27-29]. SARS outbreak has revealed that kidney damage was mainly characterized by acute tubular necrosis and increased serum creatinine and urea nitrogen concentrations [30, 31]. A recent study of 59 patients infected with SARS-CoV-2 showed mild proteinuria that was the ordinary kidney abnormality in these patients. In addition, just about 30% of these patients also had raised urea nitrogen levels and about 20% had raised serum creatinine levels [32].

Data from 138 COVID-19 patients from Zhongnan Hospital in Wuhan, China, showed that AKI occurred in 8.3% of patients was admitted to ICU vs 2% in non-ICU patients. In another study, the occurrence of AKI in SARS critically ill COVID-19 patients was as high as 29%, and AKI was also found to be an important risk factor for increased hospital mortality [43].

In series Covid-19 cases, 85 patients with severe SARS-CoV-2 infection, AKI occurred in 23 (27.1%) patients [34]. According to this report the SARS-CoV-2 might directly infect kidney tubules. Although the primary virologic mechanisms are not completely

**Fig. 2: Extrapulmonary involvement of COVID-19 (Self-modified)**
known also it is possible to assume that there is binding between virus and ACE2 receptor, which highly appears in kidney tubules due to this causes glomerulopathy, acute tubular necrosis, and leakage of protein in the Bowman’s capsule [35, 36]. However, it was possible to assume that AKI could be an epiphenomenon of both respiratory disease of syndrome-induced hypoxia and septic shock caused by the SARS-CoV-2 [37]. Other autopsy investigations have been reported that the endothelium is affected in the kidneys, and is responsible for proteinuria [49]. SARS-CoV-2 particles in renal endothelial cells may suggest that viremia is the possible cause of renal endothelial damage that resulting in AKI [34]. More recently, Sun et al. [38] have reported the occurrence of subclinical AKI as reflected by elevated urinary levels of β2-microglobulin, α1-microglobulin, N-acetyl-β-D-glycosaminidase, and retinol-binding protein (i.e., all biomarkers of kidney tubular damage) in a sample of 32 confirmed COVID-19 cases without prior chronic kidney disease [50]. In addition, the severity of kidney tubular damage was also greater in severe COVID-19 patients than in less severely affected patients [45]. Based on the available evidence, we can conclude the following considerations: (a) AKI is not uncommon in patients with COVID-19, especially in those with severe COVID-19; patients can present with proteinuria early or at hospital admission, while AKI often develops in later stages of the viral disease (i.e., critically ill patients) and is observed as an early sign of multiple organ dysfunction; (b) AKI can be related to the virus-related complications like asphyxia and shock; (c) the precise incidence of AKI in SARS-CoV-2 infected patients is not known; however, it is reasonable to assume that AKI is more common in critically ill patients than in those with mild COVID-19 disease; and (d) COVID-19 patients have a prior history of chronic kidney disease are more probable to develop AKI, and (e) COVID-19 patients with AKI have a poorer chance of recovery.

Heart diseases

Myocardial injury

According to the study of SARS-CoV-2 infected patients, patients were quarantined at the Tongji Hospital, Wuhan, China between January to February 2020, including 24 patients who were critically ill and 126 patients who were severely ill. Chen et al. [27] reported that approximately 20% of these patients had exhibit signs of myocardial injury as showed by increases in plasma N-terminal pro-B-type natriuretic peptide (NT-proBNP) and cardiac troponin I (cTnI) levels [11]. Also, in a retrospective study of 52 critically ill COVID-19 patients, 15(29%) of these patients had shown elevated cTnI levels (i.e.,>28 pg/ml) [29]. There is an estimated 12% of COVID-19 patients without pre-existing or known ischemic heart disease had elevated troponin levels and also cardiac arrest during hospitalization [54]. Particularly, cTnI levels were shown to be above the 99% upper normal limit in 46% of non-survivors, as compared to 1% of survivors [30]. It is important that these markers were elevated in patients with severe COVID-19. Further investigations are needed to determine if this is a marker of disease severity or a marker of disease outcome. In addition, elevated troponin levels together with non-inflamatory markers such as interleukin-6, lactate dehydrogenase (LDH), and D-dimer, might be indicative of cytokine storm or secondary hemophagocytic lymph histiocytosis, in addition to isolated myocardial injury. Although it is uncertain whether SARS-CoV-2 may directly damage myocardial tissue and induce a major cardiovascular event, it is currently recommended that physicians should regularly monitor plasma cTnI and NT-proBNP levels in all COVID-19 patients. However, longer-term follow-up studies of these infected patients determined by cardiac function parameters (by using transthoracic echocardiographic examination) are needed.

Cardiac arrhythmias

After the myocardial injury, arrhythmia is another cause of cardiac involvement in COVID-19 patients that fluctuate from tachycardia to bradycardia and asystole. According to the study of 121 COVID-19 patients showed that most of these patients had shown cardiac arrhythmia, including 87 (71.9%) with sinus tachycardia unrelated to fever, 18 (14.9%) with bradycardia, and one patient with paroxysmal atrial fibrillation [40]. In another study, there have been studies that cardiac arrhythmias occurred in 23 (16.7%) of 138 patients with SARS-CoV-2 infection, especially among those admitted to the ICU [28]. Another interesting observation was made among the vasoplastic population have low systemic vascular resistance in the Wuhan cohort, where a higher proportion of critically ill COVID-19 patients and non-survivors had increased blood pressure values, which might contribute to arrhythmia, potentially explaining the pathological activity of SARS-CoV-2 infection [34, 58]. However, due to the retrospective nature of these data, it is difficult to find the cause of this observed hypertension is due to physiological reactions to the viral illness, or it is a consequence of virus-induced derangements in ACE2 expression. Overall, this indicated that arrhythmia may be an important complication among patients with SARS-CoV-2 infection. These findings suggest that especially in patients with severe COVID-19, regular electrocardiogram monitoring is needed to closely monitor patients for paroxysmal tachycardias and pulse accelerations that do not match the patient’s condition [42].

Sudden cardiac death

According to the study involving 99 patients infected with SARS-CoV-2 quarantined at the Wuhan Jinyintan Hospital, China, were (11%) deaths occurs due to sudden cardiac arrest among the patients without a previous history of ischemic heart disease [28]. These results suggest that the cause of death might be caused mainly by an imbalance of the pulmonary ventilation-perfusion ratio and a decrease in the capacity of the pulmonary vasculature. While acute myocarditis might contribute to heart failure and some investigators have been reported depressed left ventricular ejection fraction due to COVID-19, the majority of COVID-19 patients with uncomplicated lymphocytic myocarditis had normal cardiac function [33, 34]. The pathophysioligic factors possibly involved a) occlusion of microvasculature b) depletion in the number of functional residual gas, which leads to increased resistance to the pulmonary vessel that resulting in subsequent pulmonary hypertension and cor pulmonale. Cardiac dysfunction is caused due to direct virus infection or systemic inflammation which might potentially cause coronary micro-circulation dysfunction and downstream myocardial ischemic consequence, hence the relationship between SARS-CoV-2 infection and heart failure remains unclear. It is important to be aware of this condition to try and prevent cardiac arrest (especially in those patients who have a previous history of ischemic heart disease) so that appropriate prevention may be taken to lower the risk of death.

Liver dysfunction

In liver dysfunction described the liver function tests, such as serum transaminases, bilirubin, LDH, and prothrombin time (PT), were notably higher in COVID-19 patients who were admitted to ICU compared to non-ICU patients. In a study of 138 critically ill COVID-19 patients without a history of chronic liver diseases, who were admitted to ICU. Also, there is mild to moderate elevations of several liver enzymes (mostly induced by severe illness) were reported in a large multicenter Chinese study of 1099 COVID-19 patients [36]. In clinical practice, the liver function test results of patients with mild SARS-CoV-2 infection were relatively common. Instead of patients with severe (but non-critically ill) SARS-CoV-2 infection had mild to moderate elevations of serum transaminase and LDH levels [35]. Jundice was noticed only in a few SARS-CoV-2 infected patients, who were died during hospital admission; however, hypoalbuminemia and a longer PT were also observed amongst patients who afterward died. Liver failure has also been observed with other organ failures in non-survivors of SARS-CoV-2 infection and thus, it is not easy at this time to express the excess risk of death caused due to liver failure alone [38]. The current proof suggests that liver injury occurs more frequently among those patients who were seriously ill with COVID-19, who had other co-existing issues of liver damage such as the liver’s potential hepatotopic therapies and the co-existence of systemic inflammatory response, respiratory distress syndrome-induced hypoxia, and multiple organ dysfunction [31]. Several studies showed that in patients with chronic liver diseases, [47, 48] especially in those with pre-existing cirrhosis, [49, 50] there is an increased risk of greater COVID-19 illness and in-hospital mortality, which suggests specialized intervention strategies in these patients might help avoid a worse outcome.

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Management of liver transplant recipients has remained a challenge for physicians during the COVID-19 outbreak. It is admitted that transplant recipients are more susceptible to SARS-CoV-2 infection, are more probably to have increased severity of illness, and prolonged viral shedding [51, 52]. The patient eventually did not be rescued due to optic shock as a result of multiple infections, possibly worsened by corticosteroid treatment (for the chronic rejection). Another reason for a post-transplant patient who successfully recovered from COVID-19 developed nosocomial infections, almost like the previous case [54].

However, early discontinuation of immunosuppressive therapy (tacrolimus and mycophenolate) was analogous with recovery. In addition, SARS-CoV-2 infection during the perioperative period may additionally represent an opportunistic infection to those patients who were treated with immunosuppressive drugs to prevent acute graft rejection, and thus, it is advised to delay the scheduled transplantation procedure. However, there was a reported case where a patient who had COVID-19 before liver transplantation, recovered from COVID-19, 60 days after transplantation related to a lowered dosage of immuno-suppressant agents [55]. Moderate immune suppression can lead to secondary infections, in comparison to acute graft rejection.

Neurological complications

Neurological symptoms

Very little information is accessible on the possible adverse effects of SARS-CoV-2 infection on the neurological system. The signs and symptoms caused by the SARS-CoV-2 infection on the neurological system can be categorized into three types: (a) Central nervous system shows, such as dizziness, headache, disturbance and consciousness, acute cerebrovascular disease, and epilepsy; (b) peripheral nervous system shows neuralgia and loss in taste, smell, and appetite; and (c) skeletal muscle injury. In a retrospective study of 214 patients diagnosed with SARS-CoV-2, 78 of these patients had shown some neurological symptoms, accounting for 36.4% of all confirmed COVID-19 patients [56]. Patients with severe COVID-19 were developed neurological symptoms, such as acute cerebrovascular disease, impaired consciousness, and skeletal muscle injury [56, 57].

Psychological disorders

The psychological effects include post-traumatic stress, confusion, and anger are observed throughout quarantine during an infectious outbreak and are well noticed [58, 59]. After the three years of the SARS epidemic that happened in 2013, two studies have been reported that are (i) alcohol abuse or (ii) dependency symptoms, as long-term effects were observed during quarantined in healthcare workers [60, 61]. Increased avoidance behaviors also have been reported to common in healthcare workers after quarantine, like observed avoiding direct contact with patients and absence of mind at work, were found significantly associated with an increase in the duration of the quarantine.

Factors influencing psychological disorders

Several factors may cause psychological disorders during quarantine.

Like the history of psychiatric illness was found to be closely related to anxiety and anger with those patients who were concerned to release from quarantine [62]. Healthcare workers appeared severe symptoms of post-traumatic stress as compared to non-healthcare workers after quarantined [63]. After quarantine some healthcare workers also felt increased levels of guilt, having more avoidance behaviors, a higher loss in income, and felt more negatively affected psychologically. The various psychological effects include increased worry, anger, fear, frustration, guilt, isolation, loneliness, and nervousness. Though one study showed that a cut-off of 10 d of quarantine duration influenced the outcome of psychological impact, [64] it is generally accepted that a longer duration of quarantine is more likely to induce poorer psychological outcomes and mental health conditions [60, 63, 64]. Other factors like fear, [62] adverse reactions to imprisonment or isolation, [65] lack of sufficient information from authorities (regarding well-being and duration of quarantine), and terror of the financial loss.

CONCLUSION

Due to the COVID-19 pandemic global crisis has been arise. On the human health as the long term health issues has been observed. Also the health care workers faces the psychological complications, like loneliness, avoidance behavior. Due to all of this complication we need to take care from Covid-19. And maintaining the social distance and always wear mask whenever going to outside.

ABBREVIATION

SARS-CoV-Severe acute respiratory syndrome coronavirus, ARDS-Acute respiratory distress syndrome, ACE2-Angiotensin-converting enzyme 2, MERS-Middle east respiratory syndrome, RNA-Ribonucleic acid, AKI-Acute kidney injury, NT-proBNP-N-terminal pro-B-type natriuretic peptide, LDH-lactate dehydrogenase, PT-Prothrombin time.

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All the authors have contributed equally.

CONFLICT OF INTEREST

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