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New onset of health complications in patient after COVID-19 recovery.

Nueva aparición de complicaciones de salud en paciente tras recuperación de COVID-19.

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Abstract

Global pandemic coronavirus caused by COVID 19 appears to cause significant morbidity and mortality worldwide. Until now, acute respiratory problems, particularly in critically ill patients, have been the primary concern of clinical communication. Several case studies and limited series have indicated that COVID-19 significantly affects the respiratory and cardiovascular processes. Older individuals are at increased risk of serious illness due to COVID-19, and frequency increases with age. Many with ongoing medical conditions can also have a greater risk of severe illness. Various organs that may induce infection are the lungs, skin, kidneys, liver, heart and GI tract. Thus, the risk of severe illness due to COVID-19 rises in patients with comorbidities linked to these organs. Patients healed after COVID-19 should now be more vigilant in their daily health check-ups and surveillance. The present analysis showed a health complication following the recovery of COVID-19 that was required to avoid chronic disease, which would again cause mortality and morbidity in patients.
Keywords – COVID-19 recovered, secondary illness, health problems of COVID-19.

RESUMEN

El coronavirus pandémico global causado por COVID 19 parece causar una morbilidad y mortalidad significativas en todo el mundo. Hasta ahora, los problemas respiratorios agudos, particularmente en pacientes críticos, han sido la principal preocupación de la comunicación clínica. Varios estudios de casos y series limitadas han indicado que el COVID-19 afecta significativamente los procesos respiratorios y

cardiovasculares. Las personas mayores tienen un mayor riesgo de enfermarse gravemente debido a la COVID-19 y la frecuencia aumenta con la edad. Muchos con condiciones médicas en curso también pueden tener un mayor riesgo de enfermedad grave. Varios órganos que pueden inducir la infección son los pulmones, la piel, los riñones, el hígado, el corazón y el tracto gastrointestinal. Así, el riesgo de enfermedad grave por COVID-19 aumenta en pacientes con comorbilidades ligadas a estos órganos. Los pacientes curados después de COVID-19 ahora deben estar más atentos en sus controles y vigilancia de salud diarios. El presente análisis mostró una complicación de salud tras la recuperación de la COVID-19 que se requería para evitar la enfermedad crónica, que volvería a causar mortalidad y morbilidad en los pacientes.
Palabras clave – Recuperados de COVID-19, enfermedad secundaria, problemas de salud de COVID-19.

INTRODUCTION

Coronavirus Disease 2019 (COVID-19) has been declared a global public health emergency by the World Health Organization (WHO) due to its pandemic status (Huang et al. 2020). Mysterious pneumonia, characterized by fever, dry cough and exhaustion, was recorded in December 2019 in Wuhan, China, with rapid dissemination across the world, and became a public health emergency of international concern. The pathogen has been classified as a member of the coronavirus family, a strain that causes the common cold and flu (Wang et al., 2020). WHO reported to date, separate corona family virus strains have been reported in which MERS-CoV, SARS-CoV and now SARS-CoV-2 (COVID Pathogen 19) are three human coronaviruses that inflict more severe illness. The novel Extreme Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is associated with elevated morbidity and mortality. After its first occurrence in Wuhan, China, Globally, as of 1:08 p.m. CEST, 20 September 2020, 30,675 confirmed cases of COVID-19, including 954,417 deaths, were reported to the WHO.

According to WHO, the mortality risk of COVID-19 patients is 3-5 per cent who experience severe pneumonia, acute respiratory distress syndrome (ARD), multi-organ failure (MOF) and death (Zhu et al., 2020). The remaining patients affected have only minor symptoms and can recover quickly. Commonly recorded signs of COVID-19 include fever, dry cough and fatigue. Maniaci et al., (2020) found much less frequent symptoms affecting individual patients include aches and discomfort, fever, nasal inflammation, conjunctivitis, sore throat, lack of taste or scent, rash or discolouration of the finger or toes. The primary

purpose of this research was a thorough analysis of existing and preprinted publications documenting all symptoms associated with the COVID-19 presentation and also a summary of recurring symptoms in patients who were released from the hospital after the COVID-19 recovery (Salehi et al., 2020). This research will continue to predict possible epidemic problems and will provide more information on the production of medicines and vaccines in current pandemics and also in possible pandemics of this kind. This study focuses on several multi-organ changes/damages that may develop as a result of COVID-19 disease and recommendations for how to cope with COVID-19 recovering patients with daily clinical follow-up and guidance about how to help them as well as the community to understand the disease and its pathogenesis.

COVID 19 IMPACTS ON HUMAN HEALTH

SARS-CoV-2 uses angiotensin-converting enzyme-2 (ACE2) receptor to enter the human body. The ACE2 receptor is highly expressed in the mouth and tongue, allowing rapid penetration of the virus to the host and the lungs in alveolar epithelial cells. It is also represented in other tissues, such as the Kidney, the heart and the intestine. SARS-CoV-2 binds to these ACE2 receptors found in multiple organs that cause COVID 19 comorbidities such as acute respiratory syndrome (ARDS), acute kidney injury (AKI), heart harm and abdominal pain (Ahmed et al., 2020 ; Zaim et al., 2020).

Impact of COVID-19 on the lungs:

COVID-19 actively impacts the lungs and destroys the alveoli (tiny airbags). The role of the alveolus is to transport oxygen to the blood vessels. These blood vessels or capillaries are bringing oxygen to the RBCs (red blood cells). It is the RBCs that eventually provide oxygen to all the internal organs of the body. After infection, the spike glycoproteins expressed in SARS-CoV-2 envelop binds ACE2 to the alveolar cells and activates the clathrin dependent endocytosis of the SARS-CoV-2-ACE2 complex, contributing to cell membrane fusion. Since joining the cell, SARS-CoV-2 uses an endogenous transcription machine of alveolar cells to multiply and propagate across the lung. As the infection progresses, it affects the normal function of ciliated cells in alveoli and clears the airway and causes a progressive accumulation of debris and fluids on the alveoli wall and thickens the linen. According to which the flow of oxygen to the red blood cells is hampered. The more massive the wall, the more difficult it is to pass oxygen to the red blood cell, which causes difficulties with breathing when the body is running out of oxygen and ultimately contributes to acute respiratory distress syndrome (ARDS). In the lungs, the activation of virus-mediated cytokines in alveolar macrophages results in lung fibrosis and injury.

Furthermore, the lack of oxygen in the internal organs results in a deficiency in the body which impairs the operation of the organs. At this juncture, the body battles to increase the oxygen consumption, and the body's first reaction is to kill the virus to avoid its reproduction, yet if the organism has low defenses, the body will not be able to escape the virus, which exacerbates the situation. Since SARS-CoV-2 may cause lung fibrosis and damage in most cases, it is mandatory to routinely monitor the health status of the recovered COVID-19 patient to understand the full mechanism and long-term impact of this disease.

The higher risk to Pneumonia:

When alveoli are weakened, there is a cascade of liquids, inflammation and plasma proteins, and this build-up of fluid contributes to pneumonia. This further impairs the absorption of oxygen by the lungs and hinders the exchange of oxygen. Owing to the novelty of the COVID-19 strain, there is no specific therapy to cure pneumonia in COVID-19 patients specifically, and the majority of patients receive supportive care. Doctors evaluate the initiation of ARDS in COVID-19 infected individuals by certain primary signs in COVID-19 infected individuals such as hypoxia (low oxygen levels in the blood due to alveolus damage), trouble breathing and shortness of breath, chest x-rays of the lungs showing a blurred and glassy appearance against the black backdrop and deterioration of symptoms over time from the day of diagnosis (Azkur et al., 2020).

Acute cardiac injury:

Studies have shown that patients in China who have been diagnosed and hospitalized with COVID-19 have experienced a variety of cardiac disorders, including arrhythmias. Researchers from Washington, U.S.A., examined patients that became severely ill due to COVID-19 and discovered that patients were also afflicted with elevated levels of heart complications. It has been presumed that COVID-19 can cause serious heart problems long after a patient has recovered from Coronavirus.

Pneumonia is the main symptom of COVID-19 infection that induces inflammation in the entire body. Owing to inflammation in the plaque vessels, the plaque can become dysfunctional, which can ultimately lead to heart attacks. Many studies from different countries suggest that SARS-CoV-2 can affect myocardium and cause myocarditis or heart failure. Myocarditis is caused by the existence of the ACE2 receptor (SARS-CoV-2 binding site) on it. Binding this virus to the ACE2 receptor will induce an acute immune response and start a cytokine storm. The resulting cytokine storm may cause an increase in the proliferation of cardiac muscle cells and may form a fibrous cap on the fatty steak that causes the breakdown of the necrotic lipid center of the blood clots resulting in myocardial

infarction. Hypoxia also refers to myocardial damage due to lung failure. Wang et al., (2020) reported that there is an improvement in the amount of troponin protein in the heart muscle of a COVID-19 patient. This may be a valuable method to detect any cardiac abnormality related to SARS-CoV-2 infection in advance. However, since this virus is new, its effects for the heart are still unclear, and the researcher may try to follow up on the patient healed from COVID-19 (Li et al., 2020).

Higher risk to secondary Diseases

A secondary infection occurs when patients develop an infection that is not linked to the first patient problem. In this scenario, if anyone with COVID-19 starts to get infected with another virus. Reviews and research performed on COVID-19 patients who have been hospitalized have shown that secondary infection is very likely, although it is not shared. That may happen when the patient's immunity is very weak; thus, whether they fight (or rebound from) the virus, they can get a bacterial infection. Strep and staph infections are prevalent perpetrators, if the virus is not treated immediately. They can be extreme enough to risk death (Goumenou et al., 2020, Van Arkel et al., 2020).

Septic shock

Sepsis happens as the body responds to an infection that goes astray. Chemicals released in the blood do not cause the right reaction to the disease, and organs are adversely affected. If the Sepsis process is not halted as quickly as possible patients may be in a state of septic shock; significantly if the blood pressure decreases too high, then the septic shock may be severe (Murthy et al., 2020).

Impact on the GIT of COVID-19

Any COVID 19 patients documented signs of GI including diarrhea, anorexia, vomiting, fatigue, stomach pain and gastrointestinal bleeding during sickness. Data from the studies shows that gastrointestinal symptoms could appear early in the course of the disease. This is concluded with a study in which neonate first displayed symptoms of GI, including vomiting and milk rejection (Zhu et al., 2020) prior to other symptoms of COVID-19. Viral particles have been isolated from the fecal matter of infected persons, involving gastrointestinal conditions in COVID-19 (Tian et al., 2020). According to Zhang et al., (2020), there is a risk for viral exposure in the liver to induce inflammation in COVID-19 patients (Zhang et al., 2020; Yang et al., 2020). Liver damage can be characterized by a difference in the number of liver enzymes aspartate aminotransferase (AST), alanine aminotransferase (ALT) (Parohan et al., 2020). A similar rise in enzymes and liver damage has been observed in a 55-day-old child infected with SARS-CoV-2 (Cui et al., 2020). In patients with SARS, there has been an improved distribution of the liver and related

markers (Chau et al., 2004). In apoptosis-related markers and cytokines, abnormalities were also observed in pediatric patients with COVID-19 (Sun et al., 2020). These symptoms of the liver must be considered when delivering treatment for COVID-19 (Rismanbaf and Zarei, 2020). However, it would be possible to implement precautionary measures if these anomalies were detected at the first level. Similar damage to the organ of the Kidney has been confirmed. More research is needed to thoroughly understand the long-term consequences of infection with COVID-19.

Acute damage to the Kidney:

This is an uncommon complication, but if it does, it may be hazardous. If kidneys stop working, the doctors will immediately start the medication to minimize the damage. Patients could be on dialysis (a system that cleans your blood) before your kidneys begin to function normally. Often the injury will not heal properly, and patients will have chronic kidney failure, which will require long-term management.

Acute renal injury (AKI) has been documented in SARS-CoV-2 patients (Yang et al., 2020) due to the existence of ACE2 receptors. Proximal convoluted renal tubules are strongly expressed with ACE2 and Trans membrane Protease Serine 2. Both of these allow viral spike glycoproteins to be activated and promote virus-host cell membrane mergers leading to host cell entry, replication and spread. Inflammation and apoptosis processes in response to immune reactions can also affect the Kidney, resulting in inflammation and kidney damage (Li et al., 2019). Radian et al., (2015) studied that in the Kidney, active nucleotide-binding domain (NOD)-like receptor protein 3 (NLRP3) signalling can induce pyroptosis and cell death. It is also necessary to carry out follow-up research and routine health status tests to ensure there no ongoing problems are arising from COVID-19.

Blood clotting:

Blood clots can be caused by a condition known as diffuse intravascular coagulation (DIC). As a result of this condition, the blood-clotting system will function differently than it should; large clots will begin to form in the major organs of the body, leading to internal bleeding and early detection of organ failure. Analysis in Chinese patients with COVID-19 found that DIC was common among deceased patients (Lemke et al., 2020)

Post-viral fatigue

Post-viral fatigue has also been observed in people suffering from Ebola virus infection. Wilson et al., (2001) reported that 28% had extreme post-Ebo-Virus fatigue. Post-Ebola Syndrome has common symptoms with ME / CFS, particularly weakness, muscle and joint pain and sleep disorders. Similarly, Rowe et al., (1999) reported that 8.1 per cent of patients suffered extreme fatigue while recovering from prior Ebola virus outbreaks.

Furthermore, this intense exhaustion was often accompanied by myalgia and joint pain. Post-infectious fatigue development has been seen in other studies. Collin et al., (2017) found that 17.5 per cent of patients had chronic fatigue within 6 months of widespread viral infection. Moreover, Rossi et al., (2010) found that approximately 31 per cent of people suffering from West Nile virus infection registered chronic fatigue, while 64 per cent were qualifying for ME / CFS.

Effect of COVID-19 on Central Nervous System (CNS)

Mahalaxmi et al., (2020) observed that The CNS serves as the central processing center of the human system, which coordinates and monitors essential aspects of body activity and retains homeostasis. The CNS may be infected with SARS-CoV-2 by an olfactory bulb infection that can cause some neurodegenerative disorders such as hyposmia, hypoplasia, hypogeusia and headache. If the CNS virus infection is acute, chronic, or latent, so the immune system does not respond to the resultant neurological condition. Various cases of neurological symptoms associated with COVID-19 infection have been reported. Another study of a 74-year-old man with a positive and medical history of chronic lung disease and Parkinson's disease have reported neurological problems such as epilepsy and speech inability (Filatov et al., 2020).

The hypothesis behind the neurological problems associated with viral infection is that viral infection causes a cytokine storm in the brain as the host cell immune system overreacts to the virus, resulting in the release of numerous pro inflammatory cytokines such as Interleukine-6 (IL-6), Interleukine-12p40 (IL-12p40), Interleukine-15 (IL-15), tumour necrosis factor-ш (TNF-ш), Chemokine (C-X-C motif) ligand 9 Since COVID-19 is novel, further studies and analysis should be needed to identify the connection between neurological damage and COVID-19 infection and to develop the appropriate steps to prevent it (Bonam et al., 2020).

Impact on the eyes of COVID-19

The ACE2 receptors are also found in the retinal pigment of the retina epithelial cells in the eye. It then also serves as a binding position for SARS-CoV-2 to enter the body system. During an eye attack, the nasolacrimal system (tears) serves as a pathway of transmission to other organs of the human brain (Qing et al., 2020). Patients with complimentary COVID-19 exhibit eye-related signs such as conjunctivitis, conjunctive chemosis, and epiphora (watering eyes). Xiong et al., (2020) confirmed that COVID-19 patients with ocular symptoms had haematological anomalies such as elevated levels of white blood cells (WBCs), neutrophils, prolactin and C-reactive protein relative to COVID-19 patients with no ocular symptoms. These studies have shown that, apart from the nose or

the lips, the ocular route can also be present as a transmission route for SARS-CoV-2 (Barro-Soria et al., 2012; Balachandar et al., 2020).

Stress and psychiatric disorders

Li et al., (2020) stated that Globally, people understand that this pandemic is making people feel anxious; sad or worried; fear of contact with the virus; stress and distress associated with separation and quarantine; frustration in dividing family members; apprehension of long-term consequences of global disruption; concern regarding their inclusion in post-infection society; and among other reasons. Most civilians are still experiencing significant traumatic traumas in conflict zones and communities ravaged by abuse.

Reproduction system

Bhutta et al., (2012) observed pregnant women with COVID-19 pneumonia can risk-averse maternity and neonatal consequences. For patients receiving ovarian stimulation, mitigation of the risk of ovarian hyperstimulation syndrome is grave, as COVID-19 infection in a woman experiencing the hypovolemic and electrolyte imbalance typically associated with the syndrome may lead to an amplified risk of lung and kidney complications. Consequently, the use of mild stimulation, GnRH antagonist control of the luteinizing hormone (LH) surge, GnRH agonist triggering, and single embryo transfer or freeze-all, are the first choice in this period for women entering in vitro fertilization (La Marca et al., 2020).

CONCLUSION

Typical signs include coughing, fatigue, dyspnea, musculoskeletal symptoms (myalgia, knee discomfort and tiredness), gastrointestinal symptoms, and anosmia/dysgeusia. A thorough medical review, including a precise diagnosis and physical examination, can be given to patients. The post-acute outpatient service COVID-19 is currently operational, and more information on the patient evaluation procedure is mentioned elsewhere. Recovered patients should be vigilant to avoid the prognosis of the secondary disease and maintain optimal quality of life, lower morbidity and mortality following the recovery of COVID 19 patients. It is essential to establish an optimum nutritional status such that the patient is an outstanding candidate for successful treatment response.

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