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A review article of pharmacology of long COVID-19 and it's management

Vijayasri K [1], Lakshmi Manisha Rajaputana [1,*], Pravalika Mustala [1], Bhavani Bachu [1] and Sudhakar Muvvala [2]

[1] *Department of Pharmacology, Malla Reddy college of pharmacy, Osmania University, Dhulapally, Secunderabad, Telangana, 500100, India.*
[2] *Department of Pharmacy, Malla Reddy college of pharmacy, Osmania University, Dhulapally, Secunderabad, Telangana, 500100, India.*

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Abstract

A pandemic has recently been declared for COVID-19.Numerous preventive strategies and non-pharmaceutical therapies, including social distance, patient isolation, and strict infection control, have been employed to restrict the spread of disease. With oxygen therapy serving as the primary treatment intervention, management is primarily concerned with providing supportive care. Important management plans have also promoted medical treatment with corticosteroids and antivirals. Nevertheless, there is presently no vaccination for COVID-19 and no particular antiviral medication that is advised for its therapy. Even with the deliberate application of these steps, the rate of newly reported cases is still rising at a very concerning pace. We assess current knowledge on managing COVID-19 and provide an evidence-based overview of current practice as new findings become available. The number of newly reported cases is still increasing at a very concerning rate in spite of the deliberate application of these procedures. We examine current knowledge on managing COVID-19 and provide an evidence-based overview of current practice as new findings become available.

Keywords: COVID-19; Non-pharmaceutical therapies; Corticosteroids; Antivirals; Oxygen therapy; Evidence-based

1. Introduction

Severe acute syndrome coronavirus 2 2 (SAR-CoV-2) Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), initially named novel coronavirus or 2019-nCoV, is a single-stranded RNA virus which forms one of the seven coronavirus- 229E, OC43, NL63, HKU1, severe acute respiratory syndrome coronavirus(SARS-CoV) and middle east respiratory syndrome coronavirus (MERS-CoV).

On December 31, 2019, news of cases of acute pneumonia came from Wuhan, China. Betacoronavirus has been identified as the outbreak's causal culprit. According to genome sequencing, this virus is closely linked to the SARS-CoV (Severe Acute Respiratory Syndrome Coronavirus), which initially surfaced in 2003 and is also known as SARS-CoV-2 (Gorbalenya et al.2020; Zhou et al.2020). More than 80,000 infectious cases, including over 3,000 fatalities, were reported in China as of March 15, 2020, in a very short period of time.

COVID-19 (Corona Virus Disease 2019) is the name of the disease, which has now spread to over 216 nations and territories, including the United States, Germany, France, Spain, Japan, Singapore, South Korea, Iran, Italy, and India. Over 14 007 791 cases and 597 105 deaths had been reported worldwide as of July 19, with the number rapidly increasing in many nations.

* Corresponding author: R.L. Manisha

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The Chinese COVID-19 outbreak peaked as an epidemic in February. In early February, the overall number of cases increased significantly at an average pace of almost 3,000 newly confirmed cases each day, according to the Chinese National Health Commission. The outbreak of COVID -19 in China reached an epidemic peak in February. China put in place previously unheard of stringent public health to COVID-19. On January 23rd, Wuhan was declared closed ,and all forms of transportation that connected the city were prohibited. Public facilities were closed in most towns and rural areas throughout the next few weeks, and all outdoor activities and gathering were prohibited 18. This actions caused China's daily new case count to gradually decline. (2)

This suggests that cool and moderately humid environments favor prolonged airborne survival of the virus. The findings help explain the seasonal behaviour of respiratory viruses. such as their increased spread in colder months. The study also highlighted that HCoV-229E, being an enveloped virus, exhibited environmental sensitivity different from non-employed viruses like poliovirus. Overall, the research remain significant in understanding viral transmission dynamics in indoor environments.(3)

It is the virus responsible for causing coronavirus disease 2019 (COVID-19), a type of lower respiratory tract infections with the potential to cause severe and possibly fatal typical novel coronavirus (2019-nCoV)- infected pneumonia (NCIP) in humans.(4)

The symptoms that people report the most frequently include cognitive impairment, also referred to as "brain fog," joint, chest, and muscle pains, anosmia, shortness of breath, hair loss, sneezing, decreased libido, and, lastly, weariness (53.1%).(5)

The development of post-COVID-19 syndrome has been demonstrated to be highly predicted by a number of risk factors.For example, female sex, smoking,comorbidities and obesity are some risk factors.(6)

2. Pathophysiology of Long COVID

Both patients who experienced moderate or asymptomatic episodes of COVID-19 as well as those who acquired severe acute COVID-19 are being diagnosed with post-acute sequelae of the virus. For example, Tabacof et al. (2020) documented a variety of chronic symptoms in a group of patients with COVID-19 who had either been diagnosed or suspected of having the virus, and whose acute symptoms were mostly treated without hospitalization. Another team documented persistent COVID-19 symptoms in 1,407 subjects with confirmed SARS-CoV-2 injection(Huang Y,et al, 2020) Symptoms included fatigue and muscle weakness, insomnia, palpitation, chronic rhinitis, dysgeusia, chills, headache, and sore throat 72% of instances were patients aged 50±20 years, and 27% of participants reported ongoing symptoms after 60 days. The prevalence of persistent symptoms was higher in women, and around 32% of participants who reported symptoms 61+ days after infection had no symptoms when they were first tested for SARS-CoV-2.(7)

Among the potential causes of long-COVID are chronic inflammation, viral persistence, and long-term tissue damage; however, the review suggests that chronic brainstem dysfunction may potentially be Long-COVID, a post-virus sickness, can strike COVID-19 survivors regardless of their age or the severity of their first illness .continuous COVID-19 symptoms, irrespective of age or the intensity of the initial illness Cognitive deficits, cardiac and gastrointestinal problems, fatigue, myalgia, and dyspnea are some of the signs of chronic COVID involved, perhaps for the first time. a role.Long-COVID, a post-virus sickness, can strike COVID-19 survivors regardless of their age or the severity of their first illness. Long-lasting COVID-19 symptoms, regardless of age or starting sickness severity. Fatigue, dyspnea, heart and gastrointestinal issues, cognitive impairments, myalgia, and other symptoms are signs of long-term COVID. Long-term tissue damage, viral persistence, and chronic inflammation are among the potential causes of long-COVID; however, the review suggests—possibly for the first time—that chronic brainstem dysfunction might also play a role.

Two components can be distinguished in this hypothesis. The first is COVID-19's brainstem injury and tropism. When compared to other brain regions, the brainstem exhibits a comparatively high level of ACE2 expression. A co-receptor of SARS-CoV-2 RNA and protein Neuropilin-1 has also been demonstrated to reside in the brainstem.COVID-19 autopsy have also shown that the brainstem is extremely susceptible to damage from vascular or pathological immune activation. The respiratory, cardiovascular, gastrointestinal, and neurological systems are all regulated by a number of distinct nuclei and subunits that make up the brainstem. The second section discusses these functions that overlap with symptoms. which can be linked to long-COVID. Because neurons are slow to recover, brainstem dysfunction has been persistent and is hence long-lasting COVID. To be sure, myalgic encephalomyelitis, chronic fatigue syndrome, migraine, and chronic pain have all been linked to brainstem dysfunction.(8)

3. Epidemiology and Risk Factors

Although there are more reports of long-lasting coronavirus disease 2019 (COVID-19) symptoms, or "long COVID," little is known about their prevalence, contributing causes, or if a protracted course may be predicted early in the illness.In 4,182 COVID-19 incident cases, participants prospectively self-reported their symptoms using the COVID symptom research app 1.The data was then analyzed. Overall, symptoms persisted for at least 8 weeks in 189 participants (4.5%), 28 days in 95 participants (2.3%), and 12 weeks in 558 persons (13.3%).The traits of protracted COVID were anorexia, headaches, exhaustion, and dyspnea, and these symptoms were predicted to rise with female sex, age, and body mass index. More than five symptoms during the first week of illness were associated with long COVID. (odds ratio = 3.53 (2.76-4.50)).2,472 people in a separate sample tested positive for the coronavirus 2 that causes severe acute respiratory syndrome. A 76% area under the receiver operating characteristics curve was displayed by the model that was used to differentiate between short and long COVID at 7 days (with a 2,149 people make up the entire sample size. This strategy could be used to build education and rehabilitation programs, as well as to identify people at risk for long-term COVID for preventative or treatment studies. programs. There is a broad range of severity for COVID-19, starting with asymptomatic symptoms. For the purpose of accurately determining the length of sickness and the prevalence of persistent symptoms, few studies prospectively record symptoms in the general population. In this work, we report a prospective observational cohort analysis of COVID-19 symptoms in 4,182 COVID symptom study participants who reported positive SARS-CoV-2 test results and started using the app when they felt physically normal, enabling accurate symptom onset determination (Method)5,6. Symptomatic controls who were matched by age, sex, and body mass index (BMI) and who did not test positive were compared to these patients' duration of symptoms for COVID-19.Following that, Users whose symptoms lasted less than 10 days (short COVID) and those whose symptoms lasted more than 28 days (LC28) were compared. Following that, we contrasted users whose symptoms lasted longer fewer than 28 days (LC28) with individuals who experienced short-term COVID symptoms (less than 10 days). In light of our earlier research showing that clusters of symptoms predicted the need for acute respiratory support v7, we postulated that early symptom patterns linked to persistent symptomatology in COVID-19 (long COVID) could be predictive.(9)

4. Clinical Manifestation of Long COVID

Long-term complications after coronavirus disease 2019 (COVID-19) are common in hospitalization patients, but the spectrum of symptoms in milder cases needs further investigation. With a long-term follow-up of 312 patients, 247 of whom were home-isolated and 65 of whom were hospitalized, we conducted a prospective cohort analysis. In the first wave of the pandemic in Norway, this was the cause of 82% of all cases in Bergen.. At 6 months, 61% (189/312) of all patients had persistent symptoms, which were independently associated with severity of initial illness, increased convalescent antibody titers and pre-existing chronic lung disease, 52% (32/61) of the 16–30 year olds who were home-isolated at 6 months had symptoms such fatigue (21%, 13/61), memory issues (11%, 7/61), respiratory issues (13%, 8/61), and loss of taste and/or smell (28%, 17/61).Our results show that young, home-isolated persons with mild COVID-19 are susceptible to persistent dyspnea and cognitive problems, underscoring the need of infection control methods, such as immunization.(10)

An adult definition is: People who have a history of suspected or confirmed SARS-CoV-19 infection that lasts for two months or longer and cannot be accounted for by another diagnosis are considered to have a post-COVID-19 disease, which typically manifests three months after the virus first appeared. Typical symptoms that typically affect day-to-day functioning include fatigue, dyspnea, and cognitive impairment. After recovering initially from an acute bout of COVID-19, symptoms may persist from the initial illness or may be new. Furthermore, with time, symptoms could alter or reappear. Children could require a different definition. This shared framework serves as a basis for current and upcoming research on epidemiology, risk factors, clinical features, and therapy, even if the consensus definition is expected to evolve as knowledge grows.(11)

COVID-19 rapid guideline: managing the long-term effects of COVID=19 clinical guideline developed quickly in response to the urgent need for healthcare professionals to address the long-lasting symptoms that some people experience after recovering from acute COVID-19. These long-term effects, often called long COVID, include a wide range of physical and mental health symptoms such as fatigue, breathlessness, brain fog, and anxiety that can persist for weeks or even months after the initial infection. The term "rapid guideline" indicates that the document was produced more quickly than usual due to the public health emergency but still follows evidence-based methods. The focus on "managing" highlights that the guidelines provide advice on how to assess, treat, and support individuals with ongoing or new symptoms of COVID-19 beyond four weeks after infection, helping healthcare provide delivery coordinated, patient-centered The TriNetX electronic health records network, which has over 81 million patients, provided the data for this retrospective cohort study and time-to-event analysis. Our primary cohort consisted of patients with a diagnostic of COVID-19; one matched control cohort consisted of patients with influenza, while the other matched control cohort included patients with any respiratory tract illness, including influenza cohort within the same time period. The control cohorts did not include patients who had been diagnosed with COVID-19 or who tested positive for SARS-CoV-2. All cohorts comprised patients aged ≥10 years who were alive on Dec. 13, 2020, and who experienced an index incident on or after Jan. 20, 2020. The incidence of 14 neurological and psychiatric outcomes, including intracranial hemorrhage, ischemic stroke, parkinsonism, Guillain-Barre syndrome, nerve, nerve root, and plexus disorder, myoneural junction and muscle disease, encephalitis, dementia, psychotic, mood, and anxiety disorder (aggregated and separately), substance use disorder, and insomnia, was estimated six months following a confirmed diagnosis of COVID-19.We used a Cox model to compare the incidence of influenza or other respiratory tract infections with that of patients in propensity score-matched cohorts The impact of COVID-19 severity, as measured by hospitalization, intensive therapy unit (ITU) admission, and encephalopathy (delirium and related diseases), on these estimations was examined. By conducting the analysis again under various conditions, we evaluated the robustness of the variations in results between cohorts. Our group was compared to four other cohorts of patients who were diagnosed over the same time period and had additional index events, such as pulmonary embolism, urolithiasis, skin infection, and major bone fracture.(12)

4.1. Diagnosis Approach

Individuals who have COVID-19 may experience post-infection consequences. This illness, which has been listed in the ICD-10 as a post-COVID-19 condition since September 2020, is known by a number of names, including long-haul COVID and prolonged COVID.in its manifestation and effect. The discovery of potential treatments and the characterization of its epidemiology are hampered by the lack of a universally accepted and consistent definition. We participated in a Delphi process led by the WHO with a global panel of 265 patients, physicians, researchers, and WHO personnel to create a consensual definition for this confusion. In order to get a final consensus, 45 items and 14 domains were examined in two rounds of the Delphi method.(13)

4.2. Pharmacological Management

Post-acute COVID-19 cardiovascular sequelae are sustained by direct viral invasion, ACE2 downregulation, inflammation, and the immune response that compromises the structural integrity of the myocardial, pericardium, and conduction system. The virus was found in the cardiac tissue of 62.5% of patients 115 in 39 COVID-19 autopsy cases. Desmosomal proteins necessary for cell-to-cell adhesion may be displaced by fibro-fatty matter and cardiomyocyte mortality as a result of the ensuing inflammatory response 116,117. The long-term examination of SARS supervisor 118 revealed that recovered patients can have a chronically elevated cardiometabolic demand. This may be associated with reduced cardiac reserve, corticosteroids use and dysregulation of the renin-angiotensin-aldosterone system(RRRS). Virus-induced cardiomyopathy and myocardial fibrosis or scarring can cause reentrant arrhythmias 119. Because cytokines like IL-6, IL-1, and tumor necrosis factor-α can prolong ventricular action potential by regulating cardiomyocyte iron channel expression, COVID-19 may not prolong arrhythmias by causing a heightened catecholaminergic state 120. It has previously been documented that adrenergic modulation causes autonomic dysfunction during viral infection, which leads to inappropriate sinus tachycardia and postural orthostatic tachycardia syndrome.

4.3. Management consideration

If a patient has persistent heart symptoms, a history of acute infection, or cardiovascular problems, a series of clinical and imaging evaluations, including an electrocardiogram and echocardiogram at 4–12 weeks, may be considered 76,123. Since there is currently insufficient data to justify routine use of advanced cardiac imaging, each case should be evaluated separately. Absence from competitive sports or aerobic exercise for three to six months until myocardial inflammation is resolved by cardiac MRI or troponin normalization is advised for competitive athletes with cardiovascular complications associated with COVID-19 124,125. In patients with stable cardiovascular disease, RAAS

inhibitors have been demonstrated to be safe and should be continued, despite early theoretical worries about elevated ACE2 levels and the possibility of acute COVID-19 started and refined to be tolerated 129.Among 3,080 COVID-19 patients in a retrospective analysis, discontinuing medical therapy based on guidelines was associated with higher acute and post-acute mortality. Adrenergic activity in patients with postural orthostatic tachycardia syndrome and inappropriate sinus tachycardiaA131.. It is necessary to pay attention to the usage of medications like anti-arrhythmic agents (such amindarone) in patients who have fibrotic pulmonary alterations following COVID-19.(14)

This research explored the long-term effects of COVID-19 on organ health considered low-risk, meaning they were generally younger and had fewer pre-existing conditions. The study took a prospective and community-based approach, assessing participants months after their initial COVID-19 infection. Findings revealed that a significant proportion of these individuals exhibited signs of impairment in one or more organs, including the heart, lungs, liver, pancreas, and kidneys, despite having no history of severe disease or hospitalization. These results highlighted the potential for persistent, multisystem involvement in post COVID-19 syndrome (also known as long COVID), even among those not previously considered vulnerable. The study underscored the importance of ongoing monitoring and management strategies for individuals recovering from COVID-19,regardless of their initial risk profile.(15)

The authors highlighted that PVOD is a relatively common complication of upper respiratory tract infections and has gained increased attention during the COVID-19 pandemic, as anosmia (loss of smell) has emerged as a frequent symptom, sometimes occurring even in the absence of other major respiratory signs. The review explains that the olfactory dysfunction may result from damage to the olfactory epithelium (the tissue in the nasal cavity responsible for detecting odors) or from inflammation-induced injury to the olfactory nerve and central olfactory pathways. In many cases, the loss of smell is temporary, but in some individuals, it may persist for months or even become permanent.(16)

4.4. Non-Pharmacological Interventions

Aimed to comprehensively document the long- term symptoms of COVID-19 and their effects on individuals' daily lives. The study analyzed survey data from 3,762 respondents across 56 countries, most of whom were not hospitalized during the acute phase of infections, indicating that long COVID can develop even after mild or moderate illness. Participants were recruited through online COVID-19 support groups and social media platforms and all online COVID-19 support groups and social media platforms, and all had experienced symptoms lasting more than 28 days. The research identified a wide array of over 200 symptoms affecting ten different organ systems, with a particular focus on the duration, severity, and timing of symptom onset. Among the most prevalent and persistent symptoms were fatigue (reported by 77.7% of respondents), post-exertional malaise (72.25% and cognitive dysfunction (55.4%),often described by patients as “brain fog.” These symptoms were shown to fluctuate over time, with many individuals experiencing relapses triggered by physical or mental exertion, stress, or lack of rest. At seven months post-infection, the majority of respondents (over 85%) still reported symptoms, with a significant proportion experiencing more than 20 concurrent symptoms. The study also found that long COVID had a substantial socioeconomic impact:45.2% of respondents required a reduced work schedule. while 22.3% were unable to work at all due to ongoing illness. The authors emphasized the multisystem nature of long COVID and its capacity to impair quality of life and daily function significantly. Importantly, the study highlighted the need for broader clinical recognition of long COVID, the development of multidisciplinary treatment protocols, and increased research funding to understand its pathophysiology. Davis et al. captured a broad range of lived experiences. provided one of the earliest and most comprehensive portraits of long COVID’s complexity and chronicity, laying essential groundwork for public health response and patient advocacy. (17)

Investigation of Long COVID prevalence and its re Relationship to Epstein-Barr Virus Reactivation, published on *Raxiv*, explored the potential link between long COVID symptoms and the reactivation of Epstein-Barr virus (EBV), a common herpes virus known to persist in a latent state in most adults. The study aimed to determine whether EBV reactivation could be a contributing factor to the persistent symptoms experienced by individuals with long COVID. The researchers evaluated 185 individuals who had previously tested positive for SARS-CoV-2 and analyzed their serological profiles for EBV antibodies, including markers of recent or reactivated infection. Their findings showed that 66.7% of participants with long COVID symptoms had evidence of EBV reactivation, compared to a significantly smaller proportion of those who recovered without prolonged symptoms. Common long COVID manifestations in the EBV-reactivated group included fatigue, brain fog, and neurocognitive dysfunction—symptoms that closely mirror those seen in post-viral fatigue syndromes and chronic EBV-related illnesses. These results suggest a possible mechanistic overlap between EBV reactivation and long COVID pathogenesis. The study did not establish causality but raised an important hypothesis that EBV reactivation might play a role in exacerbating or sustaining long COVID symptoms, potentially through immune dysregulation triggered by SARS-CoV-2. The authors emphasized the need for further research, including longitudinal and mechanistic studies, to clarify the interplay between SARS-CoV-2 infection and latent viral reactivation. This

investigation provided a novel perspective on long COVID etiology and pointed toward the potential utility of monitoring EBV status as part of broader diagnostic and therapeutic strategies for patients with persistent post-COVID symptoms.(18)

5. Drug Development and Future Therapies

The researchers conducted longitudinal profiling of 209 COVID-19 patients patients across the acute and up to two to three months post-infection, integrating immune phenotyping, proteomics, metabolomics, and transcriptomics data. Through this deep analysis, they identified several key early factors that were associated with the development of PASC. These included pre-existing type 2 diabetes, latent Epstein-Barr virus (EBV) reactivation, autoantibodies, and specific viral anemia levels. Moreover, the team found dysregulation in innate immunity and persistent inflammation in PASC patients, which correlated with the initial host response to SARS-CoV-2 infection. The study underscores the complexity of long COVID and suggests that a combination of clinical and molecular factors evident early in infection can help predict the likelihood of developing chronic symptoms. These insights provide a foundational step toward early identification of at-risk individuals and the potential development of targeted interventions.(19)

HIV remains one of the most significant public health threats, one of the most significant public health threats globally, having infected over 75 million people and caused more than 35 million deaths since the early 1980s. The virus primarily targets the immune system—specifically $CD4^+$ T cells—leading to progressive immune dysfunction and increased vulnerability to life-threatening opportunistic infections and cancers opportunistic infections and cancers.

The article explains how HIV enters the body and establishes lifelong infection by integrating its genetic material into the host's DNA. This integration allows it to persist despite the immune system's efforts to control it. In the absence of treatment, the virus causes a steady decline in $CD4^+$ cells, eventually resulting in AIDS. Importantly, HIV also triggers chronic immune activation and inflammation, which not only contribute to immune system damage but also raise the risk of non-AIDS-related conditions such as cardiovascular disease, failure of the kidneys and neurological conditions.

A key focus of the review is antiretroviral therapy (ART), which has transformed HIV from a fatal disease into a manageable chronic condition. When taken consistently, ART can suppress viral replication to undetectable levels, prevent disease progression, allow immune recovery, and significantly reduce the risk of transmission. However, despite its success, ART does not eradicate the virus from the body. This is largely due to the presence of latent viral reservoirs—cells in which the virus lies dormant and hidden from the immune system and drugs.

The article also highlights major barriers to controlling the HIV epidemic. These include limited access to testing and treatment, particularly in resource-poor settings, stigma and discrimination; and the disproportionate impact on vulnerable groups including drug injectors, sex workers, and men who have sex with males. In addition, while prevention strategies like condom use, male circumcision and pre-exposure prophylaxis (PrEP) have proven effectives, their implementation remains inconsistent across population and regions.

Finally, Deeks and colleges discuss ongoing research efforts to develop a cure for HIV. These include strategies to flush out and destroy research, modify immune response to better target infected cells, and explore gene-editing technologies. While a cure is still unattainable, technological advancements are still being made. The article concludes by emphasizing that the HIV epidemic will require a combination of scientific advances, public health strategies, and social intervention aimed at ensuring universal access to prevention, care, and treatment.(20)

6. Challenges and Limitations in Management

Remdesivir, the combination of lopinavir and ritonavir, HIV protease inhibitors, chloroquine, and hydroxychloroquine are among the drugs being studied for the treatment of COVID-19. These drugs may prevent the virus from replicating in the early stages of infection. Treatment options also often include immunomodulatory drugs like corticosteroids (dexamethasone), interferons (IFNα and IFNβ), interleukin inhibitors (IL-1 and IL-6 inhibitors), kinase inhibitors (Bruton's tyrosine kinase or Janus kinase inhibitors), and immune-based strategies like convalescent plasma, SARS-CoV-2 immunoglobulins, nonspecific intravenous immunoglobulins (IVIG), and mesenchymal stem cells.In addition to SARS-CoV-2's neurological symptoms, a number of these treatments may have negative neurological adverse outcomes. Ribavirin and interferons, for example, are associated with neuropsychiatric and retinal symptoms, whereas chloroquine and hydroxychloroquine may be associated with neuropsychiatric side effects, retinopathy, ataxia, seizures, and limbic encephalitis.[150]. Even in patients who have no prior medical history of epilepsy, seizures are a known sign of SARS-CoV-2 infection; nevertheless, antiviral drugs (such as lopinavir, ritonavir, and ribavirin) may have

the unintended consequence of increasing the frequency of seizures. To improve clinical care, more study is therefore required to differentiate the detrimental neurological effects of SARS-CoV-2 from the neurological side effects of COVID-19 treatments.(21)

7. Conclusion

More understanding of the multi-organ effects of COVID-19 beyond the acute phase of infection has resulted from the collection of data and clinical experience throughout this time. The identification and characterization of important clinical, serological, imaging, and epidemiologic characteristics of COVID-19 in the acute, subacute, and chronic stages of the disease are essential current and future research projects that will aid in our comprehension of the natural history and pathophysiology of this new disease entity. Current and upcoming clinical research, such as prospective cohorts and clinical trials, as well as regular evaluation of new data by task forces and working groups, are essential for building a solid knowledge basis and guiding clinical practice in this field. Presently, identifying, meticulously recording, examining, and treating new or persistent symptoms, as well as monitoring organ-specific problems that arose during acute illness, are critical tasks for healthcare providers serving survivors of acute COVID-19. It is also essential that physicians present information in easily comprehensible ways, such as clinical trials in which participants can participate and extra resources like support groups and patient advocacy.

Furthermore, interdisciplinary collaboration is required for the comprehensive care of patients with COVID-19 in the outpatient setting, since it is evident that treatment for these patients does not end with hospital discharge. Hospitals and healthcare systems must therefore understand the importance of setting up specialized COVID-19 clinics where experts from several fields may offer integrated care. Individuals at high risk for post-acute COVID-19, such as those who experienced severe illness during acute COVID-19 and/or needed intensive care unit care, those who are most vulnerable to complications (e.g., elderly, those with multiple organ comorbidities, those post-transplant, and those with an active history of cancer), and those who bear the greatest burden of ongoing problems may be given priority for follow-up care.

To conclude, the current results emphasise the presence of multiple symptoms and, in turn, unmet healthcare needs of this large sample of hospitalised and non hospitalised patients with confirmed or suspected COVID-19 about 3 months after the infection. There may be a "post-COVID-19 syndrome" if symptoms continue to appear months after the infection.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no competing interests.

References

[1] Rathore JS, Ghosh C. Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), a newly emerged pathogen: an overview. Pathogens and disease. 2020 Aug;78(6):ftaa042.

[2] Hu B, Guo H, Zhou P, Shi ZL. Characteristics of SARS-CoV-2 and COVID-19. Nature reviews microbiology. 2021 Mar;19(3):141-54.

[3] Ijaz MK, Brunner AH, Sattar SA, Nair RC, Johnson-Lussenburg CM. Survival characteristics of airborne human coronavirus 229E. Journal of General Virology. 1985 Dec;66(12):2743-8.

[4] Sohrabi C, Alsafi Z, O'neill N, Khan M, Kerwan A, Al-Jabir A, Iosifidis C, Agha R. World Health Organization declares global emergency: A review of the 2019 novel coronavirus (COVID-19). International journal of surgery. 2020 Apr 1;76:71-6.

[5] Carfì A, Bernabei R, Landi F. Persistent symptoms in patients after acute COVID-19. Jama. 2020 Aug 11;324(6):603-5.

[6] Tsampasian V, Elghazaly H, Chattopadhyay R, Debski M, Naing TK, Garg P, Clark A, Ntatsaki E, Vassiliou VS. Risk factors associated with post– COVID-19 condition: a systematic review and meta-analysis. JAMA internal medicine. 2023 Jun 1;183(6):566-80

[7] Proal AD, VanElzakker MB. Long COVID or Post-acute Sequelae of COVID-19 (PASC): An Overview of Biological Factors That May Contribute to Persistent Symptoms. Front Microbiol. 2021;12:698169.

[8] Yong SJ. Persistent Brainstem Dysfunction in Long COVID: A Hypothesis. Med Hypotheses. 2021;149:110538.

[9] Sudre CH, Murray B, Varsavsky T, Graham MS, Penfold RS, Bowyer RC, et al. Attributes and predictors of Long COVID. Nat med. 2021;27(4):626–31.

[10] Blomberg B, Mohn KG, Brokstad KA, Zhou F, Munchausen DW, Hansen BA, et al. Long COVID in a prospective cohort of home-isolated patients. Nat Commun. 2021;12(1):6761.

[11] Taquet M, Geddes JR, Husain M, Luciano S, Harrison PJ. Six-month Neurological and Psychiatric Outcomes in 236379 Survivors of COVID-19. Lancet Psychiatry. 2021;8(5):416–27.

[12] World Health Organization. A clinical case definition of post COVID-19 condition by a Delphi consensus. Geneva: WHO; 2021

[13] National Institute for Health and Care Excellence. COVID-19 rapid guideline: managing the long-term effects of COVID-19. NICE guideline [NG188]; 2020.

[14] Nalbandian A, Sehgal K, Gupta A, Madhavan MV, McGroder C, Stevens JS, et al. Post-acute COVID-19 syndrome. Nat Med. 2021;27(4):601–15.

[15] Dennis A, Wamil M, Alberts J, Oben J, Cuthbertson DJ, Wootton D, et al. Multiorgan impairment in low-risk individuals with post-COVID-19 syndrome: a prospective, community-based study. JACC Cardiovasc Imaging. 2021;14(11):1996–2008.

[16] Marcellino C, Di Stadio A, Mignani F, Costantino A, Ralli M, Greco A. Post-viral olfactory dysfunction: A systematic review. Curr Treat Options Neurol

[17] Davis HE, Assaf GS, McCorkell L, Wei H, Low RJ, Re'em Y, et al. Characterizing Long COVID in an International Cohort: 7 Months of Symptoms and Their Impact. EClinicalMedicine. 2021;38:101019.)

[18] Gold JE, Okyay RA, Licht WE, Hurley DJ. Investigation of Long COVID Prevalence and Its Relationship to Epstein-Barr Virus Reactivation. medRxiv [Preprint]. 2021. doi:10.1101/2021.07.12.21260391.

[19] Su Y, Yuan D, Chen DG, Ng RH, Wang K, Choi J, et al. Multiple Early Factors Anticipate Post-Acute COVID-19 Sequelae. Cell. 2022;185(5):881–95.e20.

[20] Deeks SG, Overbaugh J, Phillips A, Buchbinder S. HIV infection. Nat Rev Dis Primers. 2015;1:15035.

[21] Nalbandian A, Sehgal K, Gupta A, Madhavan MV, McGroder C, Stevens JS, et al. Post-acute COVID-19 syndrome. Nat Med. 2021;27(4):601–15.)