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Review Article

"Post-COVID Complications" – The Long-term Effects After Recovery

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ABSTRACT

Objective: The aim was to investigate the evidence of post-COVID complications.

Material and Methods: This study has summarized scientific articles retrieved from databases such as PubMed, ScienceDirect, and Google Scholar from March 2020 to October 2022 using keywords.

Results: More than 35 articles have been reviewed and found 10 leading complications that occurred widely. Bibliographic materials such as systematic reviews, case studies, meta-analyses, research, and review articles were also reviewed.

Conclusion: In this review, we focused on and summarized the leading post-COVID-19 complications that have occurred worldwide.

Keywords: Post-COVID-19, Post-COVID syndrome, COVID complications, Post-COVID sequela

INTRODUCTION AND EPIDEMIOLOGY

The crisis of the coronavirus disease 2019 (COVID-19) has resulted in an incomparable worldwide pandemic.[1] Therefore, on March 11, 2020, the World Health Organization (WHO) declared COVID-19 as a pandemic. The Coronavirus disease (COVID) is a contagious disease caused by severe acute respiratory syndrome coronavirus (SARS-CoV-2).[1,2] Regardless of the advancement in medical research, science, and technology, we are still facing challenges with new pathogens causing hazards to human lives.[2]

Symptoms of COVID-19 may vary and could range from mild to severe illness.[3] The most common symptoms are cough, fever, dyspnea-like symptoms, runny nose, nasal blockage, anosmia, and ageusia. It is been reported that COVID survivors were experiencing several symptoms even after recovery from the deadly disease and needed longer medical support and care.[4] If the symptoms are seen for more than 12 weeks after COVID-19, it is known as post-COVID syndrome.[3] The most common mild complications among recovered patients are trouble in breathing, cough, and chest tightness despite normal lung function. Brain fog, chronic fatigue, joint and muscle pain, and body aches are the common complications suffered

by most of the patients and need symptomatic treatment. A small percentage of the population, who developed long-term damage in various organs such as pulmonary fibrosis (scarring and damage of the lung tissue) and myocarditis where patients suffered breathlessness even after minimal activity, heart attack, and stroke problems because of COVID. Thus, the stroke problems increase the chances of clot formation. One such lethal complication was mucormycosis and the patients who were more susceptible were those with a medical history of diabetes mellitus, immuno-compromised, and used drugs such as steroids given during treatment of COVID.[1]

To treat COVID infection, numerous drugs were widely used such as antiviral (Remdesivir and lopinavir/ritonavir), immunomodulatory, and anti-inflammatory drugs (Hydroxychloroquine and chloroquine, Tocilizumab, Anakinra, and Sarilumab), anti-cytokine therapy, interferon, corticosteroids, convalescent plasma – immunoglobulin (IG) therapy, and angiotensin-converting enzyme (ACE) inhibitors. These drugs require strict monitoring because they are known to cause severe adverse effects. The Food and Drug Administration (FDA) issued an emergency use authorization (EUA) permitting hydroxychloroquine and chloroquine to be administered in COVID-infected patients for their anti-inflammatory and antiviral properties in March 2020; However, on June 15, 2020, the FDA withdrew these drugs because it had been observed thatthe drugs were not effective for treating COVID and caused serious cardiac problems.[4] With the combination of lopinavir and ritonavir therapy, 50% of patients experience an adverse effect. Fourteen percent of the patients discontinued this therapy due to hepatotoxicity and gastrointestinal adverse effects.[5] Under EUA, remdesivir was authorized only for the treatment of patients with suspected or laboratory-confirmed SARS-CoV-2 infection and severe COVID-19 because of its broad-spectrum activity. It was authorized for adult and pediatric patients who were admitted to the hospital.[4]

LITERATURE SEARCH STRATEGY

This is a narrative review based on scientific databases such as PubMed, ScienceDirect, and Google Scholar, which were searched for relevant articles from March 2020 to October 2022. The search words and a combination of search words such as "post-COVID-19 complications," "post-COVID Syndrome," "COVID complications," "post-COVID sequela," and complications of COVID-19." were used. The articles were screened and retrieved from the database by two authors of this article. The articles which were approved by at least two authors were reviewed in detail. The most common post-COVID complications were considered for the study and a total of 10 most prevalent complications are reviewed in this article.

HEPATOBILIARY COMPLICATIONS

Hepatic injury is one of the manifestations of prolonged COVID. Post-COVID cholangiopathy has the potential to develop biliary and liver injuries. Many patients, who recovered from COVID infection, consequently developed hepatobiliary diseases such as acalculous cholecystitis, cholangiopathy, and secondary sclerosing cholangitis. None of them had pre-existing hepatobiliary disease. Studies suggested, increase in the activity of ACE-2 receptors will directly leads to viral damage and cause cholangiopathy and associated diseases.[6] A few patients from the case reports [6-9] have been found with a medical history of hypertension, diabetes, and obesity but most of them have not been suffering from any of these diseases before. In laboratory reports, severely elevated aminotransferase levels, following bile duct obstruction, hepatocytes, cholangiocyte injury, swollen cholangiocytes, and fibrosis, were described in liver biopsy which indicates the possibility of secondary biliary cirrhosis.[8] In addition, leukocytes, D-Dimer, ferritin, and C-reactive protein (CRP) were also elevated. Endoscopic retrograde cholangiopancreatography, liver biopsy, and aminotransferase level are the gold standard methods for diagnosing hepatobiliary complications. Certain types of bacterial *Escherichia coli*, as well as fungal (candida species) infections, were reported. Antibiotic therapy such as ampicillin, vancomycin, carbapenem class of drug (meropenem and ertapenem), and piperacillin-tazobactam combinations were widely used. In addition, fluconazole as an antifungal therapy was mainly prescribed. As a treatment, urso deoxycholic acid and cholestyramine were used but the laboratory level was not improved.[8] Early diagnosis and management should be carried out to avoid the worsening of these complications.

RENAL COMPLICATION

In renal complications, acute kidney injury (AKI) is a common complication during hospitalization with COVID-19 and commonly developed in 5–15% of the patients admitted to the hospital leading to a mortality rate above 50%.[10,11] Risk factors for renal complications in COVID vary by variables such as gender, age, race, diabetes, obesity, hypertension, chronic kidney disease, heart failure, anemia, lymphopenia, leukocytosis, and increase in levels of serum inflammatory markers (D-dimer, polymerase chain reaction, and interleukin-6 [IL-6]). The use of mechanical ventilation and ACE inhibitors as well as vasoactive drugs was associated with a higher risk of AKI in critical patients with COVID.[10] A common phenomenon known as "cytokine storm" is characterized as a massive inflammatory

cytokine release, mainly IL-6 noted in severe COVID patients and therefore results in systemic vasodilation that increases endothelial and vascular permeability, which leads to AKI.[10,11] In addition, specific mechanisms of COVID in which the entry of SARS-CoV-2 into the kidneys and binding with ACE-2 receptor on the cell membrane of the host cells which appears in the apical brush borders of podocytes as well as proximal tubules in the kidneys subsequently causes an imbalanced activation of the renin-angiotensin–aldosterone system (RAAS), which leads to the down-regulation of the membrane-bound ACE-2 receptor that enhances angiotensin II accumulation by reducing its degradation into angiotensin.[6,7] Consequently, imbalanced RAAS activation causes inflammation, vasoconstriction, and fibrosis in the kidney.[11] The diagnosis of AKI as per the KDIGO (Kidney Diseases Improving Global Outcomes) criteria which is based on their creatinine value and diuretic rhythm and for treatment dialysis and renal replacement therapy was used.[10-12]

OLFACTORY COMPLICATION

Olfactory disorders (ODs) are found in two-thirds of COVID-infected patients.[13] OD is a common symptom of COVID-19 listed by the WHO.[14] The duration of the olfactory loss in COVID is still unclear but according to Wuhan's recent study,[15] using T and T Smell Test indicates ODs last longer in COVID-infected patients.[14] Mechanisms of ODs in COVID are still unknown, but it is probably the result of several patterns such as olfactory epithelial damage (including neural and non-neural epithelium), nasal mucosal edema, involvement of central olfactory pathway, severe and prolonged ODs which cause infiltrative infection of the supporting cells and sustentacular cells of the olfactory neuroepithelium.[13,16] For OD, there is no specific treatment, but systemic corticosteroids and nasal irrigation-based agents are used.[16] The most comprehensive assessment among the variety of available olfactory tests for OD is the Sniffin' Sticks because it includes discrimination, identification test, and threshold which can validate the peripheral and central olfactory system.[14]

NEUROLOGICAL AND NEUROPSYCHIATRIC SYMPTOMS

Neurological and neuropsychiatric symptoms developed after 3 months of the onset of COVID-19 infection.[17,18] Neurological post-COVID-19 symptoms were fatigue, anosmia, brain fog, attention disorder, memory issues, myalgia, dysgeusia, and headache. Neuropsychiatric post-COVID-19 symptoms include sleep disturbances, anxiety, and depression but major neurological disorders such as ischemic stroke, hemorrhagic stroke, and the seizure prevalence rate were fewer than others.[17,18] Based on the WHO Disability Assessment Schedule, cognitive dysfunction, fatigue, anxiety, and hypogeusia/hyposmia have been measured and account for more than 33% of COVID survivor disability levels.[17] It is hard to determine whether SARS-CoV-2 induces direct or indirect neurological/psychiatric damage but some studies suggested that the possibility of indirect effects such as inflammation caused by SARS-CoV-2 infection and contributes to the neurological/neuropsychiatric manifestations and the detection of the virus may be difficult due to low sensitivity of the assays, stages of infection, and timing at the time of examination.[19] Imaging techniques provide the early detection and diagnosis of neurological and neuropsychiatric manifestations, thus assist the treatment and improved understanding of pathogenesis.[20]

CARDIOVASCULAR COMPLICATIONS

Myocarditis

Myocarditis is an inflammatory condition characterized by a wide variety of symptoms. Myocardial inflammation appears in most COVID patients. The Dallas criteria set histologic, immunologic, and immunohistochemical criteria are now applied to the diagnosis of myocarditis. It is caused by a variety of infectious and/or non-infectious triggers, the most prominent of which are viral infections caused by coxsackievirus B, adenovirus, hepatitis C virus, cytomegalovirus, Epstein-Barr virus, parvovirus B19, and human herpesvirus. SARS-CoV-2 is known to cause myocarditis.[21] When SARS-CoV-2 penetrates human cells, it attaches to the membrane protein ACE-2 located on cardiomyocytes with its spike protein. The sudden onset of chest discomfort is the key symptom of myocarditis. ST-segment abnormalities, arrhythmia, and hemodynamic instability are some of the additional symptoms. Significant increase in N-terminal pro-brain natriuretic peptide (NT pro-BNP) levels, cardiac troponin, and left ventricular dilatation or motion abnormalities on echocardiogram have been used to diagnose the condition.[22]

Myocardial interstitial fibrosis

Diffuse and localized cardiomyocyte fibrosis within the hearts of COVID-infected individuals has also been suggested and it may occur without any cardiac symptoms. Necrotic cardiomyocytes are replaced by fibroblasts because of myocarditis, microinfarction, and vasculitis, which is the primary mechanism of fibrosis in COVID-19 patients. Furthermore, cytokine storm and immune cell infiltration of the myocardium trigger the conversion of fibroblast to myofibroblast resulting in matrix remodeling, which is the cause of fibrosis.[23]

Cardiac arrhythmias

Heart palpitation is one of the presenting symptoms in COVID patients. Arrhythmia is observed as sinus tachycardia; however, it is unclear whether it is related to increased cardiac output or structural alterations in the myocardium. Medication side effects, myocardial inflammation, interstitial tissue edema, conduction abnormalities, fibrosis, and myocarditis cause structural alterations in ion channels, and dysregulation which cause cardiac arrhythmias.[23]

RESPIRATORY COMPLICATIONS

Pulmonary fibrosis

A wide range of post-COVID pulmonary manifestations ranging from breathlessness, pneumonia, chest pain, and tightness to lung, and cystic fibrosis has been reported.[24] Post-COVID pulmonary fibrosis was one of the most threatening complications of the respiratory system. Ali and Ghonimy reported that 18.4% of mild group patients developed post-COVID pulmonary fibrosis whereas 42.8% of patients developed it in the severe group.[25] Studies suggested the most common risk factors associated with pulmonary fibrosis are a history of cigarette smoking, excessive alcohol consumption, older age, that is, more than 65 years, and prolonged intensive care unit (ICU) hospitalization. In laboratory parameters, CRP, IL-6, lactate dehydrogenase, and white blood cells were elevated with lymphocytopenia. Lung volumes such as total lung capacity, inspiratory reserve volume, expiratory reserve volume, and functional residual volume were decreased. On the other hand, arterial blood gases were abnormal, indicating respiratory alkalosis and hypoxemia.[26] Computed tomography (CT) scan reports state dense fibrosis with bilateral interstitial infiltrates involving a honeycomb-like structure and multiple fibroblastic foci. Steroid (dexamethasone 4–6 mg) has played a key role in managing COVID-19 ICU patients worldwide but consumption on a long-term basis could result in worsening hyperglycemia and myopathy-like conditions. Antifibrotic agents such as Nintedanib and pirfenidone showed reduced progression of pulmonary fibrosis effectively. Low molecular weight heparins such as enoxaparin were widely used along with supplemental oxygen and treamid (anti-inflammatory).

Pulmonary embolism

It is the risk factor associated with coronavirus as well as with COVID-19 vaccines. Fever, cough, chest tightness, dyspnea, and syncope were the common symptoms observed. Laboratory parameters: D-dimer, troponin, NT pro-BNP, CRP, and echocardiography are performed. Along with it, CT pulmonary angiography, chest X-ray, lung perfusion scintigraphy, and acute respiratory distress syndrome data have also been considered. Heparin/low molecular weight heparin and apixaban (factor Xa inhibitors) are widely used as treatment.[27]

MUCORMYCOSIS

It is a rare and life-threatening fungal infection associated with COVID infection and caused by a group of microorganisms called *Mucormycetes*. Mucormycosis most commonly occurs due to spore inhalation and affects nearly every organ but primarily the respiratory and central nervous systems.[28] There are numerous risk factors including long-standing diabetes, renal insufficiency, hematological malignancies, chronic sinusitis, immunocompromised condition, and medications such as steroids, broad-spectrum antibiotics, and drug abuse.[28] Furthermore, hematological malignancies, organ transplantation, hemochromatosis, and neutropenia exaggerate the condition. Clinical manifestations include sinusitis, pain, headache, fever, redness, swelling around the nasal cavity, eyelid swelling, loss of sight, and ophthalmoplegia. If it involves a cutaneous layer, then blisters and ulcers are observed, and the skin turns black. First, medical history, physical examination, and clinical manifestations are considered. Fluid analysis of the respiratory system, CT scan, magnetic resonance imaging, and tissue biopsy of the affected area are used for diagnosis.[29] Standard treatment was antifungal medications such as Amphotericin-B 5–10 mg/kg or Posaconazole 400 mg/day. Second, surgery is considered in addition to the optimum drug regimen to completely remove all the necrotic tissues.[28]

IMMUNE SYSTEM

Arthritis

Taha *et al*. reported that 37% of patients who recovered from COVID-19 infection are at substantial risk of developing post-COVID arthritis.[30] Most commonly it affects the knee, ankle, and wrist joints. The risk factor associated with post-COVID arthritis increases with age, smoking, and arthralgia. Laboratory parameters used for diagnosis are inflammatory markers (CRP and erythrocyte sedimentation rate [ESR]), IL-6, anti-cyclic citrullinated peptide antibodies, positive rheumatoid factor, and synovial fluid analysis.[31] Non-steroidal anti-inflammatory drugs (NSAIDs) are used in the treatment of rheumatoid arthritis to relieve pain associated with it. Corticosteroids (e.g., dexamethasone) at low doses are used to manage flare-up conditions at the initial phase of treatment. Disease-modifying antirheumatic drugs such as hydroxychloroquine, methotrexate, sulfasalazine, and leflunomide were also used. Vitamin D also plays a key role as an anti-inflammatory and in enhancing immunity.[32]

MUSCULOSKELETAL (MSK)

The post-acute symptoms of MSK, majorly include backache, spine pain, fatigue, myalgia, arthralgia, and generalized body ache. Medications used for treating COVID (hydroxychloroquine, chloroquine, and steroids) have the potential to develop myopathy, arthralgias, and other MSK related complications as their side effects. Studies have proven that drugs such as Lopinavir-ritonavir and ribavirin are linked with arthralgia, osteonecrosis, and various MSK disorders.[33] Inflammatory mediators contribute to the development of systemic inflammation, in which the elevated level of cytokines has a direct impact on skeletal muscles and reduces protein synthesis.[34] Laboratory parameters (complete blood count, ESR, CRP, D-dimer, and ferritin) and CT scan were used for diagnosis. Management involves NSAIDs to relieve pain, for example, ULTRACET, and exercise to increase range of motion and prevent stiffness. In chronic pain, heat cryotherapy is widely used to reduce swelling and manage pain.[35]

NERVOUS SYSTEM COMPLICATIONS

Complications associated with the central nervous system were anosmia, ageusia, headache, dizziness, convulsion, confusion, impaired consciousness, sleep disorders, hemiplegia, ataxia, encephalitis, seizures, ischemic stroke, meningitis, and cerebral hemorrhage whereas Guillain-Barre Syndrome (GBS) is the most common complication associated with peripheral nervous system.[36]

The exact pathogenesis behind the development of nervous system complications is still idiopathic; however, there are two potential mechanisms involved: First, there is an increase in the neurodegenerative process triggered by systemic inflammation which could aggravate clinical signs and symptoms of neurological complications in which, across the blood brain barrier, alteration in endothelial and leukocyte migration also occurs. Second, direct damage to the cerebral tissues across the infected neurons, trans-synaptic transfer occurs through the nerves with the interaction between ACE-2 receptors and virus spike proteins.[37]

Moreover, COVID has long-term effects on the mental state of patients particularly depression, anxiety, insomnia, and posttraumatic stress disorders.[37]

Seizures are developed due to the SARS-CoV-2 virus-induced brain damage, inflammatory mediators' spikes, and virus-induced meningitis or encephalitis. Focal seizures and generalized tonic-clonic seizures are the most reported seizures in post-COVID patients. Anti-epileptic drugs and electroencephalography monitoring are significantly considered in management.[38]

Ischemic stroke is basically due to large blood vessel occlusion. Arterial venous sinus thrombosis along with pneumonia was commonly reported in post-COVID patients.

GBS is a heterogeneous disorder, that is, more prevalent in elders and males.[37] GBS are mainly caused by *Epstein-Barr virus*, *Campylobacter jejuni*, and *cytomegalovirus*. Peripheral nerve antigens can cause inflammation and neuronal damage by cross reaction with anti pathogen antibodies. Most common neurological symptoms such as lower limb weakness, paresthesia and polyneuritis cranialis were observed in COVID-19 patients. All the GBS management is achieved by intravenous IG treatment.[37-39]

CONCLUSION

The COVID pandemic has been a significant challenge to the worldwide healthcare system. As a result of subsequent COVID-19 waves, various acute, as well as chronic "complications" or "sequela," have developed later. The post-COVID-19 syndrome is multifactorial and has been described above showing how various organ systems have been affected.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Mendelson M, Nel J, Blumberg L, Madhi SA, Dryden M, Stevens W, *et al*. Long-COVID: An Evolving Problem with an Extensive Impact. S Afr Med J 2020;111:10-2.
2. Chams N, Chams S, Badran R, Shams A, Araji A, Raad M, *et al*. COVID-19: A Multidisciplinary Review. Front Public Health 2020;8:383.
3. Suvvari TK, Kutikuppala LV, Tsagkaris C, Corriero AC, Kandi V. Post-COVID-19 Complications: Multisystemic Approach. J Med Virol 2021;93:6451-5.
4. McFee RB. COVID-19: Therapeutics and Interventions Currently Under Consideration. Dis Mon 2020;66:101058.
5. Sanders JM, Monogue ML, Jodlowski TZ, Cutrell JB. Pharmacologic Treatments for Coronavirus Disease 2019 (COVID-19): A Review. JAMA 2020;323:1824-36.
6. Roth NC, Kim A, Vitkovski T, Xia J, Ramirez G, Bernstein D, *et al*. Post-COVID-19 Cholangiopathy: A Novel Entity. Am J Gastroenterol 2021;116:1077-82.
7. Durazo FA, Nicholas AA, Mahaffey JJ, Sova S, Evans JJ, Trivella JP, *et al*. Post-Covid-19 Cholangiopathy-a New

Indication for Liver Transplantation: A Case Report. Transplant Proc 2021;53:1132-7.
8. Rojas M, Rodríguez Y, Zapata E, Hernández JC, Anaya JM. Cholangiopathy as Part of Post-COVID Syndrome. J Transl Autoimmun 2021;4:100116.
9. Edwards K, Allison M, Ghuman S. Secondary Sclerosing Cholangitis in Critically Ill Patients: A Rare Disease Precipitated by Severe SARS-CoV-2 Infection. BMJ Case Rep 2020;13:e237984.
10. Neves PD, Sato VA, Mohrbacher S, Ferreira BM, Oliveira ÉS, Pereira LV, *et al.* Acute Kidney Injury due to COVID-19 in Intensive Care Unit: An Analysis from a Latin-American Center. Front Med (Lausanne) 2021;8:620050.
11. Fabrizi F, Alfieri CM, Cerutti R, Lunghi G, Messa P. COVID-19, and Acute Kidney Injury: A Systematic Review and Meta-Analysis. Pathogens 2020;9:1052.
12. Moledina DG, Simonov M, Yamamoto Y, Alausa J, Arora T, Biswas A, *et al.* The Association of COVID-19 with Acute Kidney Injury Independent of Severity of Illness: A Multicenter Cohort Study. Am J Kidney Dis 2021;77:490-9.e1.
13. Gorzkowski V, Bevilacqua S, Charmillon A, Jankowski R, Gallet P, Rumeau C, *et al.* Evolution of Olfactory Disorders in COVID-19 Patients. Laryngoscope 2020;130:2667-73.
14. Otte MS, Eckel HN, Poluschkin L, Klussmann JP, Luers JC. Olfactory Dysfunction in Patients after Recovering from COVID-19. Acta Otolaryngol 2020;140:1032-5.
15. O'Byrne L, Webster KE, MacKeith S, Philpott C, Hopkins C, Burton MJ. Interventions for the Treatment of Persistent Post-COVID-19 Olfactory Dysfunction. Cochrane Database Syst Rev 2021;7:CD013876.
16. Chung TW, Sridhar S, Zhang AJ, Chan KH, Li HL, Wong FK, *et al.* Olfactory Dysfunction in Coronavirus Disease 2019 Patients: Observational Cohort Study and Systematic Review. Open Forum Infect Dis 2020;7:ofaa199.
17. Cacciatore M, Raggi A, Pilotto A, Cristillo V, Guastafierro E, Toppo C, *et al.* Neurological and Mental Health Symptoms Associated with Post-COVID-19 Disability in a Sample of Patients Discharged from a COVID-19 Ward: A Secondary Analysis. Int J Environ Res Public Health 2022;19:4242.
18. Premraj L, Kannapadi NV, Briggs J, Seal SM, Battaglini D, Fanning J, *et al.* Mid and Long-term Neurological and Neuropsychiatric Manifestations of Post-COVID-19 Syndrome: A Meta-Analysis. J Neurol Sci 2022;434:120162.
19. Bodnar B, Patel K, Ho W, Luo JJ, Hu W. Cellular Mechanisms Underlying Neurological/Neuropsychiatric Manifestations of COVID-19. J Med Virol 2021;93:1983-98.
20. Pantelis C, Jayaram M, Hannan AJ, Wesselingh R, Nithianantharajah J, Wannan CM, *et al.* Neurological, Neuropsychiatric and Neurodevelopmental Complications of COVID-19. Aust N Z J Psychiatry 2021;55:750-62.
21. Farshidfar F, Koleini N, Ardehali H. Cardiovascular Complications of COVID-19. JCI Insight 2021;6:e148980.
22. Meyer M, Vogel T, Meyer A, Constancias F, Porter LF, Kaltenbach G, *et al.* Presence of Active Myocarditis at the 6 Month Follow-up Appointment for a Severe Form of COVID-19: A Case Report. ESC Heart Fail 2021;8:4307-12.
23. Sulemankhil I, Abdelrahman M, Negi SI. Temporal Association between the COVID-19 Ad26.COV2.S Vaccine and Acute Myocarditis: A Case Report and Literature Review. Cardiovasc Revasc Med 2022;38:117-23.
24. Nalbandian A, Sehgal K, Gupta A, Madhavan MV, McGroder C, Stevens JS, *et al.* Post-Acute COVID-19 Syndrome. Nat Med 2021;27:601-15.
25. Ali RM, Ghonimy MB. Post-COVID-19 Pneumonia Lung Fibrosis: A Worrisome Sequelae in Surviving Patients. Egypt J Radiol Nucl Med 2021;52:101.
26. Kumar N, Gupta A, Vardhan H, Daga MK. Post-COVID-19 Pulmonary Fibrosis: An Update. J Adv Res Med 2021;8:16-26.
27. Sitani K, Verma P, Rao RD, Chandak A, Asopa RV. Pulmonary Embolism as Post-COVID-19 Sequelae: Role of Lung Perfusion Scintigraphy. Indian J Nucl Med 2021;36:455-6.
28. Sundaram N, Bhende T, Yashwant R, Jadhav S, Jain A. Mucormycosis in COVID-19 Patients. Indian J Ophthalmol 2021;69:3728-33.
29. Kumar PK. Mucormycosis: A Black Fungus-Post Covid Complications. J Regen Biol Med 2021;3:1-8.
30. Taha SI, Samaan SF, Ibrahim RA, El-Sehsah EM, Youssef MK. Post-COVID-19 Arthritis: Is It Hyperinflammation or Autoimmunity? Eur Cytokine Netw 2021;32:83-8.
31. Gasparotto M, Framba V, Piovella C, Doria A, Iaccarino L. Post-COVID-19 Arthritis: A Case Report and Literature Review. Clin Rheumatol 2021;40:3357-62.
32. Roongta R, Ghosh A. Managing Rheumatoid Arthritis during COVID-19. Clin Rheumatol 2020;39:3237-44.
33. Bakılan F, Gökmen İG, Ortanca B, Uçan A, Eker Güvenç Ş, Şahin Mutlu F, *et al.* Musculoskeletal Symptoms and Related Factors in Postacute COVID-19 Patients. Int J Clin Pract 2021;75:e14734.
34. Hasan LK, Deadwiler B, Haratian A, Bolia IK, Weber AE, Petrigliano FA. Effects of COVID-19 on the Musculoskeletal System: Clinician's Guide. Orthop Res Rev 2021;13:141-50.
35. Wang CC, Chao JK, Chang YH, Chou CL, Kao CL. Care for Patients with Musculoskeletal Pain during the COVID-19 Pandemic: Physical Therapy and Rehabilitation Suggestions for Pain Management. J Chin Med Assoc 2020;83:822-4.
36. Silva Andrade B, Siqueira S, de Assis Soares WR, de Souza Rangel F, Santos NO, Dos Santos Freitas A, *et al.* Long-COVID and Post-COVID Health Complications: An Up-to-Date Review on Clinical Conditions and Their Possible Molecular Mechanisms. Viruses 2021;13:700.
37. Rahimi K. Guillain-Barre Syndrome during COVID-19 Pandemic: An Overview of the Reports. Neurol Sci 2020;41:3149-56.
38. Shehata GA, Lord KC, Grudzinski MC, Elsayed M, Abdelnaby R, Elshabrawy HA. Neurological Complications of COVID-19: Underlying Mechanisms and Management. Int J Mol Sci 2021;22:4081.
39. Bhagat D, Sefali P, Dikkatwar M, Radhika B. Evaluation of Maternal and Neonatal IgG and IgM SARS-COV-2 Antibodies, Transfer Ratios of Antibodies via Placenta, Immune Response in Neonates by IgG and IgM SARS-COV-2 Antibodies Detection. Int J Curr Pharm Res 2022;14:95-6.

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