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Review Article

Use of Individualized Risk Factors in Patients Battling from Long Term Covid Impediments: The Intact Potential of Mini Review

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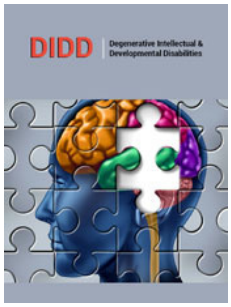

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Abstract

COVID-19 had a major impact on the world stage. COVID-19 has long-lasting effects on a small percentage of people globally, even though most of its victims recover in a few weeks these effects might persist for a few weeks or for several months. This page covers all aspects of COVID-19 side effects and highlights how homeopathy can help with them. This study focuses on several articles that highlight research on different post-Covid problems. Numerous papers that highlight the research on different post-Covid problems and the function of homeopathic remedies are the topic of this review. Angiotensin-converting enzyme 2 is the mechanism by which the severe acute respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infects host cells and causes COVID-19. Despite the fact that 80% of infections are minor or asymptomatic, people with moderate to severe COVID-19 experience a variety of symptoms, such as neurological, vascular and respiratory issues. The post-COVID situation of those who have recovered from the virus in terms of their physical, mental and sociocultural well-being poses a threat to the future of the planet, despite the encouraging fact that the number of recovered individuals exceeds the number of deaths. The post-recovery time and long-term consequences of COVID-19 are mostly unknown. Due to the fact that it has only been researched briefly and the concepts are currently in a state of flux. Using information from many study papers and the body of existing literature, this attempt aims to determine the extent and constraints of homoeopathy in post-COVID syndrome spans.

Keywords: Covid-19; Homoeopathy; Literature; Respiratory; Sars-CoV-2

Introduction

In December 2019, clusters of atypical pneumonia were reported in Wuhan, China, and were later identified as being caused by a novel coronavirus, now known as Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2). The disease caused by this virus was named Coronavirus Disease 2019 (COVID-19) and rapidly progressed into a global pandemic. The novel coronavirus was named severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2, 2019-nCoV) because of its high similarity (80%) to SARS-CoV, which caused Acute Respiratory Distress Syndrome (ARDS) and high mortality during 2002-2003 [1]. It was believed that a zoonotic transmission connected to a seafood market in Wuhan, China, was the first source of SARS-CoV-2. In the subsequent outbreak, it was eventually found that person-to-person transmission had a major role [2]. The sickness caused by this virus was named Coronavirus Disease 19 and a pandemic was declared.

Pathogenesis

Coronaviruses are positive single-stranded, enclosed, big RNA viruses that not only infect humans but a variety of other animals as well. Tyrell and Bynoe, who collected the

viruses from people with common colds, published the first description of coronaviruses in 1966 [3]. They were given the name "coronaviruses" (Latin: corona=crown) based on their morphology, which consisted of spherical virions with a core shell and surface projections resembling a solar corona. Alpha, beta, gamma and delta coronaviruses are the four subfamilies that make up this virus family. Gamma and delta viruses appear to have a different origin than alpha and beta coronaviruses, which are thought to have come from mammals, particularly bats. The beta- coronaviruses, one of the seven coronavirus subtypes that can infect people, can result in serious illness and even death, whereas the alpha-coronaviruses can produce asymptomatic or infections with quite minor symptoms. SARS-CoV2 is closely linked to SARS-CoV viruses [4,5] and the B lineage of beta-coronaviruses. The Membrane Glycoprotein (M), Small Membrane Protein (SM), Spike Protein (S) and Membrane Glycoprotein (M) are the four major structural genes, with an extra membrane Glycoprotein (HE) present in the HCoV-OC43 and HKU1 beta-coronaviruses [6].

Symptomatology

The most prevalent clinical symptoms in people with Coronavirus Disease 2019 (COVID-19) include fever, coughing, shortness of breath and other respiratory problems in addition to other non-specific symptoms such headache, dyspnea, fatigue and muscle pain [7,8]. In contrast to 81.3% of COVID-19 patients, 98-100% of SARS or MERS patients experienced fever [9]. Despite the fact that patients first experience a fever and/or respiratory symptoms, different degrees of lung abnormalities eventually emerge in all patients and these can be seen on chest CT (CT) [10]. Although diarrhea is present in approximately 20-25% of patients infected with MERS-Cov or SARS-Cov, intestinal symptoms have rarely been reported in patients with COVID-19 [11]. After more than two weeks, the lesions are gradually absorbed with residual frosted glass opacities and sub pleural parenchymal bands. In these patients who have recovered from COVID-19 pneumonia [12]. Furthermore, bilateral pneumonia and pleural effusion occurred more frequently in refractory individuals [13].

Post Covid Syndrome

Those who have contracted the COVID-19 virus are reporting a variety of symptoms. Symptoms persist for weeks or months after the COVID-19 disease has subsided. The term "post-COVID-19 syndrome" or "long COVID" is used to describe these symptoms. This syndrome is characterized by COVID-19 illness-like symptoms or by hangovers from treatment and hospitalization. These symptoms are being reported by many individuals with minor symptoms as well as asymptomatic patients, in addition to those with severe COVID- 19 sickness. The symptoms of post-COVID-19 syndrome range from minor ones like fatigue, fever and headache to more serious ones like autoimmune illnesses or multisystem inflammatory syndrome [14].

Post-Covid-19 syndrome clinical manifestations

Covid-19/long covid subacute: Long COVID or sub-acute COVID-19 is a term used to describe people who exhibit varied combinations of the following symptoms from week 4 to week 12 following their initial COVID-19 infection. The following are the long COVID-19 symptoms are extreme exhaustion, lack of appetite, mental dullness, trouble focusing, anxiety and depression, lack of sleep, headache, fever, loss of smell and taste, chest discomfort, joint pain, muscular pain, palpitations, coughing, dyspnea or shortness of breath, vertigo or dizziness, especially when standing [15].

Prominent Conditions in Post Covid Syndrome

Dyspnea

The Mount Sinai Hospital did a cross-sectional observational study titled "Post-acute COVID-19 syndrome severely impairs health and wellbeing despite. Dyspnea is discovered to be a common sign of acute COVID 19 infection, even in cases of less severe acute infection. In 60% of patients after COVID, it remained. According to this study, 87.4% of patients who had recovered from COVID19 reported that at least one symptom, particularly fatigue and dyspnea, persisted in their cases [16,17]. After recovering from COVID-19, out of 100 patients, 78 in an observational cohort study participants had abnormal cardiovascular MRI results (median, 71 days after diagnosis) and 36 of them complained dyspnea and unusual exhaustion [18]. According to recent research, COVID-19 primarily affects the lung, which exhibits pathologies like diffuse alveolar epithelium destruction, capillary damage/bleeding, hyaline membrane formation, alveolar septal fibrous proliferation and pulmonary consolidation in survivors who have been discharged from the hospital [19,20]. Treatment for interstitial lung disease frequently focuses on reducing inflammation because it can cause fibrosis in some types of the condition [21]. The inflammatory and hyper coagulable status of COVID-19 is associated with an elevated risk of thromboembolic events. Pulmonary fibrosis and pulmonary embolism must therefore be taken into account in post-COVID dyspnea [22].

Persistent cough

One of the most prevalent early symptoms of COVID-19, reported by 60-70% of symptomatic patients, is a dry cough [23,24]. Online polls have revealed that 20-30% of persons continue to have a dry cough 2-3 months after contracting COVID-19, according to a study published in The Lancet Respiratory Medicine [25-27]. The reflexive act of coughing happens when peripheral sensory nerves are activated and enter the vague nerves, which then send signals to the brainstem's solitary nucleus and spinal trigeminal nucleus [28]. There is a chance that SARS-CoV-2 will infect the sensory nerves that control coughing, causing neuro inflammation and neuro immune interactions as causes of cough hypersensitivity [29].

Fatigue

An individual's ability to perform to their normal capacity is hampered by fatigue, which is defined as "a subjective, unpleasant symptom that encompasses complete body emotions ranging from tiredness to exhaustion generating a relentless overall condition." 3 Patients experience exhaustion as both physical and mental, including weakening of memory, trouble concentrating

and problems with cognitive skills. Physical fatigue is the inability to execute physical activities as effectively as one used to before develop COVID [30]. What needs to be taken into account is the fact that exhaustion persisted for months following the initial virus attack. Chronic fatigue is defined as weariness that lasts longer than six months. Persistent fatigue is a common feature of long COVID and has a variety of underlying causes [31].

Autoimmunity

Autoimmunity has emerged as a characteristic of the Post-Covid Syndrome (PCS), which may be related to sex. Acute COVID-19 has been linked to the discovery of new-onset autoantibodies [32] and latent Poly Autoimmunity (PolyA) may have an impact on hospitalized patients' prognoses [33]. The final analysis showed 116IgG and 104IgM antibodies. Thyroglobulin IgG autoantibodies were present in more than 10% of the individuals. In between 5% and 10% of the patients, there were additional anti-cytokine IgG autoantibodies. IgM positivity was often low. Latent autoimmunity, which is defined as having just one IgG autoantibody and PolyA, which is defined as having two or more IgG autoantibodies, were discovered in 83% and 62% of patients, respectively. In >85% of patients, anti-SARSCoV-2 IgG antibodies were discovered. IgG anti-SARS-CoV-2 antibodies were linked with age and BMI. PCS is characterized by autoimmune and latent autoimmunity is associated with humoral response to SARS-CoV-2 [34].

Post-traumatic stress disorder

Post-Traumatic Stress Disorder (PTSD), an independent sequela of any traumatic incident, is the most anticipated and diagnosed condition of great public health concern as a result of the COVID-19 pandemic [35]. Numerous traumatic events that occurred during the pandemic, such as the psychological trauma of quarantine and isolation, the loss of loved ones, the morbid fear of contracting COVID-19, the stress of financial burden due to loss of employment coupled with fear of the future, domestic violence & sexual abuse in women due to home arrest during lockdowns and fear of social stigma, resulted in PTSD manifestations [36]. The incidence of PTSD was estimated to be 28.2% in a few recent surveys carried out during the first and second phases of the pandemic [37-39], confirming the prevalence reported by the studies done. One in four people subjected to a traumatic incident (natural or man-made) at some time in their lives have PTSD, which has a global frequency of 12-15% [40]. A recent systematic review conducted during the pandemic also confirmed the same [41]. It was found in a previous meta-analysis and a recent review that 17-44% of critical illness survivors, especially those who needed hospitalization or ICU care, reported clinically significant PTSD symptoms [42]. The cases were evaluated every three months for the CAPS-5 score and evaluated monthly for clinical progress. After six months, the outcome was evaluated clinically and objectively using the CAPS-5 score [43].

Mucormycosis

A rare but serious fungal infection known as Mucormycosis (formerly known as zygomycosis) is brought on by a class of molds called mucoromycetes. It is a potentially fatal illness that mostly affects immuno-compromised people, especially those with diabetes mellitus, haematological cancer, hematopoietic stem cell transplantation and solid organ transplantation [44]. Particularly in critically immunocompromised patients who are subjected to invasive emergency procedures like mechanical ventilation, Continuous Renal Replacement Therapy (CRRT), Extra Corporeal Membrane Oxygenation (ECMO), insufficient nursing ratios, prolonged hospital stays and breaches in asepsis, these events cause secondary bacterial and fungal infections [45]. Multiple cranial nerve palsies, unilateral periorbital facial pain, edema of the eyelids, orbital inflammation, blepharoptosis, proptosis, acute ocular motility changes, internal or external ophthalmoplegia, headache and acute vision loss are signs and symptoms suggestive of Mucormycosis in susceptible individuals [46]. It is not possible to diagnose Mucormycosis using circulating antigen detection tests, which are comparable to galactomannan detection for invasive aspergillus's. Because of this, diagnosis requires a biopsy of samples from clinically afflicted locations [47].

Hyperglycemia

Early research found that a group of COVID-19 people without a history of diabetes or a diagnosis of it had a greater prevalence of hyperglycemia. With a number of acute diseases and viral infections, the occurrence of moreover, new-onset diabetes following hospitalization has been recorded in the past [48]. It is unknown whether or not this diabetes will persist because there has been a limited amount of long-term follow-up on these patients. Because the exact mechanism and epidemiology of new-onset diabetes connected to COVID-19 are unknown, it is difficult to give care options for those who have the illness [49]. It has been found that, on the one hand, diabetes patients are more likely to experience a severe Covid-19 sickness and on the other hand, it has been noted that severe metabolic consequences of pre-existing diabetes, such as diabetic ketoacidosis and hyperosmolarity, require extraordinarily high doses of insulin in Covid-19-infected persons [50]. Moreover, compared to people with normal glucose levels, individuals with hyperglycemia had significantly bigger neutrophils, D-dimers and inflammatory markers [51]. Diabetes Warning Symptoms include Frequent urination, which may involve getting up in the middle of the night to go to the restroom or occurring more frequently during the day [52]. Although weariness is a common symptom, it can be difficult to differentiate between COVID-19 cases because most patients continue to experience it for a long period after their acute illness [53]. In clinical variations of type 2 diabetes [54,55]. The Pre-existing Diabetes with COVID-19 infection that is well-known during the COVID-19 infection, undiagnosed diabetes was discovered with pre-diabetes that developed into type 2 diabetes as a result of stress with complication 0f systemic illness or COVID-19 infection. Stress-and hormone-induced hyperglycemia WITH COVID-19-related new-onset diabetes mellitus.

Hematological complication

Micro and microvascular thrombotic problems have become frequent clinical sequelae of the COVID-19 pandemic, especially in critically unwell and hospitalized patients [56,57] Although

less common than venous thrombosis, the risk of pulmonary microvascular thrombosis and arterial thrombotic events (infarction, stroke, limb ischemia) is also elevated (up to 17% in a recent meta-analysis) 13-17. An immuno thrombotic condition known as COVID-19-Induced Coagulopathy (CIC) that appears to be more pro thrombotic than haemorrhagica [58]. In terms of pathogenesis, SARS-CoV-2 directly infects vascular endothelial cells and creates a favorable environment for immune cell migration and aggregation by inhibiting ACE-2 receptor activation and accumulating angiotensin II. 30,31 Endothelial damage, the production of pro-inflammatory and pro coagulant cytokines and the activation of the coagulation cascade all contribute to altered thrombin generation and hemostatic environment [59]. After the acute stage of the disease, it has been hypothesized that low-grade endothelial activation, together with subsequent downstream signaling of pro thrombotic pathways and low-level inflammation, may continue [60]. Whilst it is uncertain how long the hyper inflammatory state lasts, it is likely related to the severity and duration of the post-acute COVID-19 phase thrombotic complications risk. Long-term COVID-19 autoimmunity may also include B cells [61].

In one hospitalized cohort, antiphospholipid antibodies were found in 52% of patients (anti-phosphatidylserine/prothrombin IgG in 24%, anti-phosphatidylserine/prothrombin IgG in 18% and anti-cardio lipid IgM in 23%). There was a correlation between higher neutrophil activity and worse outcomes [60]. According to a recent study, SARS-CoV-2 can potentially disrupt the differentiation of hematopoietic stem cells, resulting in acute anemia of inflammation, thrombocytopenia and thrombosis. The cumulative incidence of thrombosis (segmental pulmonary embolism, intra cardiac thrombus, ischemic stroke and VTE) at 30 days was 2.5% in a single-center retrospective report of 163 patients who did not receive thrombin prophylaxis post discharge, 0.6% for VTE alone (median time to event: 23 days) and 3.7% for hemorrhage [62]. Four examples of catastrophic massive artery thrombose were documented by fet al.44. The patients had asymptomatic SARS-CoV-2 infection, were young, and had no known cardiovascular risk factors. There was a long latency (median time 78 days from seroconversion) between the positive serology and the incident. Three individuals suffered from large-vessel ischemic strokes, myocardial infarction and acute limb ischemia brought on by aortic emboli. All patients also exhibited a hyper-coagulable state, which was shown by considerably elevated levels of factor VIII, von Will brand factor antigen, D-dimer levels and hyperfibrinogenemia. The severity of the COVID-19 infection (ICU stay, length of hospital stays), as well as the usual risk factors for thrombosis and unfavorable outcomes, are linked to the probability of thrombotic problems in the post-acute context [63]. Although it occurs infrequently, autoimmunity Thrombocytopenic Purpura (ITP) has been identified as a COVID-19 late manifestation. Immune system dysregulation, molecular mimicry, epitope dissemination and susceptibility mutations in SOCS 1 are potential pathways driving SARS-CoV-2-mediated immunological thrombocytopenia. In the post-acute phase, between 3 and 4 weeks after the onset of COVID-19 symptoms, delayed-onset ITP has been documented. In a sizable cohort of 271 hospitalized COVID-19 patients in China, Chenet et al. observed that 11.8% of patients had delayed-phase thrombocytopenia with a presumed immunological etiology [64].

SIRT1 molecular mechanism

Sirtuin-1 (SIRT1) is a NAD^+-dependent deacetylase that regulates key cellular processes including inflammation, oxidative stress, mitochondrial biogenesis and cellular survival by de acetylating transcription factors and histones. SIRT1 suppresses pro-inflammatory signaling primarily by de acetylating the p65 subunit of NF-κB, thereby reducing the transcription of cytokines such as IL-6, TNF-α and IL-1β and attenuating chronic inflammation. SIRT1 also activates metabolic and stress-response regulators like PGC-1α and FOXO proteins, promoting mitochondrial function and resilience against oxidative damage. Under oxidative stress, NAD^+ depletion diminishes SIRT1 activity, leading to increased NF-κB activation, cellular senescence and endothelial dysfunction, which are relevant to the pathogenesis of chronic lung and systemic disease. Nutritional and pharmacological activators of SIRT1 (e.g., polyphenols) have been proposed to restore its activity, thereby improving inflammatory regulation and cellular metabolism in post-infectious and aging-related conditions [65].

Conclusion

Post-COVID Syndrome (PCS) has emerged as a significant global health concern, characterized by persistent multi-system symptoms that extend beyond the acute phase of SARS-CoV-2 infection. Respiratory compromise, chronic fatigue, neurocognitive disturbances, metabolic imbalance, thrombo-inflammatory tendencies and autoimmune manifestations collectively contribute to long-term morbidity and reduced quality of life. Despite increasing clinical recognition, the underlying pathophysiological mechanisms remain incompletely understood and definitive therapeutic strategies are still evolving. Recent scientific discussions highlight the role of immune dysregulation, endothelial dysfunction, mitochondrial stress and altered cellular repair pathways in PCS. In this context, molecular regulators such as Sirtuin-1 (SIRT1) have gained attention due to their involvement in inflammation control, metabolic balance and cellular resilience. Impaired SIRT1 activity may represent one of the biological links between viral injury and chronic post-infectious sequelae, opening avenues for integrative and supportive therapeutic approaches. From a homoeopathic perspective, PCS represents a dynamic and individualized post-infectious state rather than a uniform disease entity. The principles of individualization, totality of symptoms and holistic patient assessment may offer supportive value in addressing the diverse and fluctuating symptom patterns observed in PCS. While emerging clinical observations suggest potential benefits, rigorous, well-designed clinical studies are required to establish evidence-based roles, clarify mechanisms and define therapeutic boundaries. In summary, post-COVID syndrome demands a multidisciplinary and patient-centred approach. Integrating evolving biomedical insights with individualized therapeutic models may contribute to improved long-term recovery, provided that future research continues to emphasize scientific validation, safety and clinical relevance.

References

1. Ksiazek TG, Erdman D, Goldsmith CS, Zaki SR, Peret T, et al. (2003) A novel coronavirus associated with severe acute respiratory syndrome. N England J Med 348 (20): 1953-1966.
2. Sheeba S, Murugan M, Sankar AS (2023) Consequences of COVID-19: A wide range on efficacious action of homoeopathic medicines with stratagems tool aids: Literature research. International Journal of Homoeopathic Sciences 7(4): 460-462.
3. Zheng M, Gao Y, Wang G, Song G, Liu S, et al. (2020) Tian Functional exhaustion of antiviral lymphocytes in COVID-19 patients. Cell Molecule Immunology 17(5): 533-535.
4. Zhang J, Litvinova M, Wang W, Wang Y, Deng X, Chen X, et al. (2020) Evolving epidemiology and transmission dynamics of coronavirus disease 2019 outside Hubei province, China: A descriptive and modelling study. Lancet Infect Dis 20(7):793-802.
5. Coronavirus 2019-nCoV, CSSE. Coronavirus 2019-nCoV Global Cases by Johns Hopkins CSSE. Idealized study of long-term covid cases with precautions of complications.
6. Tyrrell DA, Bynoe ML (1966) Cultivation of viruses from a high proportion of patients with colds. Lancet 1(7428): 76-77.
7. GISAID Global Initiative on Sharing All Influenza Data. Phylogeny of SARS-like beta coronaviruses including Novel Coronavirus Sars (nCoV).
8. Zhou P, Yang XL, Wang XG, Hu B, Zhang L, et al. (2020) A pneumonia outbreak associated with a new coronavirus of probable bat origin. Nature 579(7798): 270-273.
9. Mo P, Xing Y, Xiao Y, Deng L, Zhao Q, et al. (2021) Clinical characteristics of refractory COVID-19 pneumonia in Wuhan, China. Clinical Infectious Diseases 3(11): e4208-e4213.
10. Sheeba S, Murugan M, Sankar AS (2025). Applications and challenges of nano medicine for COVID-19 outbreak: The potential relevance of therapeutic and diagnostic approaches for treatment. Nano Medicine & Nanotechnology 10(1): 000343.
11. Assiri A, Al-Tawfiq JA, Al-Rabeeah AA, Al-Rabiah FA, Al-Hajjar S, et al. (2013) Epidemiological, demographic and clinical characteristics of 47 cases of middle east respiratory syndrome coronavirus disease from Saudi Arabia: A descriptive study. The Lancet Infectious Diseases 13(9): 752-761.
12. Yin Y, Wunderink RG (2018) MERS, SARS and other coronaviruses as causes of pneumonia. Respiratory 23(2): 130-137.
13. Wang D, Hu B, Hu C, Zhu F, Liu X, et al. (2020) Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus–infected pneumonia in Wuhan, China. JAMA 323(11): 1061-1069.
14. Sahin AR, Erdogan A, Agaoglu PM, Dineri Y, Cakirci AY, et al. (2020) 2019 novel coronavirus (COVID-19) outbreak: A review of the current literature. Eurasian Journal of Medicine and Oncology 4(1): 1-7.
15. Pan F, Ye T, Sun P, et al. (2020) Time course of lung changes on chest CT during recovery from 2019 novel coronavirus (COVID-19) pneumonia. Radiology 295(3): 200370.
16. Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, et al. (2020) Clinical characteristics of 2019 novel coronavirus infection in China. N Endl J Med 382(18): 1708-1720.
17. Center for Disease Control and Prevention (2021) Long term effects of COVID-19.
18. Tabacof L, Tosto-Mancuso J, Wood J, Cortes M, Kontorovich A, McCarthy D, et al. (2020) Post-acute COVID19 syndrome negatively impacts health and wellbeing despite less severe acute infection.
19. Carfì A, Bernabei R, Landi F (2020) Gemelli against COVID19 Post-Acute Care Study Group. Persistent symptoms in patients after acute COVID-19. JAMA 324(6): 603-605.
20. Puntmann VO, Carerj ML, Wieters I, Fahim M, Arendt C, et al. (2020) Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID19). JAMA Cardiology 5(11): 1265-1273.
21. Lakshmi LG, Elias HS, Dhini S (2021) Post COVID dyspnea and scope of homoeopathy. International Jornal of Homoeopathic Science 5(1): 129-131.
22. Fraser E (2020) Long term respiratory complications of COVID-19. The BMJ 370: m3001.
23. Sheeba S, Murugan M, Sankar AS, Gopukumar ST, Reshma R, et al. (2024) Covid-19 and homeopathy: Systematic review and meta-analysis of clinical outcomes. Journal of Natural & Ayurvedic Medicine 8(4): 000453.
24. Lakshmi G (2021) Persistent cough after Covid 19 pathophysiology and homoeopathic management. International Journal of Homoeopathic Science 5(3): 221-223.
25. Lancet Respiratory Medicine with complication and recurrent infections (2021) The history of positive cases 9: 53344.
26. Canning BJ, Chang AB, Bolser DC, Smith JA, Mazzone SB, et al. (2014) Anatomy and neurophysiology of cough: Chest Guideline and Expert Panel report. Chest 146(6): 1633-1648.
27. Acosta-Ampudia Y, Monsalve DM, Rojas M, Rodríguez Y, Zapata E, et al. (2022) Persistent autoimmune activation and proin-fammatory state in post-COVID syndrome. J Infect Dis 225(12): 2155-2162.
28. Chang SE, Feng A, Meng W, Apostolidis SA, Mack E, et al. (2021) New-onset IgG autoantibodies in hospitalized patients with COVID-19. National Community 12(1): 5417.
29. Anaya JM, Monsalve DM, Rojas M, Rodríguez Y, Montoya-García N, et al. Latent rheumatic, thyroid and phospholipid autoimmunity in hospitalized patients with COVID-19. J Transl Autoimmunity 4: 100091.
30. Sheeba S (2024) Innovative advances in homeopathy: Based on nano particles exploration. Nano Medicine & Nanotechnology Open Access 9(4): 1-5.
31. Rojas M, Rodríguez Y, Acosta-Ampudia Y, Monsalve DM, Zhu C, et al. (2022) Autoimmunity is a hallmark of post- COVID syndrome with other systemic disorders especially in old age cases. Journal of Medicine 20(1): 129.
32. Brooks SK, Webster RK, Smith LE, Woodland L, Wessely S, et al. (2020) The psychological impact of quarantine and how to reduce it: Rapid review of the evidence. The Lancet 395(10227): 912-920.
33. Cao C, Wang L, Fang R, Liu P, Bi Y, et al. (2022) Anxiety, depression, and PTSD symptoms among high school students in China in response to the COVID-19 pandemic and lockdown. Journal of Affected Disorders with specific 296: 126-129.
34. Forneris CA, Gartlehner G, Brownley KA, Gaynes BN, Sonis J, et al. (2013) Interventions to prevent post-traumatic stress disorder: A systematic review. American Journal of Preventive Medicine 44(6): 635-650.
35. Giannopoulou I, Galinaki S, Kollintza E, Adamakiv M, Kympouropoulos S, et al. (2021) COVID-19 and post-traumatic stress disorder: The perfect 'storm' for mental health (Review). Experience of Theoretical Medicine.;22(4):1162.
36. Hardy KV, MueserKT (2017) Editorial: Trauma, psychosis and posttraumatic stress disorder. Front Psychiatry 8: 220.
37. Herring C (1991) The guiding symptoms of our materia medica. B Jain Publishers, New Delhi, India.
38. Hompath Zomeo Ultimate software (2020) Mind Technologies Pvt. Ltd, Lan 13(7):
39. Jonathan C, Ipser DJ (2012) Stein, evidence-based pharmacotherapy of Post-Traumatic Stress Disorder (PTSD). International Journal of Neuropsychopharmacology 15(6): 825-840.

40. Sheeba S, Murugan M, Sankar AS (2024) Analyzing the individualized homoeopathic treatment of post-COVID syndrome with effectiveness of calcarea carbonica 200 potency and determination of IgG level. Chinese Journal of Evidence-Based Medicine 20: (1).

41. Jeong W, Keighley C, Wolfe R, Lee WL, Slavin MA, et al. (2019) The epidemiology and clinical manifestations of mucor mycosis: A systematic review and meta-analysis of case reports. Clinical Microbiology Infect 25(1): 26-34.

42. Soman R, Sunavala A (2021) Post COVID-19 Mucormycosis-from the frying pan into the fire. J Assoc Phys India 69(1): 13-14.

43. Petrikkos G, Skiada A, Lortholary O, Roilides E, Walsh TJ, et al. (2012) Epidemiology and clinical manifestations of mucormycosis. Clinical. Infectious Disease 54(Suppl 1): S23-S34.

44. Sheeba S, Murugan M, Sankar AS, Sylum VS, Gopukumar ST (2022) A clinical case study of treating post covid syndrome by administrating calcarea carbonica 200 potency with assessment of IgG level. Nano Medicine & Nanotechnology Open Access 7(2): 1-5.

45. Manjusha Nambiar, Sudhir Rama Varma, Marah Damdoum (2021) Post-Covid alliance with complications mucormycosis, A fatal sequel to the pandemic in India. Saudi Journal of Biological Sciences 28(11): 6461-6464.

46. Sanyaolu A, Okorie C, Marinkovic A, Patidar R, Younis K, et al. (2020) Comorbidity and its impact on patients with COVID-19. Sn ComprClin Med 2(8): 1069-1076.

47. Khunti K, Prato SD, Mathieu C, Kahn SE, Gabbay RA, et al. (2021) COVID-19, Hyperglycemia and new onset diabetes. Diabetes Care 44(12): 2645-2655.

48. New-onset diabetes in Covid-19 (2020) NEJM 383(8): 789-790.

49. Coppelli A, Giannarelli R, Aragona M, Penno G, Falcone M, et al. (2020) Hyper glycaemia at Hospital Admission Is Associated with Severity of the Prognosis in Patients Hospitalized for COVID-19: The Pisa COVID-19 Study. Diabetes Care 43(10): 2345-2348.

50. Pardhan S, Islam MDS, López-Sánchez GF, Upadhyaya T, Sapkota RP (2021) Self-isolation negatively impacts self-management of diabetes during the coronavirus (COVID-19) pandemic. Diabetic Metabolic Syndrome 13(1): 123.

51. Joensen LE, Madsen KP, Holm L, Nielsen KA, Rod MH, et al. (2020) Diabetes and COVID-19: Psychosocial consequences of the COVID-19 pandemic in people with diabetes in Denmark what characterizes people with high levels of COVID-19-related worries? Diabetology Med 37(7):1146-1154.

52. Sheeba S, Gopukumar ST, Ramya S, Sanju S, Reghu R, et al. (2024) Exploring the nexus of nanoparticles and homoeopathic potentized medicine: A narrative review. Nano Medicine & Nanotechnology Open Access 9(2): 000309.

53. Piazza G, Campia U, Hurwitz S, Snyder JE, Rizzo SM, et al. (2020) Registry of arterial and venous thromboembolic complications in patients with COVID-19. Journal of American Collection of Cardiology Cases 76(18): 2060-2072.

54. Sheeba S, Gokul Krishna K, Serine SS, Sudha V, Chinchu B, et al. (2024) A clinical study to evaluate the effect of arsenicum album 200c in Covid patients and FTIR characteristic analysis. Journal of Neonatal Surgery 13: 764-769.

55. Pillai P, Joseph JP, Fadzillah NHM, Mahmod M (2021) COVID-19 and major organ thromboembolism: Manifestations in neurovascular and cardio-vascular systems'. Stroke Cerebral Vascular Disorders 30(1): 105427.

56. Vernuccio F, Lombardo FP, Cannella R, Panzuto F, Giambelluca D, et al. (2020) Thromboembolic complications of COVID-19: the combined effect of a pro-coagulant pattern and an endothelial thrombin-inflammatory syndrome. Clinical Radiology 75(11): 804-810.

57. Vinayagam S, Sattu K (2020) SARS-CoV-2 and coagulation disorders in different organs. Life Science 260: 118431.

58. Andrade BS, Siqueira S, Assis Soares WR, Rangel FS, Santos NO, et al. (2021) Long-COVID and post-COVID health complications: An up-to-date review on clinical conditions and their possible molecular mechanisms. Viruses 13(4): 700.

59. Sheeba S, Murugan M, Suman Sankar AS (2024) Exploring the shadows of recovery: A systematic narrative review of homoeopathy in post-covid syndrome. Journal of Neonatal Surgery 13: 758-763.

60. Patell R, Bogue T, Koshy A, Bindal P, Merrill M, et al. (2020) Post discharge thrombosis and hemorrhage in patients with COVID-19. Blood 136(11): 1342-1346.

61. Roberts LN, Whyte MB, Georgiou L, Giron G, Czuprynska J, et al. (2020) Post discharge venous thromboembolism following hospital admission with COVID-19. Blood 136(11): 1347-1350.

62. Engelen MM, Vandenbriele C, Balthazar T, Claeys E, Gunst J, et al. (2021) Venous thrombus embolism in patients discharged after COVID-19 hospitalization. Thrombosis Hemostasis 47(4): 362-371.

63. Bourguignon A, Beaulieu C, Belkaid W, Desilets A, Blais N (2020) Incidence of thrombotic outcomes for patients hospitalized and discharged afterCOVID-19 infection. Thrombosis of Respiratory Note 196: 491-493.

64. Enzymatic activity of sirtuins and NAD biosynthesis by using proper method in most advanced level.

65. Huang C, Huang L, Wang Y, Li X, Ren L, et al. (2021) 6-month consequences of COVID-19 in patients discharged from hospital: A cohort study. Lancet 97(10270): 220-232.