REVIEW STUDY | UHD JOURNAL OF SCIENCE AND TECHNOLOGY

# Combining Antibodies, COVID-19, and Neuroscience: Review Study

**Dimah Shaban[1], Muzhir Shaban Al-Ani[2], Wasfi Kahwachi[3]**

*[1]Department of Life Sciences, University of Coimbra, Faculty of Science and Technology, Coimbra, Portugal, [2]Department of Information Technology, University of Human Development, College of Science and Technology, Sulaymaniyah, KRG, Iraq, [3]Department of Dentistry, Faculty of Dentistry, Tishk International University, Erbil, Iraq*

## ABSTRACT

Emphasis on neuroscience, this research examines antibodies in Coronavirus-19 (COVID-19). We used neuroscience as a foundational field to begin our investigation. On February 10, 2024, this search was conducted using the ScienceDirect database. 552,211 papers with the keyword "neuroscience" were found. Next, we found 309,196 papers using the COVID-19 keyword and 106,473 publications using the antibodies keyword. Furthermore, we got 9039 papers by combining COVID-19 and neuroscience, and 3576 publications by combining antibodies and COVID-19. Last but not least, the investigation will concentrate on the 243 papers; we acquired by integrating antibodies, COVID-19, and neuroscience. Two hundred and forty-three publications make it through many filtering stages, ultimately yielding 41 publications.

**Index Terms:** Antibodies, Autoantibodies, Neuroscience, Coronavirus-19, Coronavirus

## 1. INTRODUCTION

Coronavirus (COVID-19) is a respiratory disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus [1], [2]. The World Health Organization declared this a pandemic in March 2020 due to the virus spreading around the world very quickly [3], [4]. The latest statistics in October 2024 showed that the number of cases of coronavirus worldwide about 770 million [5], [6]. In addition, the number of deaths related to coronavirus exceeded 6.96 million [7], [8]. Some countries are still recording new cases and deaths, especially with the emergence of new mutants, despite the relative control of the virus thanks to extensive vaccination [9], [10]. COVID-19 begins to enter the human body, it starts attacking the respiratory system; then, tissues start falling in the lung [11], [12]. Hence, the immune system starts reacting, causing damage to the healthy tissues in the lung and leading to difficulties in breathing [13], [14].

Autoantibodies are immune system proteins that mistakenly attack the body's own tissues instead of foreign invaders such as bacteria and viruses [15], [16]. Normally, the immune system produces antibodies to recognize and treat pathogens [17]. Sometimes, the immune system malfunctions and produces autoantibodies, which can lead to autoimmune diseases [18], [19]. The function of autoantibodies is to attach to the body's cells and tissues, mistakenly marking them for destruction by the immune system [20]. This leads to inflammation and damage to the organs of the body [21]. There are many types of autoantibodies: Thyroid antibodies (TA), antinuclear antibodies (ANA), and TA [22], [23]. In addition, research has shown that COVID-19 can trigger the production of autoantibodies in some patients, which can worsen disease outcomes [24]. Research has found that COVID-19 can lead to the production of autoantibodies in some patients, which may lead to deterioration of disease outcomes [25].

Access this article online

DOI:10.21928/uhdjst.v8n2y2024.pp111-123 | E-ISSN: 2521-4217 | P-ISSN: 2521-4209



**Corresponding author's e-mail:** Muzhir Shaban Al-Ani, Department of Information Technology, University of Human Development, College of Science and Technology, Erbil, Iraq. E-mail: muzhir.al-ani@uhd.edu.iq

Received: 10-11-2024 Accepted: 14-12-2024 Published: 29-12-2024

Neuroscience is the scientific discipline of the nervous system knowledge [26]. It is a multidisciplinary science that integrates biology, chemistry, medicine, physics, cytology, psychology, anatomy, statistics, and computer science to understand the function and structure of the nervous system and the disorders it causes [27]. It is concerned with the study of the brain, spinal cord, and networks of nerve cells that control all behavior and functions of the body [28]. Recently, scientific studies of the nervous system have increased dramatically [29]. This is due to great advances in electrophysiology, molecular biology, and computational neuroscience [30]. This has led scientists and researchers to study the nervous system in detail: how it works, how it is built, how it breaks down, and how it is modified and developed, in addition, the introduction of generative artificial intelligence leading to big data neuroscience [31].

## 2. METHODS AND MATERIALS

### 2.1. Study Publications Distribution Based on Years

The Corona pandemic began in December 2019, so research on this topic was limited after that, i.e. in 2020, and continued to increase gradually. Combining Autoantibodies, COVID-19, and Neuroscience as keywords in our search on Science Direct database to get 243 articles. The period is between 2020 and 2025. The publication rates in the 5 years are 2020 (2.8%), 2021 (22.2%), 2022 (24.7%), 2023 (30.5%), 2024 (18.9%), and 2025 (0.9%) as shown in Fig. 1. The year 2020 has a lower rate because COVID-19 just started; then, the other years increased gradually.

### 2.2. Study Publications Distribution Based on Article Type

According to the classifying based on the type of research, we notice that there are different types of publications as demonstrated in Fig. 2. The effective types of publications are review articles (28%), research articles (23.4%), book chapters (11.5%), conference abstracts (10.3%), case reports (4.2%), and correspondence (4.2%) in addition to others (9.5%). Hence, the most effective publication types are review articles (28%) and research articles (23.4%), in addition, the main aim focusing on the research articles to be our guided in the research.

### 2.3. Study Publications Distribution Based on Article Subject Area

According to the classifying based on the article subject area, we notice that there are different types of subject areas publications. These publications are sorted starting from higher rate as demonstrated in Fig. 3. The higher rates of publications according to the subject area are focused on the first four areas: Medicine and dentistry (41%), neuroscience

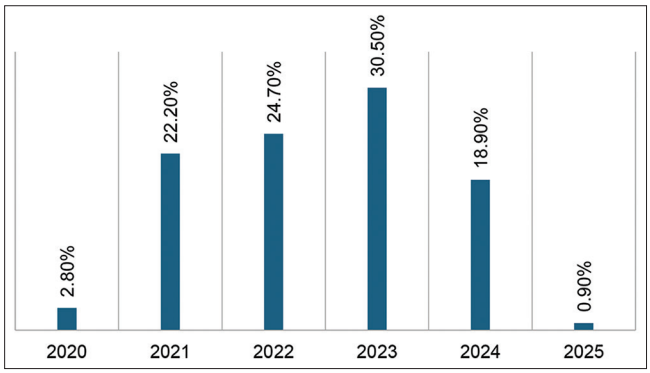


**Fig. 1.** Publications distribution based on years.

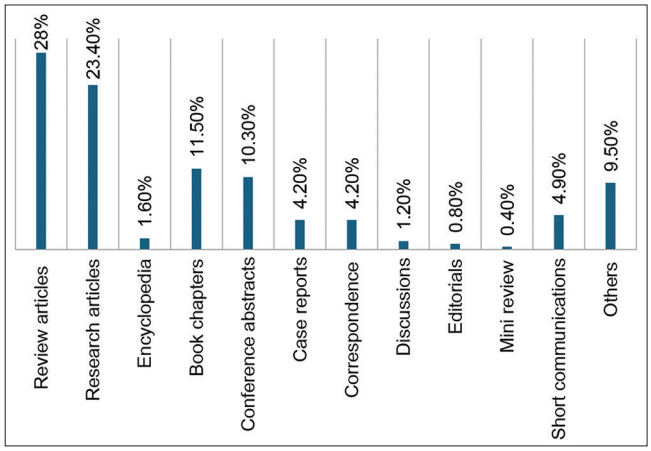


**Fig. 2.** Publications distribution based on article type.

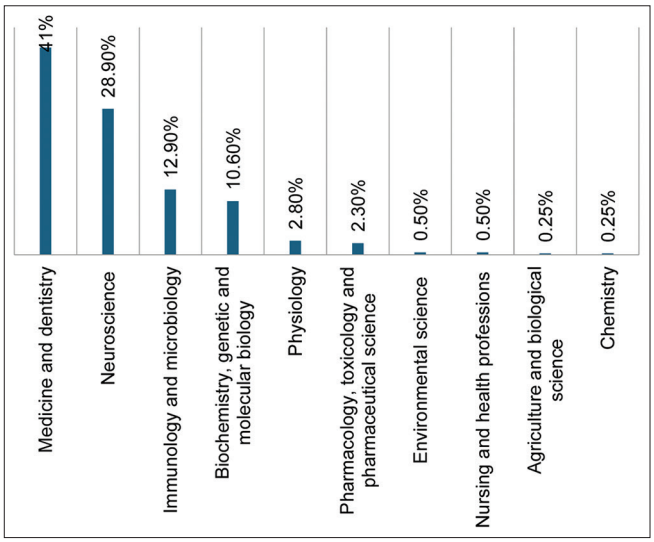


**Fig. 3.** Publications distribution based on article subject area.

(28.9%), immunology and microbiology (12.9%), and biochemistry, genetic and molecular biology (10.6%), combining all these four areas together leading to 93.4% of publications that are dominant most of publications.

## 3. RESULTS

A total of 243 publications presented as a literature search applied on ScienceDirect database on October 2, 2024 as demonstrated in Fig. 4. One hundred and nine redundancies and duplications have been found, so, the considered fields are 134 publications. Excluded reports and abstract papers of 25 publications, leading to 109 publications as effective fields. Excluded 16 publications as review papers, leading to 93 publications to be passing to the screening procedure. The first screening operation focused on the paper title, in which 35 papers are excluded as non-relevant items to get 58 papers. The second screening operation focused on the paper abstract, in which 17 papers are excluded as non-relevant items to get 41 papers as a final result. These 40 studies are the ones that are analyzed carefully so that we can reach a table to compare these studies.

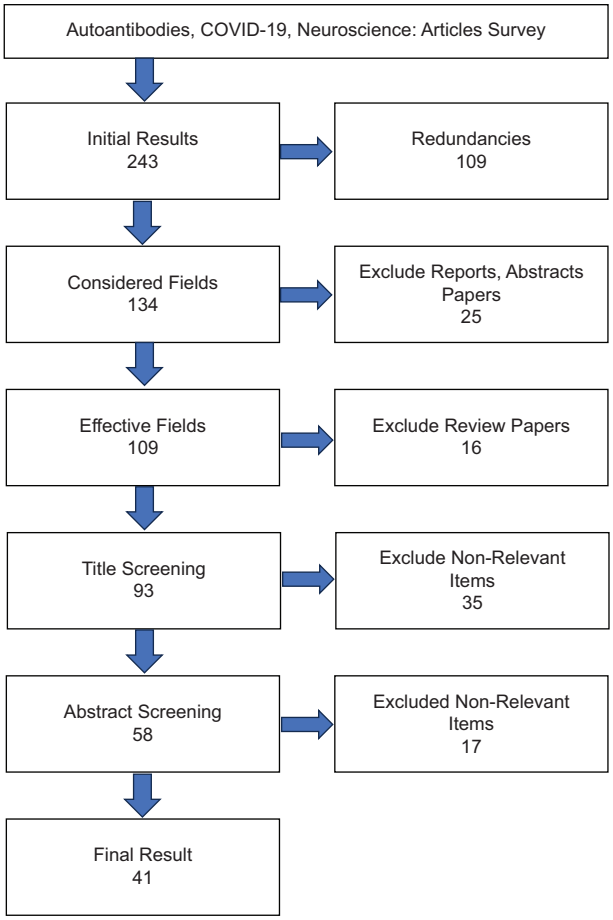


**Fig. 4.** Flow diagram of reporting systematic review.

## 4. ANALYSIS AND DISCUSSION

Many cleaning steps are implemented in the publications to get the final results of 41 publications as shown in Table 1.

Morris *et al.* (2020) [32] demonstrated Toll-like receptors on endosomes can be inhibited using pharmacological agents that increase endosomal pH, thereby reducing the activity of acid-dependent endosomal proteases required for their activity. This in turn leads to decreased autoantibody secretion, decreased nuclear factor kappa B activity, and decreased production of pro-inflammatory cytokines, that is, suppression of antigen-presenting cell activity. In addition, SARS-CoV-2 inhibits autophagy, rendering infected cells susceptible to apoptosis. Therefore, it is also proposed that further pharmacological inhibition of autophagy may promote apoptotic clearance of SARS-CoV-2-infected cells.

Vaschetto *et al.* (2020) [33] described a panel of common autoantibodies, including extractable nuclear antigen, ANA, antineutrophil cytoplasmic antibodies, and anti-double-stranded DNA antibodies, was negative. Only a slight alteration of lupus anticoagulant was detected, but without detection of anticardiolipin or anti-Beta2 glycoprotein, suggesting false positivity, as expected in critical care patients. Based on these laboratory results and without a history of systemic autoimmune disease, central nervous system (CNS) vasculitis was suspected.

Hu *et al.* (2020) [34] explained that coronavirus outbreak began in December 2019 and its symptoms include shortness of breath, cough, and fever, as well as CNS symptoms, especially in those with severe illness. The focus has been on how the virus reaches the CNS and what it will do. Some SARS-CoV-2 diseases share the same receptor, angiotensin-converting enzyme 2, which is present in the brain during infection. An abnormal immune response in the CNS and a direct attack by SARS will contribute to the disease.

Bertin *et al.* (2021) [35] followed-up of a patient of 58-year-old woman after recovery from COVID-19, who presented with new and persistent symptoms including neurological complications that could be explained by post-eclampsia syndrome. This is because they are independent factors associated with the severity of COVID-19 and the

**Table 1: Distribution of the publications**

| ID | Author | Year | No. of cases | Study design | Conclusion |
|---|---|---|---|---|---|
| [32] | Morris *et al*. | 2020 | large meta-analyses, involving almost 50,000 patients | Representing pathophysiology of acute respiratory distress syndrome. | Hydroxychloroquine is an effective immunomodulator in chronic use, but its slow in action that is usefulness as an immunomodulator in the treatment of COVID-19. |
| [33] | Vaschetto *et al*. | 2020 | 749 Covid-19 positive patients | Description of a patient with COVID-19 acute respiratory distress syndrome and SARS-CoV-2 vasculitis. | Clinical improvement after combined steroids suggests a diagnosis of central nervous system vasculitis caused by SARS-CoV-2 infection. |
| [34] | Hu *et al*. | 2020 | Different cases. | Discussing how coronavirus reached the central nervous system. In addition, the older age has critical conditions. | The possibility of the coronavirus attacking the central nervous system lies in the weakness of the immune response, infection of immune cells, and a malfunction of the cytokine/ chemokine system. |
| [35] | Bertin *et al*. | 2021 | One case 58-year-old female. | Follow-up of a patient after recovery from COVID-19, who presented with new and persistent symptoms including neurological complications that could be explained by post-eclampsia syndrome. | Neurological complications have been reported that cannot be explained by any cause other than denoted post-COVID syndrome. One year after infection, low-grade inflammation was present, and IgG aCL positivity and eosinophilia were observed. |
| [36] | Song *et al*. | 2021 | Antibodies in cerebrospinal fluid (*n*=5) as well as in blood (*n*=4). | Monitoring common neurological symptoms in individuals with severe COVID-19 compared to individuals without COVID-19 who also have neurological symptoms. | SARS antibodies within the spinal cord are not elicited by lung infection and are only present during brain infection with the virus. All individuals with COVID-19 have antibodies in their cerebrospinal fluid. |
| [37] | Dutta *et al*. | 2021 | One case 51-year-old male | Followed-up of a 51-year-old male with migraine headache over a period of 14 days. All complete blood count, blood sugar, lipid profile, renal function tests, and liver function tests were normal. | It should be noted that the benefits of receiving any of the approved COVID-19 vaccines far outweigh the potential risks of not taking the vaccine. |
| [38] | Chakraborty *et al*. | 2021 | Eight cases with COVID-19 associated Guillain-Barre syndrome. | A monitoring study was conducted among eight patients with Guillain-Barré syndrome after infection. | Guillain-Barré syndrome must be differentiated from critical illness and neuropathy in patients with COVID-19. |
| [39] | Metya *et al*. | 2021 | Presented a patient of 54 years old female. | Applied several neurological manifestations associated with COVID-19. | Imaging test revealed MRI-negative radicular myelopathy after COVID-19. |
| [40] | Elrashdy *et al*. | 2021 | Many cases after COVID-19 vaccination | Some aspects of COVID-19 and the main risk factors associated with the disease have been studied. | CCOVID-19 vaccines based on lipid nanoparticles cause rare cases of deep vein thrombosis. |
| [41] | Pandey *et al*. | 2021 | 14 Potential COVID-19 vaccine candidates | The impact of COVID-19 on the central nervous system has been studied by identifying the direct and indirect factors and influences that contribute to the deterioration of people's mental health. | All agencies, especially those responsible for decision-making, as well as all health-related agencies, the World Health Organization, and others, should work to improve the social determinants of mental health and amend all procedures that have gone wrong. |

(*Contd...*)

**Table 1: (*Continued*)**

| ID | Author | Year | No. of cases | Study design | Conclusion |
|---|---|---|---|---|---|
| [42] | Benjamin *et al*. | 2021 | 106 adult patients | This study covered 106 adult patients: 47 non-neurological COVID-hospitalized controls, 30 hospitalized COVID-neurological, and 29 COVID-non-hospitalized controls, recorded between March and July 2020. | High antibodies in the non-neurological COVID group (76.6%) and the neurological COVID group (73.3%), but low antibodies in the non-neurological COVID group (48.2%). |
| [43] | Hampshirea *et al*. | 2021 | 81,337 participants | Study of the relationship between cross-sectional cognitive Performance data from 81,337 participants who underwent an IQ test assessment. | COVID-19 infection causes cognitive deficits that persist beyond recovery. The observed deficits varied in magnitude with the severity of respiratory symptoms. |
| [44] | Lin *et al*. | 2021 | About 6 million cases in the USA as of September 2020, with children's cases up 8.4%. | Neurological conditions seen in the context of COVID-19, as well as the long-term effects of COVID-19 in the pediatric population, were studied through the literature and primary data collected at Morgan Stanley Children's Hospital. | It appears from the study that the Corona virus affects adults and children differently. This difference is due to the lack of associated diseases in children, in addition to the continuous development of the child's nervous system. |
| [45] | Evans *et al*. | 2021 | 830 participants were followed during the process. | Post-hospital coronavirus study of adults discharged from hospital in the UK with a clinical diagnosis of coronavirus, including assessment between 2 and 7 months after released. | After followed-up, only 239 participants felt fully recovered, 158 had a new disability. |
| [46] | Dobrindt *et al*. | 2021 | Not mentioned | To explore the host genes of SARS-CoV-2, human induced pluripotent stem cell -based models and CRISPR engineering were applied. | Analyses suggest that although SARS-CoV-2 neuro infection can occur in response to a high multiplicity of infection *in vitro*, it may occur less frequently under clinically relevant conditions. |
| [47] | Algahtani *et al*. | 2022 | A 38-year-old male | Description of a case of acute cervical dystonia that occurred after the first dose of the coronavirus vaccine BNT162b2. | The contribution leading to the occurrence of cervical dystonia caused by the vaccine due to the temporal relationship. |
| [48] | Sukocheva *et al*. | 2022 | 22 groups of study | Studied the progression of symptoms in patients after COVID-19 infection and compared with myalgic encephalitis/chronic fatigue syndrome. | The absence of effective treatment after infection with the Corona virus is not clear, and therefore cases cannot be targeted correctly until a correct and clear mechanism is established and confirmed. |
| [49] | Butler *et al*. | 2022 | Not mentioned | Following up on researchers' suggestions on post-COVID-19 side effects. | The study has shown that many COVID-19 patients will suffer from psychological and neurological complications. |
| [50] | Das *et al*. | 2022 | Two index cases of young patient | Studied a young patient with two conditions, ischemic attack and stroke. | Arterial stroke has been reported after COVID-19 vaccination. However, this is rare and when vasculopathy exists, may not be so rare. |

(*Contd...*)

**Table 1: (*Continued*)**

| ID | Author | Year | No. of cases | Study design | Conclusion |
|---|---|---|---|---|---|
| [51] | Vanderheiden and Klein | 2022 | Not mentioned | Presentation of Neuropathology in COVID-19 patients and its immunological causes. | Later complications of SARS-CoV-2 cause cognitive impairment, although there is no evidence of a productive CNS infection. |
| [52] | Reis *et al*. | 2022 | Not mentioned | Studying the impact of the novel coronavirus on neurological components, sleep and wakefulness. | There is a need to increase awareness and prepare for future challenges. |
| [53] | Wild *et al*. | 2022 | 478 volunteers in the COVID+group. | Studied 478 COVID-19 survivors and tested 30 days after infection. | After COVID-19 infection they showed significant cognitive impairment. |
| [54] | Sadeghizadeh *et al*. | 2022 | Two cases of pediatric COVID-19 | Studied two cases of children with COVID-19 with severe multisystem inflammatory syndrome. | In cases of headache with neurological signs, RCVS should be considered an important diagnosis. |
| [55] | Aricò *et al*. | 2022 | 12 literatures of COVID-19 patients. | During the COVID-19 pandemic, the speed of development of vaccines and specific drugs against emerging viruses has been observed. | The progression of the disease in many patients is attributed to insufficient type I interferon activity. |
| [56] | Rastogi *et al*. | 2022 | A 59-year-old woman post COVID-19 vaccine. | Study of the first case after COVID-19 vaccine with untreated encephalitis, MRI performed in conjunction with clinical improvement. | The result indicated no edema on MRI, but excising some loss of blood-brain barrier. |
| [57] | Aubart *et al*. | 2022 | 21 children with central nervous system inflammatory. | The selected patients tested positive for circulating antibodies against SARS-CoV-2. | SARS2-CoV-2 is a new agent for post-infectious central nervous system disease in children. |
| [58] | Monje and Iwasaki | 2022 | Not mentioned | The mechanisms neurobiological underpinnings affecting COVID-19 survivors were discussed explained. | Covid affected the quality of life and the inability to cope have led to neurocognitive and neuropsychiatric problems. |
| [59] | Aschman *et al*. | 2022 | Few (usually not more than four) cases. | Study the effects of SARS-CoV-2 on to the human brain. | Focusing on the host immune response to understand the neurological symptoms associated with SARS-CoV-2 disease, as there is no evidence of intact SARS-CoV-2 particles in the central nervous system. |
| [60] | Koh *et al*. | 2022 | A 45-year-old gentleman | Study the case of 45-year-old gentleman after 31 days of vaccination have some features of meningoencephalitis | After injection of methylprednisolone and globulins, clinical improvement occurred. |
| [61] | Lambe *et al*. | 2022 | Nine patients diagnosed with MOGAD | Study of nine patients diagnosed with MOGAD in the post-SARS-CoV-2 period. | These 9 cases highlight a potential role of infection in the immunopathogenesis of MOGAD. |
| [62] | Jammoul *et al*. | 2023 | Follow-up eight cases | Follow-up of long-term COVID patients to detect neurological manifestations that persist after infection. | The study showed that COVID patients develop post-COVID autonomic dystonia symptoms, and these symptoms are unknown. |
| [63] | Ulucay *et al*. | 2023 | Not mentioned | Study the effects of mRNA COVID-19 vaccines on the patients. | mRNA COVID-19 vaccines are effective in reducing the severity of COVID-19 and its complications. |

(*Contd*...)

**Table 1: (*Continued*)**

| ID | Author | Year | No. of cases | Study design | Conclusion |
|---|---|---|---|---|---|
| [64] | Takenaka and Nakamori | 2023 | One case of a 25-year-old. | Study A case of a 25-year-old who developed acute encephalopathy 2 days after the second dose of the COVID-19 vaccine | He was given intravenous methylprednisolone after being diagnosed with autoimmune encephalitis. |
| [65] | Sarkanen *et al*. | 2023 | Study population (18,785). | Applied the COVID-19 International Sleep Study questionnaire. | The study found an increase in EDS, EQS, ETFS. |
| [66] | Hira and Baker | 2023 | Participants=126. | Studied patients after several months recovery from COVID-19. | Patients with severe complications after COVID-19 have evidence of autonomic cardiovascular abnormalities. |
| [67] | Chauvin *et al*. | 2023 | 69 COVID-19 patients. | The study was conducted in 69 COVID-19 patients. | They extracted that Plasma from treated and untreated patients infected with the original variant showed strong neutralizing activity against the original virus. |
| [68] | Seeck and Tankisi | 2023 | Not mentioned | Studied the mutations of the original COVID virus. | COVID disease also affects both the central and peripheral systems. |
| [69] | Khoo *et al*. | 2023 | Not mentioned | Study the effects of COVID on children and adults under the same disease severity, | Children infected with COVID-19 are less severely affected than adults. |
| [70] | Ebrahimi *et al*. | 2023 | Twelve cases of development Multiple sclerosis. | Studied different vaccines and their relationship with demyelinating disorders in the central nervous system. | Common drugs were teriflunomide, natalizumab, rituximab and dimethyl fumarate. |
| [71] | Chaumont *et al*. | 2023 | Data from 24 Neuro- COVID adults. | Studied of 24 Neuro-COVID patients, seven (29%) had meningoencephalitis and 17 (71%) had encephalopathy. | CSF Aß1-42 was reduced in 52.4% of patients with no change in the Aß1-42/Aß1-40 ratio. |
| [72] | Bergantini *et al*. | 2024 | Blood samples of 89 adult patients. | 89 adult patients previously hospitalized for COVID, including patients with and without HRCT evidence. | Post-COVID syndrome is a major health and social burden, with symptoms lasting for months or even years. |

EQS: Excessive sleep quantity, EDS: Excessive daytime sleepiness

pathophysiological mechanism of post-recovery syndrome is unknown. However, it can be suggested that the virus may persist, especially in the nervous system, and what is present after infection is the occurrence of an inflammatory or autoimmune reaction.

Song *et al*. (2021) [36] examined B cell responses and activation of CNS-specific fractionated T cells were detected by blood and cerebrospinal fluid cytokine analysis of individuals with COVID-19 who had neurological symptoms by single-cell RNA sequencing. Antibodies to SARS were detected in the cerebrospinal fluid of all individuals with COVID-19. In addition, all individuals with COVID-19 have antibodies in their cerebrospinal fluid.

Dutta *et al*. (2021) [37] followed-up of a 51-year-old male without any medical history, who presented with a persistent, subacute migraine headache over a period of 14 days, for which he was admitted to the hospital. The headache was accompanied by vomiting. The headache worsened and developed 6 days after vaccination against COVID-19 with the first dose of Covishield vaccine. Then, he started complaining of double vision that appeared in the horizontal gaze with loss of parallelism in the eyeballs. All complete blood count, blood sugar, lipid profile, renal function tests, and liver function tests were normal.

Chakraborty *et al*., (2021) [38] presented a comprehensive overview from clinical presentation to outcome in eight cases of infectious/post-infectious Guillain-Barré syndrome (GBS). They were reported various extrapulmonary symptoms of COVID-19, and GBS has been widely reported in the literature. GBS is known to be associated with infectious causes, and the association of COVID-19 with

Para infectious/post-infectious GBS has been of interest for a long time.

Metya *et al.*, (2021) [39] applied several neurological manifestations associated with COVID-19 in the form of post-infectious transverse myelitis and Para infectious transverse myelitis. A 54-year-old female patient was admitted to the hospital with acute urinary retention and bilateral upper motor neuron-type lower limb weakness, 12 days after COVID-19.

Elrashdy *et al.* (2021) [40], found that coronavirus vaccines have a significant impact in reducing this pandemic. In addition, that vaccines based on lipid nanoparticles cause rare cases of deep vein thrombosis. Some aspects related to the disease and the main risk factors, as well as the specifics of diagnosis and treatment of this rare condition were studied.

Pandey *et al.* (2021) [41], indicated that the involvement of international organizations is instrumental in mitigating and managing the disease. Most countries have closed their borders and are working remotely, causing the global economy to deteriorate. Poor health, lack of healthy diet, and unhealthy practices among the neighborhood population are the main sources of the spread of disease.

Benjamin *et al.* (2021) [42], studied a single-center cross-sectional including 106 adult patients: 47 non-neurological COVID-19 cases, 30 neurological COVID-19 cases, and 29 non-COVID-19 cases, studied between March and July 2020. Nine antiphospholipid antibodies were evaluated. There was a high prevalence of antiphospholipid antibodies in the non-neurological COVID group (76.6%) and neurological COVID group (73.3%), as well as a low prevalence of antiphospholipid antibodies in the non-COVID group (48.2%).

Hampshirea *et al.* (2021) [43], provided that COVID-19 infection has been shown to cause cognitive deficits that persist in recovery. These deficits vary in magnitude with the severity of respiratory symptoms. They cannot be explained by differences in demographic and socioeconomic variables, age, or education, and persist even in those who do not develop any other residual symptoms.

Lin *et al.* (2021) [44], developed that coronavirus disease in children is mild and sometimes causes serious complications in addition to multisystem inflammatory syndrome in children. Neurological symptoms range from mild headaches to seizures, neuropathy, and encephalopathy. Neurological complications appear differently based on age and comorbidities. A brief study of neurological conditions associated with coronavirus and long-term consequences of the disease in the pediatric population was conducted from preliminary data collected at Morgan Stanley Children's Hospital.

Evans *et al.* (2021) [45], studied a long-term, multicenter follow-up of adults over 18 years of age discharged from hospital in the UK with a clinical diagnosis of COVID-19 and followed up 2–7 months after discharge, including detailed recording of symptoms, biochemical, and physiological tests. Multivariable logistic regression was performed and associations with several covariates were investigated.

Dobrindt *et al.* (2021) [46], explored that the host genes of SARS-CoV-2, human induced pluripotent stem cell-based models, and CRISPR engineering were applied. A single nucleotide polymorphism (rs4702), common in the population and located in the 30 untranslated region of the protease FURIN, affects neurons and alveoli by this virus.

Algahtani *et al.* (2022) [47], explained that all types of vaccines lead to neurological complications of varying severity. A case of severe cervical dystonia that occurred after the first dose of the BNT162b2 COVID-19 vaccine was described, which had not been previously reported after the vaccine. Thus, one of the rare potential side effects of the COVID-19 vaccine was revealed, in addition to the need to deepen research in this area to identify more complications associated with the vaccine.

Sukocheva *et al.* (2022) [48], studied the progression of symptoms in patients after COVID-19 infection. Given the pro-inflammatory effects of this virus, clinical complications after COVID-19 are likely to develop in a proportion of the affected population. A debilitating syndrome has been shown to develop, characterized by headache, fatigue, malaise, cognitive impairment, and dyspnea. Understanding the long-term effects of viral infection requires long-term monitoring of patients after COVID-19.

Butler *et al.* (2022) [49], denoted many authors use the term delirium for post-COVID-19 patients, but use the term acute encephalopathy for clinical syndromes involving rapidly evolving pathological brain processes with additional neurological symptoms.

Das *et al.* (2022) [50], studied a young patient with two conditions, ischemic attack and stroke, after the COVID-19

vaccine (COVISHIELD) and moyamoya vasculopathy was observed. In addition, arterial stroke has been reported after COVID-19 vaccination. However, this is rare and, in the presence of vasculopathy, may not be so rare.

Vanderheiden and Klein (2022) [51], presented neuropathology in COVID-19 patients and its immunological causes. The function of the CNS and its impact on imbalanced peripheral immunity was studied. Then, the blood-brain barrier dysregulation during SARS-CoV-2 infection was examined. Then, the enhancement of COVID-19-associated neuropathology was studied through the possible role of myeloid cells. Finally, the focus was on COVID-19-associated neuroinflammation and the role of innate immunity in it.

Reis *et al*. (2022) [52], demonstrated that neuroscientists are interested in SARS-CoV-2 because the disease has a significant impact on the nervous system during infection and post-COVID. Some of the features that have supported the COVID-19 pandemic are defined from an environmental neuroscience perspective. They considered SARS-CoV-2 and its spread as influenced by environmental factors, its impact on the brain, and some brain health containment measures.

Wild *et al*. (2022) [53], studied 478 COVID-19 survivors and tested 30 days after infection, and it was found that cognition was affected in certain areas. Cognitive performance was found to be positively correlated with physical health and not mental health, suggesting that long COVID leads to physical and cognitive symptoms.

Sadeghizadeh *et al*. (2022) [54], studied two cases of children with COVID-19. Several symptoms such as headaches were seen in pediatric COVID-19 patients with multisystem inflammatory syndrome. However, reversible cerebral vasoconstriction syndrome should be considered as a differential diagnosis in cases of headache with neurological signs.

Aricò *et al*. (2022) [55], studied the COVID-19 outbreak, and the lessons learned that help to better identify strategies to combat future pandemics. Innate and innate immunity constitute the first line of defense against viruses. After a period of infection, antiviral molecules are produced. The progression of the disease in many patients is attributed to insufficient type I interferon activity.

Rastogi *et al*. (2022) [56], studied the first case after COVID-19 vaccine with untreated encephalitis, magnetic resonance imaging performed in conjunction with clinical improvement. Neurological deterioration was described 12 days after the second dose of COVID-19 vaccine. There was no evidence of edema on magnetic resonance imaging, but there was evidence of loss of blood-brain barrier.

Aubart *et al*. (2022) [57], studied 21 children with CNS inflammatory encephalopathy (CNS). A study of children with CNS inflammatory disease describes laboratory, neurological, and radiological features during SARS-CoV-2 infection. All 19 selected patients tested positive for circulating antibodies against SARS-CoV-2, meaning they had a history of exposure to SARS-CoV-2.

Monje and Iwasaki (2022) [58], developed a significant proportion of people with COVID-19 neuropsychiatric symptoms and they were a major component of post-acute COVID-19 syndrome. The neurobiological underpinnings affecting COVID-19 survivors were discussed and mechanisms that may contribute to this neurological health crisis are proposed.

Aschman *et al*. (2022) [59], studies several SARS-CoV-2 patients and have shown neurological symptoms, but the cause of these symptoms is unclear. They could be due to brain cell infection or immune response to the virus, in addition to common factors, since there is no significant evidence for the presence of SARS-CoV particles in the nervous system.

Koh *et al*. (2022) [60], demonstrated a case of a patient who received RNA vaccination for SARS-CoV.A 45-year-old man developed brain, nervous system and spinal cord dysfunction in which autoantibodies were detected. After injection of methylprednisolone and globulins, clinical improvement occurred.

Lambe *et al*. (2022) [61], guided nine cases of SARS-CoV-2 patients and subsequently diagnosed with myelin glial glycoprotein associated disorder. After infection with the coronavirus, some of the patients developed neurological symptoms, another developed myelitis, another developed optic neuritis, and encephalopathy was observed in several patients.

Jammoul *et al*. (2023) [62], controlled a long-term COVID patient experience a variety of neurological manifestations that persist for months after infection. One complication of long-term COVID is autonomic dystonia, which causes dizziness, fatigue, shortness of breath, fainting, and palpitations. The onset of autonomic dystonia after COVID is not definitively known. This study aims to focus on the

potential mechanisms of autonomic dystonia after long-term COVID.

Ulucay *et al*. (2023) [63], studied that mRNA COVID-19 vaccines have been shown to be effective in reducing the severity of illness and complications of COVID-19 infection. The use of mRNA COVID-19 vaccines has also been associated with cardiovascular complications. In addition, COVID-19 infection has been associated with cardiovascular complications.

Takenaka and Nakamori (2023) [64], reported various neurological complications after COVID-19 vaccination. The focus was on the case of a 25-year-old Japanese man who developed acute encephalopathy 2 days after the second dose of the COVID-19 vaccine. He was treated with nivolumab. The antibody test was positive, and he had a high fever and was confused on admission to the hospital. He was given intravenous methylprednisolone after being diagnosed with autoimmune encephalitis, after which the patient's symptoms subsided rapidly.

Sarkanen *et al*. (2023) [65], explained that the COVID-19 pandemic has greatly affected our daily lives around the world. Its effects on fatigue and excessive sleepiness are still unclear. The COVID-19 International Sleep Study questionnaire, which included excessive sleep quantity and excessive daytime sleepiness, as well as socio-demographic factors, was distributed around the world from May to September in 2020.

Hira and Baker (2023) [66], studied patients after several months' recovery from COVID-19, they developed severe post-operative complications. The prevalence of objective dynamic cardiovascular abnormalities was then determined, and gender differences were explored. They demonstrated that patients with severe complications after COVID-19 have evidence of autonomic cardiovascular abnormalities.

Chauvin *et al*. (2023) [67], showed that Tocilizumab treatment in severe COVID-19 patients has been to be effective, but there is little evidence to support the effect of short-term IL-6 receptor blocking therapy on B cell subsets. The study was conducted in 69 COVID-19 patients treated and untreated with tocilizumab. We observed that SARS-CoV-2-specific IgG1 titers were independent of tocilizumab administration but dependent on disease severity.

Seeck and Tankisi (2023) [68], studied the mutations of the original COVID virus that associated with less severe disease. The increase in long COVID cases suggests that the less severe disease is a result of vaccination and still has the potential to cause serious long-term disabilities. Because of respiratory distress, medicine focuses on intensive care, but it is becoming clear that the disease also affects both the central and peripheral systems, so it is preferable to examine it by neurophysiological methods.

Khoo *et al*. (2023) [69], demonstrated that children infected with COVID-19 are less severely affected than adults. They showed through longitudinal multimodal analysis that SARS-CoV-2 leaves a small COVID-19 signature in the T cell compartment of infected children but not in adults with the same disease. This was associated with the development of strong memory T cell responses in adults but not in children. It is the rapid viral clearance in children that restores their ability to resist reinfection.

Ebrahimi *et al*. (2023) [70], collected medical information for 12 patients who had attention-deficit/hyperactivity disorder (ADHD) or multiple sclerosis several weeks after receiving the Covid vaccine. Issues related to the administration of different SARS-CoV vaccines and their relationship with demyelinating disorders in the CNS were studied. Many vaccines have been used as triggers of CNS demyelination. Further investigations are needed to clarify the actual mechanisms and true associations in this field.

Chaumont *et al*. (2023) [71], studied Acute brain injury in COVID-19 patients, which is poorly understood. Preventing long-term neurological complications lies in better understanding these mechanisms. A panel of biomarkers of neuroinflammation in the cerebrospinal fluid of COVID-19 patients should be studied. They studied of 24 Neuro COVID patients, seven (29%) had meningoencephalitis and 17 (71%) had encephalopathy. Several of these patients also had stroke (21%) or new movement disorder (33%).

Bergantini *et al*. (2024) [72], monitoring patients admitted to hospital are most likely to have severe diseases. The etiology of post-COVID syndrome remains unknown. A panel of inflammatory and immune cytokines was evaluated in individuals without HRCT-variable features and in patients with High-resolution computed tomography variable features as part of long-COVID symptoms. A sample of 89 adult patients previously hospitalized for COVID, including patients with and without HRCT evidence.

## 5. CONCLUSION

We acquired a significant number of papers by conducting research using the keywords COVID-19, neuroscience, and autoantibodies. Two hundred and forty-three publications were found once the data were cleaned. Starting with reference 32 and continuing through reference 72, 243 articles undergo many steps of systematic review before arriving at the outcome of 41 publications. These sources are taken from research design, conclusion, and number of instances. An examination of this research revealed that the majority of them concentrate on the long-term, or post-COVID, impact of the condition. The most common of these symptoms, along with encephalitis and associated consequences, is nervous system inflammation. Furthermore, adverse consequences follow COVID immunization.

These three fields of study are still essential for developing public health plans, refining treatment modalities, and comprehending the wider ramifications of this worldwide outbreak. One of the important determinants of this research, especially since we are talking about the human health, is that there are some papers focusing on specific case with specific condition and cannot be generalized, in addition to the fact that there are various problems that were associated with each vaccine.

## REFERENCES


[1] M. Al-Ani and D. M. Alani. "Review study on sciencedirect library based on coronavirus Covid-19". *UHD Journal of Science and Technology (UHDJST)*, vol. 4, no. 2, p. 46, 2020.

[2] A. Fletcher-Sandersjöö and B. M. Bellander. "Is COVID-19 associated thrombosis caused by over activation of the complement cascade? A literature review". *Thrombosis Research*, vol. 194, pp. 36-41, 2020.

[3] B. A. Mohammed and M. S. Al-Ani. "Follow-up and diagnose COVID-19 using deep learning technique". *Advances in Science Technology and Engineering Systems Journal*, vol. 6, no. 2, pp. 971-976, 2021.

[4] J. Cui, F. Li and Z. L. Shi. "Origin and evolution of pathogenic coronaviruses". *Nature Reviews Microbiology*, vol. 17, no. 3, pp. 181-192, 2019.

[5] M. S. Al-Ani, T. S. Mohammed, A. I. Sultan, H. I. Sultan, K. M. Ali Alheeti and K. M. Aljebory. "Detection and diagnostic approach of COVID-19 based on cough sound analysis". *Journal of Computer Science*, vol. 17, no. 6, pp. 580-597, 2021.

[6] F. Dono, B. Nucera, J. Lanzone, G. Evangelista, F. Rinaldi, R. Speranza, S. Troisi, L. Tinti,… & F. Brigo. "Status epilepticus and COVID-19: A systematic review". *Epilepsy and Behavior*, vol. 118, p. 107887, 2021.

[7] T. S. Mohammed, A. I. Sultan, K. M. Ali Alheeti, K. M. Aljebory, H. I. Sultan and M. S. Al-Ani. "COVID-19 diagnosis using spectral and statistical analysis of cough recordings based on the combination of SVD and DWT". *Baghdad Science Journal*, vol. 20, pp. 372-385.

[8] K. Nemani, C. Li, M. Olfson, E. M. Blessing, N. Razavian, J. Chen, E. Petkova and D. C. Goff. "Association of psychiatric disorders with mortality among patients with COVID-19". *JAMA Psychiatry*, vol. 78, pp. 380-386.

[9] M. S. Al-Ani, Q. Al-Shayea, S. M. Al-Barzinji, D. M. S. Al-Ani and Z. M. S. Al-Ani. "COVID-19 diagnosis applied DWT and CNN on X-ray chest images". *UHD Journal of Science and Technology* (*UHDJST*), vol. 7, no. 2, pp. 69-76.

[10] L. M. Khosroshahi, M. Rokni and F. Noorbakhsh. "Immunology, immunopathogenesis and immunotherapeutics of COVID-19; an overview". *International Immunopharmacology*, vol. 93, p. 107364.

[11] K. M. Ali Alheeti, M. S. Al-Ani, S. M. Nejrs, D. M. S. Al-Ani and Z. M. S. Al-Ani. "Tracing fields, methods and origins of Covid-19". *AIP Conference Proceedings*, vol. 3009, p. 020003.

[12] M. Tandon, S. Kataria, J. Patel, T. R. Mehta, M. Daimee, V. Patel, A. Prasad, A. A. Chowdhary, S. Jaiswal and S. Sriwastava. "A Comprehensive systematic review of csf analysis that defines neurological manifestations of COVID-19". *International Journal of Infectious Diseases*, vol. 104, pp. 390-397, 2021.

[13] S. Sriwastava, S. Kataria, M. Tandon, J. Patel, R. Patel, A. Jowkar, M. Daimee, E. Bernitsas, P. Jaiswal and R. P. Lisak. "Guillain Barré Syndrome and its variants as a manifestation of COVID-19: A systematic review of case reports and case series". *Journal of the Neurological Sciences*, vol. 420, p. 117263, 2021.

[14] S. Katal. "Neuroimaging findings in COVID-19: A narrative review". *Neuroscience Letters*, vol. 742, p. 135529, 2021.

[15] A. Nath and B. Smith. "Neurological issues during COVID-19: An overview". *Neuroscience Letters*, vol. 742, p. 135533, 2021.

[16] A. Emmia, I. Boura, V. Raeder, D. Mathew, D. Sulzer, J. E. Goldman and V. Leta. "Covid-19, nervous system pathology, and Parkinson's disease: Bench to bedside". *International Review of Neurobiology*, vol. 165, pp. 17-34, 2022.

[17] M. Van Der Neut Kolfschoten, J. Schuurman, M. Losen, W. K. Bleeker, P. Martínez-Martínez, E. Vermeulen, T. H. den Bleker, L. Wiegman, T. Vink, L. A. Aarden, M. H. De Baets, J. G. J. Van de Winkel, R. C. Aalberse and P. W. H. I. Parren. "Anti-inflammatory activity of human IgG4 antibodies by dynamic Fab arm exchange". *Science*, vol. 317, pp. 1554-1557, 2007.

[18] A. McShane and S. E. Mole. "Sex bias and omission exists in Batten disease research: Systematic review of the use of animal disease models". *Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease*, vol. 1, p. 166489, 2022.

[19] E. Calabria, F. Canfora, M. Mascolo, S. Varricchio, M. D. Mignogna and D. Adamo. "Autoimmune mucocutaneous blistering diseases after SARS-Cov-2 vaccination: A case report of pemphigus vulgaris and a literature review". *Pathology-Research and Practice*, vol. 232, p. 153834, 2022.

[20] M. M. Samim, D. Dhar, F. Arshad, D. D. S. Anudeep, V. G. Patel, S. R. Neeharika, K. Dhamija, C. M. Ravindranath, R. Yadav, P. Raja, M. Netravathi, D. Menon, V. V. Holla, N. L. Kamble, P. K. Pal, A. Nalini and S. Vengalil. "Co-VAN study: COVID-19 vaccine associated neurological diseases- an experience from an apex neurosciences center and review of the literature". *Journal of Clinical Neuroscience*, vol. 108, pp. 37-75, 2023.

[21] R. G. Kamperman, E. M. Van Leeuwen, M. Willems, A. J. Van der Kooi, K. A. Gelderman, D. Hamann, E. G. Van Lochem, B. Meek, R. G. Van der Molen, A. C. Platteel,… & J. Raaphorst. "Does COVID-19 impact the prevalence of myositis specific antibodies

in the Netherlands? A comparative nationwide study". *Journal of Autoimmunity*, vol. 139, p. 103081, 2023.

[22] F. Nabizadeh, M. Noori, S. Rahmani and H. Hosseini. "Acute disseminated encephalomyelitis (ADEM) following COVID-19 vaccination: A systematic review". *Journal of Clinical Neuroscience*, vol. 111, pp. 57-70, 2023.

[23] S. Najdaghi, D. N. Davani and M. Hashemian and N. Ebrahimi. "Cerebellitis following COVID-19 infection: A case-based systematic review and pooled analysis". *Heliyon*, vol. 10, p. e34497.

[24] D. Duricka and L. Liu. "Reduction of long COVID symptoms after stellate ganglion block: A retrospective chart review study". *Autonomic Neuroscience: Basic and Clinical*, vol. 254, p. 103195, 2024.

[25] M. Dasara, F. Dono, G. Evangelista, P. Quintieri, D. Liviello, S. Cipollone, V. Tomassini and S. L. Sensi. "Status epilepticus as a complication of SARS-CoV-2 vaccination: Two case reports and systematic review with individual patients' data analysis". *Seizure: European Journal of Epilepsy*, vol. 121, pp. 8-16, 2024.

[26] M. Bozbıyık and T. Morton. "Epistemic positioning and knowledge-building in postgraduate neuroscience classroom interaction". *Journal of Pragmatics*, vol. 232, p. 72-90, 2024.

[27] A. J. Beetsma, R. Reezigt and M. F. Reneman. "Knowledge and attitudes toward musculoskeletal pain neuroscience of manual therapy postgraduate students in the Netherlands". *Musculoskeletal Science and Practice*, vol. 52, p. 102350, 2021.

[28] L. E. Wool. "Knowledge across networks: how to build a global neuroscience collaboration". *Neurobiology*, vol. 65, pp. 100-107, 2020.

[29] K. Walsh. "Translating neuroscience to early childhood education: A scoping review of neuroscience-based professional learning for early childhood educators". *Educational Research Review*, vol. 45, p. 100644, 2024.

[30] K. Simmers and I. Davidesco. "Neuroscience literacy and evidence-based practices in pre-service teachers: A pilot study". *Trends in Neuroscience and Education*, vol. 35, p. 100228, 2024.

[31] Y. G., Chung, Y. Jeon, S. Yoo, H. Kim and H. Hwang. "Big data analysis and artificial intelligence in epilepsy—common data model analysis and machine learning-based seizure detection and forecasting". *Clinical and Experimental Medicine*, vol. 65, pp. 272-282, 2022.

[32] G. Morris, E. Athan, K. Walder, C. C. Bortolasci, A. O'Neil, W. Marx, M. Berk, A. F. Carvalho, M. Maes and B. K. Puri. "Can endolysosomal deacidification and inhibition of autophagy prevent severe COVID-19?". *Life Sciences*, vol. 262, p. 118541, 2020.

[33] R. Vaschetto, T. Cena, T. T. Sainaghi, G. Meneghetti, S. Bazzano, D. Vecchio, M. Pirisi, D. Brustia, M. Barini, G. Cammarota, L. Castello and F. D. Corte. "Cerebral nervous system vasculitis in a Covid-19 patient with pneumonia". *Journal of Clinical Neuroscience*, vol. 79, pp. 71-73, 2020.

[34] J. Hu, J. Jolkkonen and C. Zhao. "Neurotropism of SARS-CoV-2 and its neuropathological alterations: Similarities with other coronaviruses". *Neuroscience and Biobehavioral Reviews*, vol. 119, pp. 184-193, 2020.

[35] D. Bertin, E. Kaphan, S. Weber, B. Babacci, R. Arcani, B. Faucher, A. Ménard, A. Brodovitch, J. L. Mege and N. Bardin. "Persistent IgG anticardiolipin autoantibodies are associated with post-COVID syndrome". *International Journal of Infectious Diseases,* vol. 113, pp. 23-25, 2021.

[36] E. Song, C. M. Bartley, R. D. Chow, T. T. Ngo, R. Jiang, C. R. Zamecnik, R. Dandekar, R. P. Loudermilk, Y. Dai, F. Liu, S. Sunshine, J. Liu, W. Wu,… & S. F. Farhadian. Divergent and self-reactive immune responses in the CNS of COVID-19 patients with neurological symptoms". vol. 2, no. 5, p. 100288, 2021.

[37] A. Dutta, R. Ghosh, D. Bhattacharya, S. Bhat, A. Ray, A. Pandit, S. Das and S. Dubey. "Anti-PF4 antibody negative cerebral venous sinus thrombosis without thrombocytopenia following immunization with COVID-19 vaccine in an elderly non-comorbid Indian male, managed with conventional heparin-warfarin based anticoagulation". *Diabetes and Metabolic Syndrome: Clinical Research and Reviews*, vol. 15, p. 102184, 2021.

[38] U. Chakraborty, A. Hati and A. Chandra. "Covid-19 associated Guillain-Barr_e syndrome: A series of a relatively uncommon neurological complication". *Diabetes and Metabolic Syndrome: Clinical Research and Reviews*, vol. 15, p. 102326, 2021.

[39] S. Metya, S. Shaw, S. Mondal, B. Chakraborty, S. Das, S. Roy, M. K. Das, S. Kanjilal, B. K. Ray and S. Dubey. "MRI-negative myeloradiculoneuropathy following Covid-19 infection: An index case". *Diabetes and Metabolic Syndrome: Clinical Research and Reviews*, vol. 15, p. 102305, 2021.

[40] F. Elrashdy, M. M. Tambuwala, S. S. Hassan, P. Adadi, M. Seyran, T. M. Abd El-Aziz, N. Rezaei, A. Lal, A. A. A. Aljabali, R. Kandimalla, N. G. Bazan,… & V. N. Uversky. "Autoimmunity roots of the thrombotic events after COVID-19 vaccination". *Autoimmunity Reviews*, vol. 20, p. 102941, 2021.

[41] K. Pandey, M. Thurman, S. D. Johnson, A. Acharya, M. Johnston, E. A. Klug, O. A. Olwenyi, R. Rajaiah and S. N. Byrareddy. "Mental health issues during and after COVID-19 vaccine era". *Brain Research Bulletin*, vol. 176, pp. 161-173, 2021.

[42] L. A. Benjamin, R. W. Paterson, R. Moll, C. Pericleous, R. Brown, P. R. Mehta, D. Athauda, O. J. Ziff, J. Heaney, A. M. Checkley, C. F. Houlihan, M. Chou,… & UCLH Queen Square COVID-19 Biomarker Study Group. "Antiphospholipid antibodies and neurological manifestations in acute COVID-19: A single-center cross-sectional study". *EClinicalMedicine*, vol. 39, p. 101070, 2021.

[43] A. Hampshirea, W. Trender, S. R. Chamberlain, A. E. Jolly, J. E. Grant, F. Patrick, N. Mazibuko, S. C. Williams, J. M. Barnby, P. Hellyer and M. A. Mehta. "Cognitive deficits in people who have recovered from COVID-19". *EClinicalMedicine*, vol. 39, p. 101044, 2021.

[44] J. E. Lin, A. Asfour, T. B. Sewell, B. Hooe, P. Pryce, C. Earley, M. Y. Shen, M. Kerner-Rossi, K. T. Thakur, W. S. Vargas, W. G. Silver and A. S. Geneslaw. "Neurological issues in children with COVID-19". *Neuroscience Letters*, vol. 743, p. 135567, 2021.

[45] R. A. Evans, H. McAuley, E. M. Harrison, A. Shikotra, A. Singapuri, M. Sereno, O. Elneima, A. B. Docherty, N. I. Lone, O. C. Leavy, L. Daines, J. K. Baillie, J. S. Brown,… & PHOSP-COVID Collaborative Group. "Physical, cognitive, and mental health impacts of COVID-19 after hospitalization (PHOSP-COVID): A UK multicenter, prospective cohort study". *The Lancet Respiratory Medicine*, vol. 9, no. 11, pp. 1275-1287.

[46] K. Dobrindt, D. A. Hoagland, C. Seah, B. Kassim, C. P. O'Shea, A. Murphy, M. Iskhakova, M. B. Fernando, S. K. Powell, P. J. M. Deans, B. Javidfar, C. Peter, R. Møller,… & S. A. Uhl. "Common genetic variation in humans impacts *in vitro* susceptibility to SARSCoV-2 infection". *Stem Cell Reports*, vol. 16, pp. 505-518, 2021.

[47] H. A. Algahtani, B. H. Shirah and E. Alwafi. "Acute cervical dystonia following the BNT162b2 mRNA COVID-19 vaccine". *Clinical Neurology and Neurosurgery*, vol. 218, p. 107304, 2022.

[48] O. A. Sukocheva, R. Maksoud, N. M. Beeraka, S. V. Madhunapantula, S. G. Klochkov, M. A. Kamal, D. R. Staines and S. Marshall-Gradisnik. "Analysis of post COVID-19 condition and its overlap with myalgic encephalomyelitis/chronic fatigue syndrome". *Journal of Advanced Research*, vol. 40, pp. 179-196, 2022.

[49] M. Butler, B. Cross, D. Hafeez and M. F. Lim. "Emerging knowledge of the neurobiology of COVID-19". *Psychiatric Clinics of North America*, vol. 45, pp. 29-43, 2022.

[50] S. Das, B. K. Ray, A. Pandit, A. Dutta, S. Bhat, D. Bhattacharyya, A. P. Chakraborty, R. Agrawal and S. Dubey. "Covid-19 vaccine (covishield) heralding Moyamoya angiopathy", *Neuroimmunology Reports*, vol. 2, pp. 100100, 2022.

[51] A. Vanderheiden and R. S. Klein. "Neuroinflammation and COVID-19". *Current Opinion in Neurobiology*, vol. 76, p. 102608, 2022.

[52] J. Reis, A. Buguet, G. C. Román and P. S. Spencer. "The COVID-19 pandemic, an environmental neurology perspective". *Revue Neurologique*, vol. 178, pp. 499-511, 2022.

[53] C. J. Wild, L. Norton, D. K. Menon, D. A. Ripsman, R. H. Swartz and A. M. Owen. "Disentangling the cognitive, physical, and mental health sequelae of COVID-19". *Cell Reports Medicine*, vol. 3, p. 100750, 2022.

[54] A. Sadeghizadeh, Z. Pourmoghaddas, A. Zandifar, S. Z. Tara, H. Rahimi, R. Saleh, S. Ramezani, M. Ghazavi, O. Yaghini, N. Hosseini, N. Aslani, M. Saadatnia and A. Vossough. "Reversible cerebral vasoconstriction syndrome and multisystem inflammatory syndrome in children with COVID-19". *Pediatric Neurology*, vol. 129, pp. 1-6, 2022.

[55] E. Aricò, L. Bracci, L. Castiello, F. Urbani, J. L. Casanova and F. Belardelli. "Exploiting natural antiviral immunity for the control of pandemics: Lessons from Covid-19". *Cytokine and Growth Factor Reviews*, vol. 63, pp. 23-33, 2022.

[56] A. Rastogi, A. Bingeliene, A.P. Strafella, D. F. Tang-Wai, P. E. Wu and D. M. Mandell. "Reversible neurological and brain MRI changes following COVID-19 vaccination: A case report". *Journal of Neuroradiology*, vol. 49, pp. 428-430, 2022.

[57] M. Aubart, C.J. Roux, C. Durrleman, C. Gins, M. Hully, M. Kossorotoff, C. Gitiaux, R. Levy, F. Moulin, A. Debray, Z. Belhadjer, E. Georget, T. Kom and P. Blanc. "Neuroinflammatory disease following severe acute respiratory syndrome coronavirus 2 infection in children". *The Journal of Pediatrics*, vol. 247, pp. 22-28.e2, 2022.

[58] M. Monje and A. Iwasaki. "The neurobiology of long COVID". *Neuron*, vol. 110, no. 21, p. 3484-3496, 2022.

[59] T. Aschman, R. Mothes and F. L. Heppner. "What SARS-CoV-2 does to our brains". *Immunity*, 55, pp. 1159-1172, 2022.

[60] P. X. Koh, K. Y. Tay, T. Yeo, D. R. Singh, J. S. Koh, U. N. Thirugnanam and M. Saini. "Glial fibrillary acidic protein astrocytopathy in a patient with recent mRNA SARS-CoV-2 vaccination". *Neuroimmunology Reports*, vol. 2, p. 100053, 2022.

[61] J. Lambe, M. P. McGinley, B. P. Moss, Y. Mao-Draayer, R. Kassa, J. R. Ciotti, S. Mariotto and A. Kunchok. "Myelin oligodendrocyte glycoprotein-IgG associated disorders (MOGAD) following SARS-CoV-2 infection: A case series". *Journal of Neuroimmunology*, vol. 370, p. 577933, 2022.

[62] M. Jammoul, J. Naddour, A. Madi, M. A. Reslan, F. Hatoum, J. Zeineddine and W. Abou-Kheir, N. Lawand. "Investigating the possible mechanisms of autonomic dysfunction post-COVID-19". *Autonomic Neuroscience: Basic and Clinical*, vol. 245, p. 103071, 2023.

[63] A. S. Ulucay. "Risk of myocarditis? Review of the current evidence, insights, and future directions". *Indian Heart Journal*, 75, p. 217e223, 2023.

[64] M. Takenaka, M. Nakamori, R. Ishikawa, S. Aoki and H. Maruyama. "Encephalopathy after COVID-19 vaccination during treatment with nivolumab: A case report". *Clinical Neurology and Neurosurgery*, vol. 226, p. 107632, 2023.

[65] T. Sarkanen, M. Partinen, B. Bjorvatn, I. Merikanto, C. Benedict, M. R. Nadorff, C. J. Bolstad, C. Espie, K. Matsui, F. Chung, C. M. Morin, Y. K. Wing, T. Penzel, T. Macêdo and S. Mota-Rolim. "Association between hypersomnolence and the COVID-19 pandemic: The international COVID-19 sleep study (ICOSS)". *Sleep Medicine*, 107, p. 108e115, 2023.

[66] R. Hira and J. R. Baker. "Objective hemodynamic cardiovascular autonomic abnormalities in post-acute sequelae of COVID-19". *Canadian Journal of Cardiology*, vol. 39, p. 767e775, 2023.

[67] C. Chauvin, L. Levillayer, M. Roumier, H. Nielly, C. Roth, A. Karnam, S. R. Bonam, A. Bourgarit, C. Dubost, A. Bousquet, S. Le Burel, R. Mestiri, D. Sene and J. Galland. "Tocilizumab-treated convalescent COVID-19 patients retain the cross-neutralization potential against SARS-CoV-2 variants". *iScience*, vol. 26, p. 106124, 2023.

[68] M. Seeck and H. Tankisi. "Clinical neurophysiological tests as objective measures for acute and long-term COVID-19". *Clinical Neurophysiology Practice*, vol. 8, pp. 1-2, 2023.

[69] W. H. Khoo, K. Jackson, C. Phetsouphanh, J. J. Zaunders, J. Alquicira-Hernandez, S. Yazar, S. Ruiz-Diaz, M. Singh, R. Dhenni, W. Kyaw, F. Tea, V. Merheb and F. X. Z. Lee. "Tracking the clonal dynamics of SARS-CoV-2-specific T cells in children and adults with mild/asymptomatic COVID-19". *Clinical Immunology*, vol. 246, p. 109209, 2023.

[70] N. Ebrahimi, M. Mazdak, V. Shaygannejad and O. Mirmosayyeb. "CNS demyelinating disease following inactivated or viral vector SARS-CoV-2 vaccines: A case series". *Vaccine*, vol. 41, pp. 1003-1008, 2023.

[71] H. Chaumont, F. Kaczorowski, A. San-Galli, P. P. Michel, B. Tressières, E. Roze, I. Quadrio and A. Lannuzel. "Cerebrospinal fluid biomarkers in SARS-CoV-2 patients with acute neurological syndromes". *Revueneurologique* (*Paris*), vol. 179, pp. 208-217, 2023.

[72] L. Bergantini, S. Gangi, B. Perea, M. Meocci, G. Fabbri, F. Bianchi and E. Bargagli. "Altered serum concentrations of IL-8, IL-32 and IL-10 in patients with lung impairment 6 months after COVID-19". *Immunobiology*, vol. 229, p. 152813, 2024.