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A Review of Covid-19 Autoimmune and Neurologic Complications

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Abstract
The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has significantly impacted the immune system. COVID-19 is considered to be linked to various autoimmune disorders. Immunologically, SARS-CoV-2 could trigger excessive inflammatory responses, leading to cytokine storms, autoimmune reactions, and dysregulation of B cell and T cell responses. These responses might contribute to different autoimmune disorders affecting different systems. Besides, the virus impacts both the central and peripheral nervous systems through a variety of neurological mechanisms. Numerous case reports and review articles have detailed the complexities arising from infection. Nonetheless, this review aims to thoroughly examine the broad spectrum of neurological and autoimmune complications linked to COVID-19 infection, emphasizing the urgent need for preventive measures and the crucial role of early detection and timely management of the complications.

Keywords: COVID-19, Immune System, Neurologic Disorders, Autoimmune Disorders

1. Introduction

The coronavirus disease 2019 (COVID-19) pandemic was caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which belongs to the Coronaviridae family. The current coronavirus is closely associated with other lethal human coronaviruses, including the severe acute respiratory syndrome coronavirus (SARS-CoV) and the Middle East respiratory syndrome coronavirus (MERS-CoV) (Li et al., 2021). SARS-CoV-2 is a single-stranded RNA virus with a spherical shape and four structural proteins (Figure 1): Spike (S) protein, Membrane (M) protein, Envelope (E) protein, and Nucleocapsid protein (N) (Chen et al., 2020). The Spike protein binds to the host cell via the angiotensin-converting enzyme 2 (AC2) receptor on the host cell membrane, a critical step in the virus's entry into the cells (Figure 2). Given the presence of this receptor on various cell types, this

interaction has the potential to affect multiple organs, including the skin, kidneys, respiratory system, cardiovascular system, digestive system, nervous system, and hematological system (Jackson et al., 2022).

It is widely acknowledged that certain viruses may potentially induce autoimmune disorders (Chen et al., 2020). For instance, COVID-19 has been observed to elicit immunological effects, such as the dysregulation of B cells and T cells, the hyperactivation of CD8+ T cells and natural killer cells (NK), and elevated levels of inflammatory cytokines such as tumor necrosis factor (TNF-α), interleukin (IL)-1, and IL-6. These factors could potentially exert a significant influence on the body (Zhou et al., 2021).

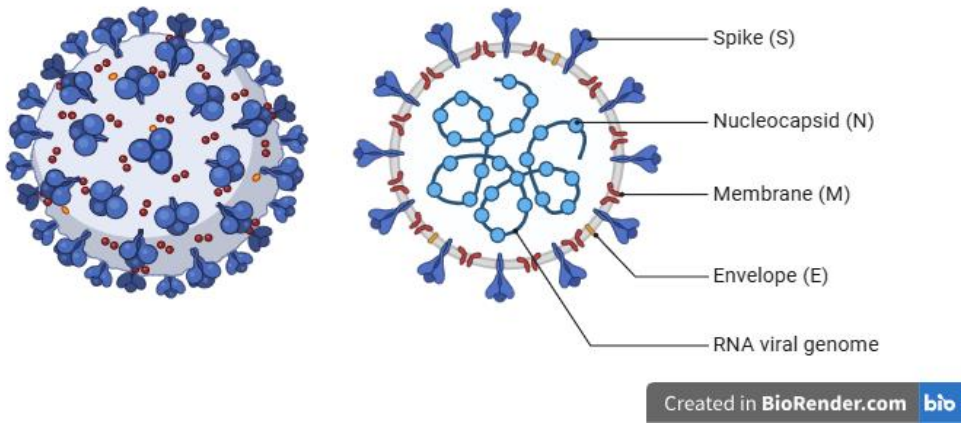

Figure 1: Coronavirus structure

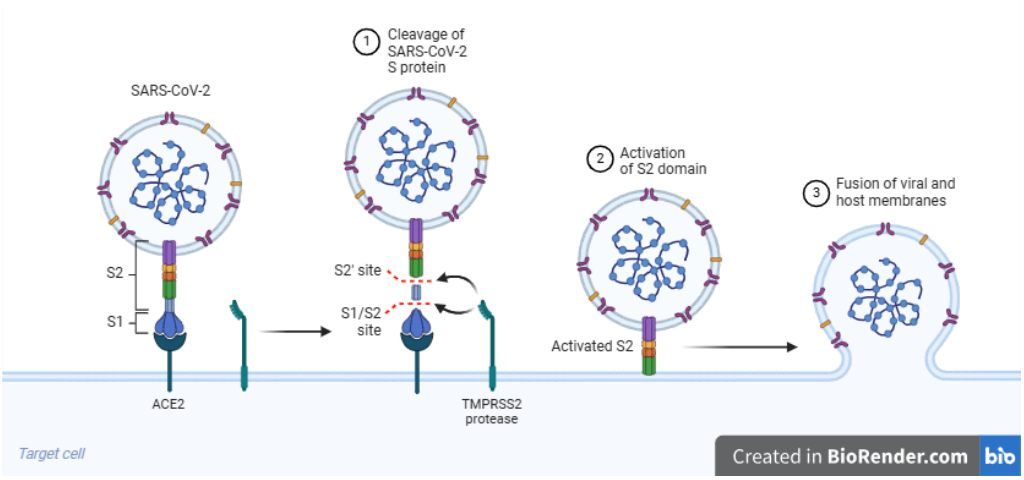

Figure 2: Mechanism of SARS-CoV-2 viral entry. SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2

S and M proteins are part of the virus and play crucial roles in immune responses. The interaction of the S protein with monocyte could increase IL-8 and IL-6 secretion, while the M protein can suppress IL-1 production (Oslon et al., 2001). Moreover, evidence suggests that coronavirus may result in cross-reactivity, bystander activation, long-lasting viral presence, and an exaggerated innate inflammatory response. These factors could potentially enhance autoimmune reactions and impact autoimmunity (Wong et al., 2004). Bystander activation is the release of cytokines, triggering T cell auto-reactivation and can initiate autoimmune disease (Sacchi et al., 2021). However, the body's innate immunity which is the initial defense against infections and plays a crucial role in controlling them. Following COVID-19, this immune response may persist and lead to a hyperinflammatory condition known as cytokine storm. Moreover, excessive activation of macrophages and monocytes may result in

tissue damage (Vahabi et al., 2022). The complement system is among the first immune responses, and when overactivated, it can result in further cell damage (Stoermer et al., 2011). Adaptive immunity, which is essential for clearing infections, may also lead to an autoimmune response. It consists of cellular and humoral responses. In cellular immunity, CD4+ and CD8+ T cells have distinct functions. Type 1 helper T cells (Th1) from CD4+ cells can release inflammatory cytokines and high interferon (IFN)γ levels, activating macrophages and leading to delayed hypersensitivity reactions (Chen et al., 2020). Moreover, COVID-19 may trigger humoral immunity in predisposed individuals. Excessive circulatory autoantibodies have been observed in coronavirus-infected patients (Shikh et al., 2010). The immune responses to the infection are shown in Figure 3. The following discussion will explore the immunological impact of this infection on various organs and examine potential neurological complications.

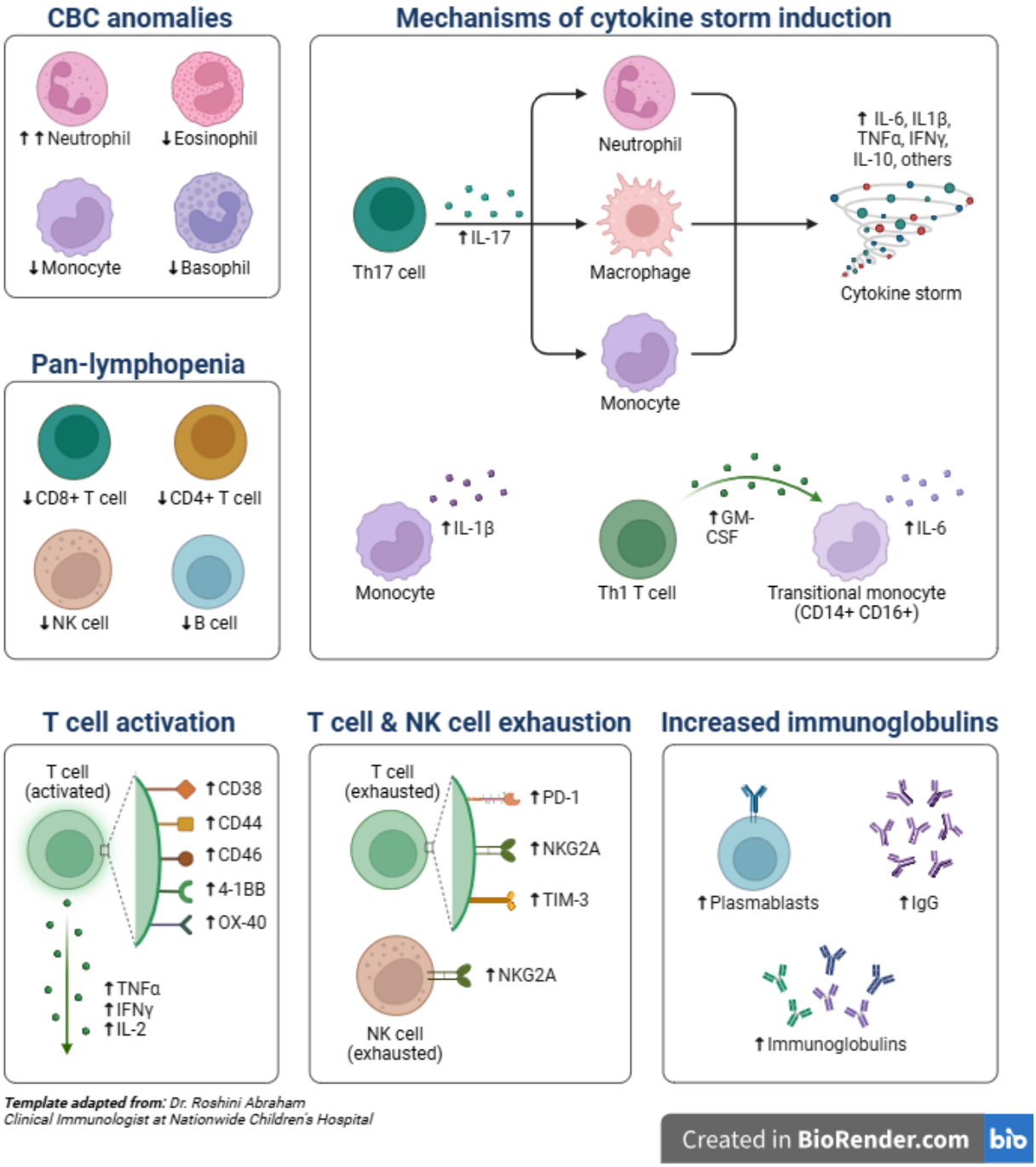

Figure 3: Immune mechanisms affected by coronavirus

2. Endocrine Complications

The overactivity of the immune system, particularly involving Th1/Th17 lymphocytes, may result in the release of pro-inflammatory cytokines (IL1-6, TNFα) and cytokine storms. This could lead to acute and delayed thyroid dysregulation (Caron et al., 2020). There were reports of abnormal thyroid function during the acute and sub-acute phases of COVID-19 infection (Ippolito, 2020). An increase in thyroperoxidase antibodies (TPO Ab) was reported in some studies after infection (Anaya et al., 2021. Lui et al., 2021). In their comprehensive review, Vamshidhar and colleagues (2023) suggested that COVID-19 may have the capacity to trigger Graves' disease in susceptible individuals by affecting the immune system. A systematic review conducted by Tutal et al. (2022) highlighted that COVID-19 might trigger and initiate autoimmune thyroid disease and exacerbate pre-existing thyroid conditions. They demonstrated that both Graves' disease and Hashimoto's thyroiditis could develop as a result of the infection. It has been observed that the likelihood of developing subacute thyroiditis is doubled within the first six months following the onset of COVID-19 (Lee et al., 2023). Additionally, an elevated risk of complications was observed beyond six months from the onset of the infection, with a higher incidence among female individuals. Aref Zade et al. (2024) have indicated that COVID-19 infection may be linked to euthyroid sick syndrome, thyroiditis, as well as clinical and subclinical hypothyroidism and hyperthyroidism. The systematic review by Rehman et al. (2021) identified a correlation between COVID-19 infection and subacute thyroiditis. They found that 71.4% of involved patients were women, and the mean days of thyroiditis symptoms onset after infection was 25.2+/-10.1.

Diabetes mellitus (DM) is recognized as a predisposing factor for severe COVID-19 illness. A potential cause is thought to be the hyperinflammatory response, as diabetic patients continuously have low-grade inflammation. Conversely, this chronic inflammation is magnified with COVID-19 infection (Azar et al., 2020). New onset diabetes cases were reported in those without a history of disease, and it was expressed that cytokine activation provokes insulin resistance and actuates hyperglycemia (Boddu et al., 2020). However, other mechanisms have been suggested, such as oxidative stress and drug side effects (Shrestha et al., 2021). Shrestha et al. (2021) conducted a systematic review to evaluate the correlation between the risk of diabetes mellitus (DM) and COVID-19 infection. Their findings suggest that COVID-19 may be linked to the new-onset DM in adults. In a meta-analysis organized by Rahmati and colleagues (2023), an increased risk of new cases of type-1 DM in children and adults was reported. Recently, Bellia et al. (2023) performed a meta-analysis and found a higher incidence of diabetes mellitus in individuals with a history of COVID-19 infection. They also noted an increased risk in those who needed hospitalization due to illness severity and those older than 60 years old. Stathi et al.'s meta-analysis (2023) announced the increased risk of type-1 DM and diabetic ketoacidosis due to immunity destroying pancreatic beta cells. According to their results, after infection, patients experienced a latency period ranging from 3 to 84 days before the onset of symptoms related to DM.

During the COVID-19 infection, some cases were reported that presented with atypic symptoms of infection; they showed symptoms of adrenal insufficiency and were diagnosed with Addison’s disease (Bhattarai et al., 2021. Beshay et al., 2022). Sanchez et al. (2022) reported a case of Addison’s disease five months after infection and suggested an autoimmune etiology for the disease. However, other mechanisms, such as adrenal hemorrhage and infarction, have been reported as causative factors in some instances of adrenal insufficiency (Elhassan et al., 2023).

3. Connective Tissue Disorders

In 2020, Ciaffi et al. (2020) conducted a comprehensive review and found that rheumatologic symptoms may be the initial sign of COVID-19 or arise during infection. According to Mudge and colleagues (2024), the time from infection diagnosis to inflammatory musculoskeletal presentations could range from 0 to 120 days.

Based on the Migliorine meta-analysis (2023), COVID-19 could provoke reactive arthritis. Chaudhry et al. (2022), reported different types of acute arthritis with a latency period of 4-14 weeks after COVID-19 presentations. Cioffi

et al. meta-analysis (2023) determined that a range of rheumatic musculoskeletal disorders, including joint pain, inflammatory arthritis, and fibromyalgia, have been frequently observed within 1-12 months following COVID-19 infection. Bouden et al. (2024) examined case reports of post-COVID arthritis and showed an incidence of 9.2% for rheumatoid arthritis. However, reactive arthritis was reported to be the most frequent form of articular involvement.

Kouranloo et al. meta-analysis (2023) assessed the relationship between COVID-19 infection and connective tissue disorders. They found that idiopathic inflammatory myositis and systemic lupus erythematosus were the most common disorders following infection, and young women were most affected. Additionally, they suggested a potential association between these two conditions. After the emergence of the coronavirus, there has been a notable increase in dermatomyositis cases. In response, Holzer and colleagues (2022) conducted a review study, revealing that COVID-19 can induce new-onset dermatomyositis by activating the interferon pathway of inflammation.

Different types of vasculitis have been described as complications of COVID-19 infection. Wong et al. (2022), in their systematic review, examined various types of rash and found a man-to-woman ratio of 4:5. Giryes et al. (2022) assessed cases of large vessel vasculitis as well as medium and small vessel vasculitis presenting during or following infection. They stated there is a low risk for these types of complications. Immunoglobulin A vasculitis, a condition more prevalent in pediatric patients, has shown an increased incidence among adults with COVID-19 infection, as indicated by a meta-analysis conducted by Messova and colleagues (2022). The analysis revealed a higher prevalence of renal involvement and a male predominance. Additionally, the time lapse between the onset of infection and vasculitis presentations ranged from 2 to 120 days.

Regarding inflammatory conditions in the pediatric population, Sharma et al. (2022) examined Kawasaki syndrome and multisystem inflammatory syndrome case reports. They estimated a pooled prevalence of 29%. Batu et al. (2022) assessed COVID-19 related vasculitis in children. They found a median age of 13 and a male-female ratio of 2:3, with a latency period of 2-120 days after infection. The IgA vasculitis was shown to be more common, and the skin was the most affected organ. However, the overall risk for vasculitis complications was low. Sachdeva et al. meta-analysis (2022) expressed an incidence of 32% neurological involvement in multisystem inflammatory syndrome in pediatrics.

Respecting systemic lupus erythematosus (SLE), the Kouranloo review (2023) reported a female predominance and found 50% of renal involvement. Based on the data from Assar et al.'s review article (2022), the mean time between lupus presentations and infection was 24.86 (13-60 days). Renal and pulmonary involvement were more common. All cases tested positive for antinuclear antibody (ANA), and the second most common autoantibody reported was anti-double-stranded DNA (anti-ds DNA). In the study conducted by Lupu et al. (2023), cases of SLE in the pediatric population were documented. Most occurrences were observed in children between 11 and 13, and symptoms of SLE typically appeared within two months following infection.

Fineschi et al. (2021), Carroll et al. (2023), and Zou et al. (2024), outlined cases of scleroderma following infection. Zou et al. found a 1:3 male-female ratio after COVID-19 infection. According to the Carroll report, the latency period was about two weeks. In their review article, Aram and colleagues (2021) provided examples of scleroderma flare-ups. Interestingly, Yu et al. (2023) reported a case of a known scleroderma who progressively developed Sjogren's disease following COVID-19 infection.

4. Dermatologic Diseases

Lim et al. (2023) reported that about 60% of the included infected population presented with skin conditions. Regarding evaluating complications of COVID-19, Igbal et al. (2023) conducted a meta-analysis and expressed a prevalence of 25% dermatologic complications. There have been reported cases of new-onset vitiligo following coronavirus infection (Herzum et al., 2022. Kasmikha et al., 2023. Vigilizzo et al., 2023). Symptoms of vitiligo have been noticed to appear 7-10 days following a COVID-19 infection (Kasmikha et al., 2023).

Janodia and colleagues (2022) documented instances of guttate psoriasis emerging for the first time following a COVID-19 infection, and they introduced certain cases of psoriasis flare-up after infection. Additionally, Dadras (2021) reported a case of pustular psoriasis following the infection with a latency period of 26 days. In their systematic review, Aram et al. (2021) examined case reports detailing dermatological complications associated with COVID-19. Their findings indicate that psoriasis and alopecia areata were the most predominant presentations.

Reports of simultaneous COVID-19 infection and dermatomyositis have been documented (Borges et al., 2021). However, Albakri et al. (2022) reported dermatomyositis flare-up symptoms six weeks after the infection. New cases of dermatomyositis following infection were reported by Hadis and colleagues (2022) and Hozler et al (2022). The patient introduced by Hadis had a latent period of two months after infection. Ortega et al. (2021) documented a new case of dermatomyositis in an 11-year-old girl four months following the coronavirus infection.

In their report, Kim et al. (2023) presented a case of pemphigus vulgaris occurring concurrently with infection symptoms. After contracting COVID-19, there have been new instances of pemphigus vulgaris, with a latency period ranging between 14 and 40 days (Medeiros et al. 2021. Zou et al. 2022. Pastukhova et al. 2024). Additionally, Xie et al. (2023) reported a case of pemphigus foliaceus after the infection presentations. In their review article, Aram et al. (2021) presented a case of pemphigus vulgaris, though the patient tested positive during hospitalization for bullous lesions and stated to have simultaneous conditions.

Lim et al. (2023) evaluated 354527 patients with coronavirus infection and found a 1.12% prevalence of alopecia areata and 1.74% for alopecia totalis post-coronavirus infection. They also indicated a correlation between infection severity and the risk of alopecia totalis. Christensen et al. (2022) investigated the association between coronavirus and alopecia areata. They concluded that alopecia could be a dermatologic coronavirus manifestation or appear 1-2 months after the infection. Additionally, Nguyen et al. (2022) mentioned that the common forms of alopecia associated with COVID-19 are androgenic alopecia, telogen effluvium, and alopecia areata.

5. Hematologic Diseases

In their review, Yazdanpanah et al. (2022) reported cases of autoimmune hemolytic anemia and immune thrombocytopenic purpura (ITP) following COVID-19 infection. Alharbi's meta-analysis (2022) found that ITP associated with coronavirus infection was more prevalent among male and older patients (mean: 59.5± 19), and the mean latency period was 18.1± 21.9 and up to 125 days.

A systematic review by Jacobs et al. (2022) on hemolytic complications associated with infection revealed a slight male predominance. The onset of hemolysis occurred within 0-20 days from the initial onset of infection (median time of 7 days).

Taherianfard et al. (2021) reviewed cases of infected patients who have developed hematologic conditions. According to their results, ITP was the most common disorder. Ono et al. (2024) investigated cases of ITP after COVID-19 infection. The findings revealed that the median age of affected individuals was 61 years, with 12% of cases occurring in pediatric patients. Moreover, over half of the patients presented with moderate to severe symptoms. Additionally, thrombotic thrombocytopenic purpura (TTP) patients have been described following COVID-19 infection (Tehrani et al., 2021. Mushtaq et al., 2023). The Chaudhary review (2022) found that 72% of TTP cases were in females. The mean age was 48.2 years. The average time from COVID-19 symptoms to hematologic presentations was ten days, and 27.3% of cases exhibited neurological symptoms.

6. Gastrointestinal and Hepatic Diseases

Cases of ulcerative colitis (UC) have been presented after COVID-19 infection (Aydin et al., 2021. Taxonera et al., 2021. Elbadry et al., 2022. Kartsoli et al., 2022). Senthamizhselvan (2021) and Tursi (2022) reported Crohn's

disease triggered by a coronavirus infection. Additionally, Kim et al. (2023) presented cases of pediatric patients with inflammatory bowel disease (IBD) following infection.

During their cohort investigation, Hileman and colleagues (2024) assessed the risk of new IBD among 3,908,592 individuals after contracting COVID-19. They found an adjusted risk ratio of 0.84 for Crohn's disease and 1.25 for UC. According to the cohort study by Syed et al. (2023), there was evidence to suggest that COVID-19 infection might act as a trigger for the onset of IBD. Inokuchi et al. cohort study (2024) expressed a notable risk for autoimmune diseases, such as IBD conditions, which remained significantly elevated even after 56 weeks from the infection onset.

By running an observational study, Cakir et al. (2022) stated the increased frequency of celiac disease in pediatrics during the pandemic in Turkey. Interestingly, Mostafavi et al. (2023) described a new case of celiac in combination with autoimmune hepatitis three months after COVID-19 presentations in a 13-year-old female individual. However, Lexner et al. (2023) did not find any increase in the prevalence of celiac disease in their retrospective observational study in Sweden.

Cases of autoimmune hepatitis have been outlined following COVID-19 infection (Kabacam et al., 2021. Durazo et al., 2022. Volchkova et al., 2022. Martini et al., 2023). Balraj (2021) and Cunha-Silva (2023) documented instances of overlap syndrome involving autoimmune hepatitis and primary biliary cholangitis in a patient who had previously experienced symptoms of infection. The patient in Balraj's case had a latency period of approximately one month.

7. Neurologic Disorders

It has been proposed that the coronavirus may affect the nervous system through various mechanisms. The virus could enter the brain through the bloodstream and immune cells. Also, it can reach the brain through the olfactory, vagus, and trigeminal nerves. Additionally, it could disrupt the blood-brain barrier (Kempuraj et al., 2024). During the initial stage of an infection, the nervous system can be impacted by a lack of oxygen, inflammatory response, and hypercoagulable state, leading to conditions such as acute encephalopathy, encephalitis, cerebrovascular disease, and Guillain-Barre syndrome (Bridwell et al. 2020. Lodigiani et al., 2020. Poyiadji et al., 2020. Sedaghat et al., 2020). Some authors found that approximately 36% of their infected patients presented with neurological symptoms. These findings were more prevalent in severe cases of infection. They also revealed a potential relationship between anosmia or dysgeusia and neurological findings (Mao et al., 2020). According to Li et al. (2023), encephalopathies and stroke are cited as the most frequent complications of COVID-19. Luo et al. (2022) reported an approximate 2.0% risk of ischemic strokes in infected patients in their meta-analysis. Hippisley et al.'s (2021) examination of 12 million UK patients revealed an elevated susceptibility to arterial and venous thromboembolism, as well as an increased likelihood of stroke and cerebral venous sinus thrombosis.

Delayed complications such as headaches, brain fog, and mood disorders have been diagnosed, and the incidence of these symptoms does not correlate with the severity of the acute infection (Dangayach et al., 2022). Other neurologic issues, like sleep disorders and dizziness, have been reported (Pinzon et al., 2022). Based on the meta-analysis by Yassin et al. (2021), neurological sequelae have been reported at a rate of 2.5%. On the other hand, neurological sequels have been reported in pediatric-infected individuals. Antoon et al. (2022) revealed a frequency of 7.0% of neurological complications in this group. In the upcoming chapters, we will assess the typical neurological complications associated with COVID-19 as well as autoimmune nervous system complications.

7.1. Neuropathic Pain

Burakzagi et al. (2022) reported cases of neuropathic pain as a presentation of long COVID-19. In an observational cohort, Odozor and colleagues (2022) found a higher risk of peripheral neuropathy within three months post-infection. Montes et al. (2022) follow-up of 77 hospitalized patients for a mean of six months after discharge showed a prevalence of 25% neuropathic pain disorders in their study population. Joshi et al. (2022) conducted a

meta-analysis assessing reported cases of neuropathic pain. They discovered that the onset of symptoms could be from 15 days before to 45 days after respiratory symptoms. Stefano and colleagues (2023), in their meta-analysis, reported a frequency of 0.4%-25% with a pooled estimate of 10% for neuropathic pain after COVID-19 infection. Odozor et al. (2022) conducted an observational study to evaluate the sensory complications of infection. The study revealed that these complications manifested in 1-6% of cases subsequent to a COVID-19 infection, exerting a more pronounced effect on the lower extremities.

7.2. Cognitive Impairment

By conducting a systematic review, Fanshawe et al. (2024) assessed articles regarding cognitive issues in recovered COVID-19 individuals. They found an increased risk of moderate impairment in various aspects of cognition from one month to one year after recovery. They suggested that this damage may remain over time and should be considered by healthcare workers. The results of a meta-analysis performed by Perrottelli and colleagues (2022) are surprising. They included 72 studies in their review and found generalized cognitive impairment in 31 works. According to their findings, executive functions and memory were the most affected areas. Regarding memory assessment, working memory and visuospatial were more influenced. In individuals having a single impairment, attention was the prominent affected domain. Speed processing, that is, the ability to concentrate and cognitive speed, was severely affected by coronavirus. The results of the meta-analysis of Tavares et al. (2020) suggested the likelihood of cognitive decline in mildly infected patients even six months after COVID-19. Jaiswal et al. (2019) explored that Parkinson's disease may get worse due to COVID-19 infection.

7.3. Demyelinating Complications

Schulta et al. (2021) conducted a comprehensive review of 20 reported transverse myelitis cases in 2020. They found a mean latency period of 10 days from infection onset, and the majority of cases had mild symptoms of infection. However, transverse myelitis has been reported after COVID-19 infection with different latency periods; as in Adamec et al. systematic review (2022), most cases had an intermediate (8-21 days) latency duration.

Mahmoud and colleagues (2023) evaluated 32 cases of Guillain-Barre syndrome after infection. They reported a median latency period of 11.5 days in patients. Pimentel et al. (2023) included 436 case reports of Guillain-Barre syndrome in their meta-analysis. Their investigation revealed an average latent period of 19 days. Regarding the symptoms, most cases presented with generalized weakness, and both lower and upper limb involvement was common and the facial nerve was the most common cranial nerve affected in these patients. Respecting autonomic manifestations, blood pressure abnormality was more common. Neophytou et al. (2023) performed a meta-analysis of individuals with Miller-fisher syndrome following COVID-19 infection. They found an average latency of 11 days from infection onset and neurologic symptoms, and 95% of patients complained of ophthalmoplegia, while 73% presented with the classic triad of disease.

Zelada-Rios et al. (2021) collected 30 case reports of disseminated encephalomyelitis. They found a latency period of 10-54 days after infection and neurologic presentations. The condition was not associated with the severity of respiratory symptoms, and they stated cases of affected children. Manzano et al.'s systematic review (2021) revealed higher mortality in cases of demyelinating encephalitis after COVID-19 infection. However, two-thirds of the cases in their study had severe respiratory symptoms requiring intensive care unit admission.

In 2021, some systematic reviews investigated the influence of COVID-19 on neuromyelitis optica spectrum disorders and multiple sclerosis (Aghajanian et al., 2024. Seyedmirzaei et al., 2024. No increase in relapse rates or new disease cases was found in their studies. Lotan and colleagues (2022) conducted a comprehensive review of inflammatory demyelinating diseases of the central nervous system, encompassing 67 articles. They discovered an average latency period of 11.5 days (ranging from 6 to 90 days) from the onset of infection to the development of neuromyelitis optica spectrum disorders. Indeed, the researchers identified an average latency period of 13.5 days, with a maximum duration of 180 days after COVID-19 disease. They reported a relatively low rate of demyelinating disorders in relation to the infection rate. Parsonage-Turner syndrome, an inflammatory condition of the brachial plexus, also has been reported following infection (Cornea et al., 2023).

8. Conclusion

COVID-19 has proven to be a complex condition, with acute manifestations and the potential to trigger autoimmune conditions and influence the nervous system through different mechanisms**.** Dysregulation of the immune system can result in early or late complications that impact nearly every system within the body. This might lead to the development of conditions such as diabetes, autoimmune thyroiditis, and different connective tissue disorders. Additionally, it can lead to demyelinating diseases and non-immunologic neurological conditions like neuropathic pain and cognitive decline**.** As we move forward, it is essential to acknowledge the potential for various complications in both immediate and delayed scenarios. Early recognition and timely management of these issues are essential. Further research is needed to fully comprehend the exact mechanisms of these conditions and to develop policies for prevention and treatment in affected individuals.

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