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LEADING TOPIC

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SARS-CoV-2 and the nervous system

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As guest editor of the current issue of Leading Topic published in the Polish Journal of Neurology and Neurosurgery, I am pleased to present a series of articles regarding the influence of SARS-CoV-2 infection on the nervous system, the occurrence of post-COVID-19 neurological symptoms, and the response to vaccination against COVID-19 in patients suffering from neurological disorders.

SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), the pathogen causing COVID-19 disease (coronavirus disease 2019), has caused a pandemic that has resulted in unprecedented global health, social and economic consequences. Worldwide, more than 670 million people were taken ill, of whom 6,823,598 have died. In Poland, the first case of COVID-19 was recorded on 4 March, 2020, 6,378,299 people have fallen ill, and over 118,000 have lost their lives for this reason (data correct as of 29 January, 2023) [1].

SARS-CoV-2 is a single-stranded sense RNA virus belonging to the family Coronaviridae. There are four major structural proteins in the viral particle: the spike (S protein — responsible for binding to the host cell receptor), membrane (M), envelope (E), and nucleocapsid (N) protein. From the beginning of the pandemic, SARS-CoV-2 has evolved numerous times, and mutations to the viral genome have significantly affected its pathogenic potential. Mutations of the virus connected with the highest threat to the population have been named by the World Health Organisation: 'Variants of Concern' (VOCs). Omicron, the current SARS-CoV-2 mutation, has been the most mutated VOC so far [2].

Although SARS-CoV-2 is not a classic neurotropic virus, neurological symptoms in COVID-19 patients have been reported since the outbreak of the pandemic. The maintenance of homeostasis within the central nervous system (CNS) is secured by a number of protective mechanisms, among which the blood-brain-barrier (BBB) plays a key role. Passage through the BBB is rigorously restricted through physical (intercellular and adherence junctions) and metabolic barriers [3]. BBB disruption is one of the key pathological processes involved in various diseases of the CNS [4, 5]. Proinflammatory cytokines and disease-specific proteins (e.g. SARS-CoV-2 derived S protein) can cause BBB disturbances, leading to its increased permeability. Suprewicz et al. [6] have described interactions of the SARS-CoV-2 and its S protein with brain structures and the endothelium that form the BBB. Although SARS-CoV-2 cannot easily cross the BBB, it can enter the CNS through various direct (e.g. using immune cells as a carriage — 'Trojan horse' phenomenon) and indirect (e.g. increased permeability of the BBB caused by chronic hypoxia and cytokine storm) mechanisms.

In the acute period of COVID-19, the most frequently noted problems of the nervous system are: encephalopathy, coma and strokes, as well as headaches and olfactory disorders (Tab. 1). On the other hand, meningoencephalitis and encephalitis have been rarely diagnosed [7–9]. Predisposing factors for the onset of neurological disorders include older age, male sex, Caucasian race, and a history of neurological disease. The co-occurrence of encephalopathy, coma or stroke during SARS-CoV-2 infection significantly increases the risk

Table 1. The most common neurological and psychiatric disorders in patients with COVID-19 [7, 35]

In the acute period of COVID-19	At 6 months after COVID-19
Encephalopathy	Anxiety disorders
Stroke	Mood disorders
Coma	Insomnia
Headache	Cognitive deficit ('brain fog')
Olfactory and gustatory disorders	Psychotic disorders

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of death [7]. The pathomechanism of encephalopathy in the course of COVID-19 is not yet fully understood. However, strokes are usually caused by thromboembolic disorders observed in SARS-CoV-2 infection [10]. Polish neurologists concerned with the treatment of stroke have diligently conducted epidemiological research into stroke in the course of COVID-19 [11–15], and have actively participated in international studies [16].

Headaches and disorders of smell and taste are also frequently reported by patients with COVID-19 [7]. Headaches are symptomatic, and their causative factors may include fever and chronic hypoxia in the course of the underlying disease, or drugs used in its treatment. Straburzynski et al. [17] have attempted to explain the aetiopathogenesis of headaches due to COVID-19 in relation to the innate immune response to viral infection. Some factors in innate immunity have been shown to facilitate headache (e.g. interferons, interleukin (IL) –1-β, IL-6, and tumour necrosis factor). The virus may also cause headache by the activation of pattern recognition receptors (e.g. Toll-like receptor 7). In turn, olfactory disorders in most patients occur in the acute period of COVID-19 and are transient, resolving after a few weeks, or even days. It is assumed that in such cases the cause of anosmia/hyposmia is the depletion of sustentacular cells caused by the SARS-CoV-2 infection and their subsequent restoration by days 10–14 post-infection. However, in some patients, the sense of smell is permanently damaged [18]. This is quite difficult to explain because olfactory sensory neurons do not express a receptor protein for SARS-CoV-2, and are not infected or destroyed by this virus.

However, it has been shown by Zazhytska et al. that the virus can alter cellular morphology and the transcriptome of cells it cannot infect, e.g. olfactory sensory neurons [19]. SARS-CoV-2-mediated nuclear reorganisation is non-cell autonomous. Downregulation of olfactory pathways may explain COVID-19-induced anosmia. These findings help us to understand how it is possible that SARS-CoV-2, which directly infects only about 1% of the patient's body cells, can cause severe damage to so many different organs. The results obtained by Zazhytska et al. [19] provide a potential explanation for neurological disorders caused by a virus with no tropism to neurons.

Analysis of the results of magnetic resonance imaging collected by the UK Biobank showed that patients who had COVID-19, compared to those who did not suffer from the infection, have changes suggesting damage in areas functionally related to the primary olfactory cortex, reduction in the thickness of grey matter in the orbitofrontal cortex and parahippocampal gyrus, and greater generalised brain atrophy. People who have recovered from SARS-CoV-2 infection have also shown impairment of executive functions [20]. These results raise some concerns about the future risk of developing neurodegenerative diseases in patients who have had this infection.

However, as indicated in the article published in this issue [21], based on the analysis of publications that appeared up until the end of November, 2022, post-COVID-19 parkinsonism is very rare, and the described cases of parkinsonism, being temporally related to COVID-19, seem to have a very diverse aetiology. An analysis of the literature [22] indicates that the most common hyperkinetic movement disorders associated with a history of SARS-CoV-2 infection include myoclonus and ataxia, whereas chorea, tremor and dystonia are extremely rare. Interestingly, hyperkinetic disorders found after COVID-19 seem to have an autoimmune basis and respond well to treatment with glucocorticoids and immunoglobulins [23].

Clinical observations indicate that many COVID-19 patients, after the acute period of the disease when the virus RNA is no longer detected in their body, continue to report chronic signs/symptoms resulting from the dysfunction of various organs and systems [24, 25]. The phenomenon of persistence of signs/symptoms four weeks after the onset of SARS-CoV-2 infection has been referred to as 'post-COVID syndrome'. A similar persistence of symptoms after infection was also observed during epidemics caused by other coronaviruses, such as SARS (severe acute respiratory syndrome coronavirus) in 2003 and MERS (Middle East respiratory syndrome) in 2012. Generally, there are two periods of post-COVID syndrome: the interval between the 4^{th} and 12^{th} weeks from the onset of the disease (subacute post-COVID syndrome) and the period beyond 12 weeks from the onset of the first symptoms of COVID-19 (chronic post-COVID syndrome) [26].

Reports on the health consequences of COVID-19 indicate that post-COVID syndrome is relatively common. A follow-up of 488 patients in the USA 60 days after the onset of COVID-19 showed that 32.6% had persistent symptoms, including 18.9% who developed new symptoms or experienced exacerbation of their existing symptoms. The most commonly reported symptoms were shortness of breath when climbing stairs (22.9%), cough (15.4%), and persistent loss of smell and/or taste (13.1%) [27]. A post-acute COVID-19 Italian study [2] showed the persistence of symptoms after approximately 60 days from the onset of COVID-19 in 87.4% of 143 patients. Fatigue (53.1%), dyspnoea (43.4%), arthralgia (27.3%) and chest pain (21.7%) were most frequent, with 55% of the patients reporting at least three symptoms. Quality of life, as assessed in the EuroQol scale [28], was reduced in 44.1% of the respondents. Similarly, a French study which included 150 patients after COVID-19 showed the persistence of symptoms in approximately two thirds of them after approximately 60 days from the onset of the disease [29]. Other studies, including those conducted in the UK (assessment of 110 patients 8–12 weeks after admission to hospital) [30] and in Spain (277 patients 10–14 weeks after disease onset) [31] yielded similar results. Fatigue, shortness of breath, post-traumatic stress disorder, depression, anxiety, trouble concentrating and sleep disturbances were reported in at least 30% of subjects.

Table 2. The risk trajectories of neurological and psychiatric disorders at 2 years after COVID-19 [38]

Risk returned to baseline	Risk still increased
Anxiety disorders	Cognitive deficit ('brain fog')
Mood disorders	Dementia
	Psychotic disorders
	Epilepsy or seizures

In Wuhan, China, a prospective study was conducted (The Post-acute COVID-19 Chinese Study) to assess the long-term consequences of COVID-19 [32]. A total of 1,733 patients were examined six months after the onset of the disease. The follow-up involved an interview (the subjects also completed appropriate questionnaires), physical examination, a 6-minute walk test (6MWT) [33], laboratory blood tests, and in some cases specialist examinations evaluating the function of the respiratory system, computed tomography and ultrasound imaging. Most patients (76%) reported at least one complaint. As in other studies, muscle fatigue/weakness (63%), sleep disorders (26%), anxiety/depression (23%), disorders of smell (11%) and appetite (7%), headache and muscle pain (2%) were most commonly reported. It has been shown that female sex predisposes to fatigue and anxiety/depression [32].

In the course of post-COVID syndrome, symptoms from various organs may occur, but it is the nervous system that is most frequently affected [34]. In order to assess the neurological and psychiatric consequences of COVID-19, a retrospective study was conducted in the USA using electronic medical records. Data from 236,379 patients was analysed. It was shown that within six months of contracting COVID-19, 33.62% of them had a neurological or psychiatric diagnosis, and in 12.8% this diagnosis was made for the first time (Tab. 1). A more severe course of COVID-19 increased the risk of developing a disease of the nervous system — a neurological or psychiatric issue was found in 46.42% of patients who required hospitalisation in an intensive care unit due to COVID-19 [35].

Most observations indicate that a more severe course of SARS-CoV-2 infection (defined, for instance, as the need for hospitalisation in an ICU or the need for passive and/or active ventilation) predisposes to post-COVID syndrome [30, 32, 36]. This fact is also confirmed by the observation of Polish patients suffering from multiple sclerosis (MS) treated with disease-modifying drugs who have survived SARS-CoV-2 infections [37].

Taquet et al., continuing their previous retrospective study [35] over a longer 2-year period, analysed the persistence of various nervous system ailments in a group of 1,284,437 patients who had recovered from COVID-19. It was shown that the increased incidence of mood and anxiety disorders was transient. However, the increased risk of psychotic disorder, cognitive deficit, dementia, epilepsy or seizures persisted throughout (Tab. 2). The differing trajectories may suggest a different pathogenesis for these outcomes. The neurological and psychiatric outcomes were similar during the Delta and Omicron variant waves [38].

In turn, Chatys-Bogacka et al., retrospectively using a neuropsychological questionnaire specially designed for this purpose, searched for demographic factors influencing the incidence and course of post-COVID symptoms over time. They found that in patients who did not require hospitalisation due to COVID-19, the occurrence and duration of COVID-19-associated brain fog depended on sex. Women reported more frequent problems with writing, reading, counting and communicating thoughts. However, there were no sex-related differences in experiencing problems with multitasking, remembering information from the past, determining the current date or field orientation [39]. Similarly, retrospectively assessing post-COVID fatigue in patients requiring prior hospitalisation due to COVID-19, Mazurkiewicz et al. found this symptom in the majority of patients, and noted that the presence of fatigue was predicted by female sex [40].

Since the beginning of the COVID-19 pandemic, much attention has been paid to patients suffering from autoimmune diseases, and neurologists have been particularly concerned about patients treated with immunosuppressive drugs [41–43].

In this issue, we also present two articles concerning the impact of SARS-CoV-2 infection on the course of multiple sclerosis [44] and other autoimmune diseases of the nervous system [45]. The introduction of vaccination against SARS-CoV-2 was another challenge in this group of patients. On the one hand, the necessity for vaccinations was beyond doubt [46]; on the other hand, there were concerns about the effectiveness of preventive vaccinations in patients treated with immunosuppressive therapies. Observations of potential side effects of the vaccines used, which ultimately turned out to be safe and well tolerated [47, 48], aroused great interest as well. This edition also includes two articles discussing the immune response to SARS-CoV-2 infection and COVID-19 vaccination [49, 50].

I hope that readers of the Polish Journal of Neurology and Neurosurgery will find this Leading Topic not only interesting but also useful in clinical practice. However, it must be remembered that the story of the COVID-19 pandemic is not yet at an end, and there are still many unknowns about the effects of SARS-CoV-2 on the nervous system.

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