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Autoimmune mediated hyperkinetic movement disorders in SARS-CoV-2 infection — a systematic review

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ABSTRACT

Introduction. Various neurological symptoms have been confirmed in the course of SARS-CoV-2 infection. Some of these are undoubtedly the aftermath of the developing inflammation and increased coagulation processes. However, there is also a group of symptoms that derive from possible autoimmune processes. These include primary hyperkinetic movement disorders such as myoclonus, ataxia, opsoclonus, and tremors. This study systematically reviews scientific reports presenting patients with hyperkinetic movement disorders as one of the neurological symptoms.

Material and methods. The available literature was systematically reviewed as per the recommendations of Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). The PubMed database was used in the range from 1 April, 2020, to 31 July, 2021.

Results. The PubMed database search identified 102 cases of patients with SARS-CoV-2 infection who developed hyperkinetic movement disorders. After excluding patients undergoing mechanical ventilation (n = 46) and a few other cases (n = 7), a group of 49 non-intubated patients was obtained. The mean age of the patients was 57.92 years, and 75.51% of the patients were male. The most common hyperkinetic movement disorders were ataxia (83.67%), myoclonus (67.35%), and tremor (30.61%). Symptoms appeared on average within two weeks of the first symptoms of infection. Most patients had symptoms significantly reduced or withdrawn (67.44%) or early partial improvement (30.23%).

Conclusions. Based on the meta-analysis, it can be concluded that hyperkinetic movement disorders in the course of SARS-CoV-2 infection are an early symptom with a potential autoimmune background. They have a good prognosis with the applied treatment. Further observations are needed to determine their frequency and the most effective methods of treatment.

Key words: Covid, SARS-CoV-2, ataxia, myoclonus, autoimmune

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Introduction

Severe acute respiratory syndrome coronavirus 2 was first detected in December 2019 in Wuhan, China [1]. The pathogen-induced disease syndrome, known as COVID-19, triggered a pandemic that has affected over 156 million people and claimed over 3 million lives [2]. In the classic course of SARS-CoV-2 infection, most patients present with respiratory symptoms and general influenza-like symptoms (myalgia, fever) [3]. Sometimes the disease takes a sparse or asymptomatic form [4]. Nevertheless, 80% of convalescents suffer from various types of long-term ailments [5], in some cases lasting up to months [6]. Various neurological symptoms occurring during COVID-19 have been described, including, for example, loss of smell, loss of taste, mono- and polyneuropathies, encephalopathies, headaches, dizziness, muscle pain, problems with concentration, and chronic fatigue syndrome [3, 7]. The incidence of neurological symptoms varies significantly depending on the methodology used in the study (4.3–73.0%) [7]. In current reports, one of the most common neurological manifestations was headache (32.9–41.1%) and changes

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in taste and smell (18.8–36.8% and 18.2–37.9%) [8, 9]. In the latter case, earlier reports indicated a much higher frequency (88.0% and 85.6%) [10]. In the course of SARS-CoV-2 infection, well-characterised life-threatening neurological syndromes may also occur i.e. stroke, Guillain-Barre syndrome, transverse myelitis, or acute disseminated encephalomyelitis [3, 7, 11–13]. Symptoms that have so far received relatively little attention include various types of primary movement disorders present in COVID-19. Reports have described their occurrence among patients with a frequency of 1.2–2.0% [14]. This type of disorder includes, among others, myoclonus, ataxia, opsoclonus, or tremor [15]. This article aimed to systematically review the available case reports of hyperkinetic movement disorders occurring in the course of COVID-19.

Material and methods

To ensure the highest reliability for this systematic review, the PRISMA recommendations were followed [16]. The time period was 1 April, 2020 to 31 July, 2021. It was limited only to English-language peer-reviewed articles. To search for case descriptions, the PubMed database was used. The following keywords were used: "covid", "sars-cov-2", "coronavirus" in multiple combinations with "ataxia", "myoclonus", and "tremor". Only articles containing case reports that presented a detailed description of neurological symptoms and a confirmed diagnosis of SARSCoV-2 infection using objective laboratory methods were considered acceptable. The effects of searching the PubMed database following the adopted methodology are presented in Fig. 1. During the analysis of each case report, the following information was identified: age, gender, the need for mechanical ventilation, the presence of hyperkinetic movement disorders such as myoclonus, opsoclonus, ataxia and others, time of symptoms from the onset of infection, time of symptoms disappearance, treatment applied, and the occurrence of changes in imaging studies. To prevent potential cases of movement disorders caused by factors other than the SARS-CoV-2 infection itself, patients diagnosed with organic brain damage or a history of neurodegenerative disease were excluded. In addition, paediatric cases were not taken into account, analysing only those concerning adults. Moreover, post-extubated patients were assigned to a separate group for comparison with the target group.

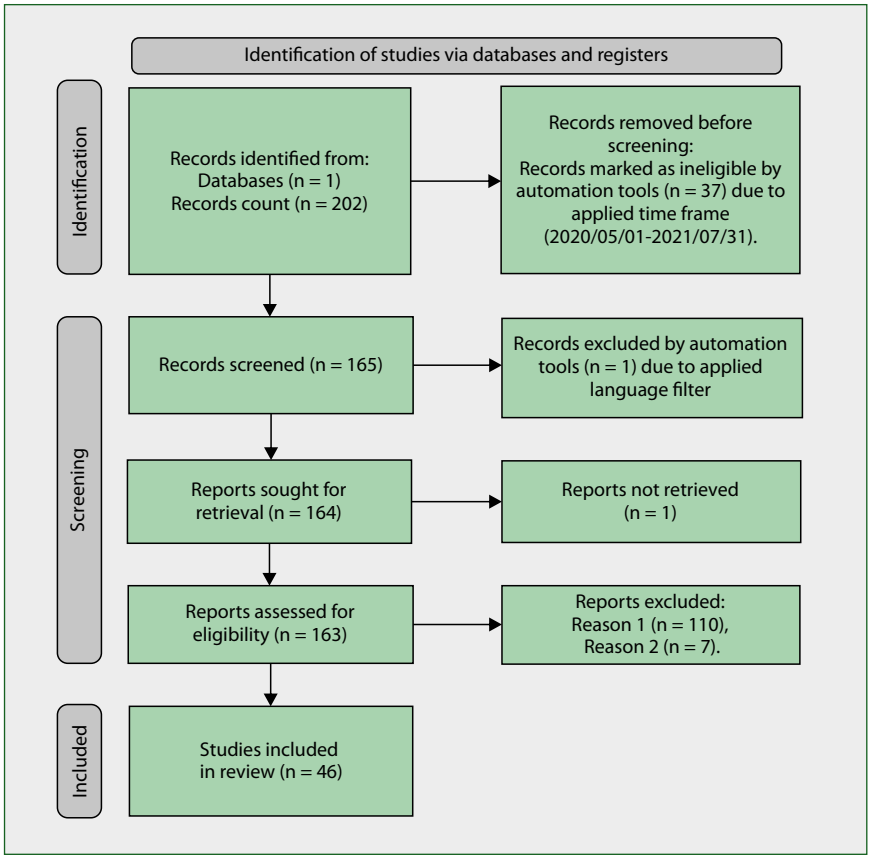

Figure 1. Protocol used during systematic review. Database used: PubMed. Query used: 'sars-cov-2' or 'Covid' or 'coronavirus' and 'ataxia' or 'myoclonus' or 'tremor'. Reason 1 — not detailed case description, not case report. Reason 2 — significant organic damage to central nervous system, or absence of information as to identification of pathogen

Results

It was possible to identify 38 articles describing 49 COVID-19 non-intubated patients with various types of hyperkinetic movement disorders (Tab. 1). These cases were included into a primary group and further analysed in detail. This primary group did not include cases of people undergoing mechanical ventilation, or with significant organic damage to the central nervous system, or in the absence of information as to the identification of the pathogen; a total of 53 cases [12, 13, 27, 40, 46, 55–64]. 46 of them were intubated, three patients did not have a SARS-CoV-2 confirmation test, in two patients the primary diagnosis was a stroke, one case presented with subacute thyroiditis, and one case concerned a patient with multiple system atrophy. To compare to the primary group, a group of post-extubated patients was briefly characterised.

In the descriptions of all non-intubated cases (n = 49), the neurological symptoms present were listed. The most common hyperkinetic movement disorder was ataxia, followed by myoclonus, tremor, and opsoclonus (Tab. 2). Of all case reports, 75.51% were male. The mean age at onset of the disease slightly differed depending on gender: 55.84 years for men and 64.33 for women. In the group of n = 47 case reports, information on imaging tests performed on the central nervous system was included. In 87.23% (n = 41), the test result showed no clinically significant changes. Except for two cases of cerebellitis, in the remaining cases the detected changes were non-specific (Tab. 1). In the group of n = 28 case reports, it was possible to define the exact onset of hyperkinetic movement disorders to the classically occurring symptoms of upper respiratory tract infections. In 32.14% (n = 9) of patients, hyperkinetic movement disorders occurred along with influenza-like symptoms. In the remaining 67.86% (n = 19), hyperkinetic movement disorders appeared along with the resolution of influenza-like symptoms, on average 3.32 days (1–12) after. In the group of n = 45 case reports, pharmacotherapy was reported. In the treatment of hyperkinetic movement disorders, anti-seizure drugs were used — clonazepam (33.3%, n = 15), levetiracetam (28.89%, n = 13), and valproic acid (15.56%, n = 7). In 82.22% (n = 37) of patients, immunomodulatory drugs, including intravenous immunoglobulins (56.56%, n = 25) and steroids (56.56%, n = 25), were also used. In the group of n = 43 case reports, it was possible to establish the further course of the hyperkinetic movement disorders. Complete or significant improvement was achieved by 67.44% (n = 29) of patients. On the other hand, partial improvement was achieved by 30.23% (n = 13), and in one case, (2.33%), no improvement was observed in the 2-month follow-up.

Of the patients undergoing mechanical ventilation, 39 were men (84.78%), and seven were women (15.22%). The mean age was 59.87 (28–85); 59.13 (28–85) for men, and 64.00 (53–72) for women. In available detailed data, hyperkinetic movement disorders occurred on average 7.78 days (n = 18, 1–34) after extubation. However, in some cases they occurred while reducing the dose of anaesthetic agents. 80.43% (n = 37) of them had myoclonus, 17.39% (n = 8) tremors, 15.22% (n = 7) ataxia, and 6.52% (n = 3) opsoclonus. Simultaneous ataxia and myoclonus occurred in only four cases (8.70%) and myoclonus plus opsoclonus in one (2.17%).

Discussion

To the best of my knowledge, this is the first systematic review of available cases of hyperkinetic movement disorders caused by autoimmune processes in the course of SARS--CoV-2 infection. The mere estimation of the occurrence of hyperkinetic movement disorders in the course of SARS--CoV-2 infection is difficult due to the relatively rare listing of these symptoms in the available reports. The incidence of ataxia among all patients in three cohort studies was estimated at 0.3%–5.0% [65–67]. In a total of 98 cases of patients with neurological symptoms, 3% presented myoclonus [7]. One report assessing a group of 646 COVID-19 patients showed that 22.7% of them developed only neurological symptoms. Also, despite the seemingly infrequent occurrence of movement disorders in this disease, undoubtedly attention should be paid to them, especially in seasons of high morbidity. Interestingly, in the analysed case reports, the occurrence of opsoclonus was always associated with the simultaneous presence of myoclonus and ataxia (Tab. 1). Myoclonus occurred more often with accompanying ataxia than without it (28.57% vs. 16.33%).

Therefore, it can be expected that hyperkinetic movement disorders in the course of SARS-CoV-2 infection will take the form of a symptom complex more often than an isolated manifestation. Undoubtedly, opsoclonus-myoclonus-ataxia syndrome belongs to this type of syndrome [19]. However, one should be vigilant due to its possible paraneoplastic background [10]. It should also be noted that the opsoclonus--myoclonus-ataxia syndrome occurred in 22.45% of patients, whereas, in the group of post-intubated patients, there was no such case.

In the analysed case reports, myoclonus was characterised by a different course: they appeared both in generalised and isolated form, action and resting, and there were also differences in their severity. Unfortunately, few studies have analysed the exact genesis of the emerging myoclonus, and those that have done so indicated both their cortical and subcortical sources [45, 55, 57]. Furthermore, in some cases, the EEG record remained without clinically significant changes [24, 34]. This indicates a variable genesis of myoclonus depending on the brain structures involved. Ataxia accompanied other neurological symptoms in 83.67% of all non-intubated cases. Thus, the cerebellum remains a structure frequently affected by the pathology of autoimmune processes [68], and its participation in the pathogenesis of cortical myoclonus has been reported [69]. However, it has not yet been demonstrated that myoclonus and ataxia occur in patients with a more severe course of COVID-19 [45].

Table 1. Presentation of available case reports

Publication	Age	Sex	Movement disorders	Symptoms onset day[1]	Treatment	Changes in CT/MRI of brain	Resolution of movement disorders
Emamikhah M. et al. [17]	51	M	Myoclonus, opsoclonus, ataxia, voice tremor	14	CLO, LEV, HCHL, IVIG	No	Total after 4w
Emamikhah M. et al. [17]	54	M	Myoclonus, ataxia	4	VAL, LEV, IVIG	No	Partial after 1w
Emamikhah M. et al. [17]	52	M	Myoclonus, ataxia, voice tremor	16	VAL, CLO, lopinavir, ritonavir, HCH	No	Partial after 2m
Emamikhah M. et al. [17]	42	F	Myoclonus, ataxia, voice tremor	10	VAL, CLO	N/A	N/A
Emamikhah M. et al. [17]	44	M	Myoclonus, opsoclonus, ataxia, voice tremor	3	VAL, CLO, IVIG	No	Total after 2m
Emamikhah M. et al. [17]	52	M	Myoclonus, ataxia, voice tremor	21	CLO, IVIG	No	Significant after 4w
Emamikhah M. et al. [17]	39	M	Myoclonus, opsoclonus, ataxia, voice tremor	10	VAL, CLO, LEV, IVIG	No	N/A
Werner et al. [18]	62	M	Ataxia, scanning speech	16	Acyclovir, STER	Cerebellar atrophy	Total after 17w
Fernandes J. et al. [19]	58	F	Myoclonus, opsoclonus, ataxia, tremor	17	Acyclovir, CLO, IVIG, STER	No	N/A
Foucard C. et al. [20]	63	M	Myoclonus, ataxia	42	IVIG	No	Significant after 1w
Foucard C. et al. [20]	83	M	Myoclonus, opsoclonus, ataxia	10	IVIG, STER, Diazepam	No	Significant after 1w
Schellekens MMI. et al. [21]	48	M	Myoclonus, ataxia	13	LEV	No	Partial after 62d
Urrea-Mendoza E. et al. [22]	32	M	Myoclonus, opsoclonus, ataxia	12	VAL, CLO, STER	No	Significant after 24d
El Otmani H. et al. [23]	59	M	Myoclonus	N/A	VAL, IVIG, STER	No	Total after 21d
Chan JL. et al. [15]	44	M	Myoclonus, ataxia, dysarthria	12	CLO, LEV, STER	No	Total after 25d
Borroni B. et al. [24]	80	M	Myoclonus	22	LEV	No	Total after 3d
Borroni B. et al. [24]	54	F	Myoclonus	14	CLO	N/A	Partial after 50d
Grimaldi S. et al. [25]	72	M	Myoclonus, ataxia, dysarthria	17	CLO, IVIG, STER	No	Significant after 37d
Wright D. et al. [26]	79	M	Myoclonus, opsoclonus, ataxia	13	N/A	Chronic ischaemic changes	Partial after 32d
Anand P. et al. [27]	71	M	Myoclonus, ataxia	7	VAL, LEV	Diffuse meningeal hyper-intensity	Total after 14d
Delorme C. et al. [28]	72	M	Myoclonus, ataxia	15	IVIG	No	Total after 6w
Delorme C. et al. [28]	66	F	Ataxia, bradykinesia	7	IVIG, STER	No	Total after 41d
Delorme C. et al. [28]	60	F	Ataxia, akathisia	1	STER	No	Total after 20d
Dijkstra F. et al. [29]	44	M	Myoclonus, ataxia	14	IVIG, STER	No	Total after 2m
Sanguinetti SY. et al. [30]	57	M	Myoclonus, opsoclonus, ataxia, limb tremor	10	CLO, IVIG, STER	No	Partial after 18d
Khoo A. et al. [31]	65	F	Myoclonus	21	CLO, LEV, STER	No	Partial after 31d
Balestrino R. et al. [32]	63	M	Ataxia	1	lopinavir, ritonavir, HCHL, STER	No	Total after 3w
Diezma-Martín AM. et al. [33]	70	M	Ataxia, limbs tremor, voice tremor	17	CLO, HCHL, STER	No	Partial after 1m

[1]Day of movement disorders onset since COVID-19 first symptoms; VAL — valproic acid; LEV — levetiracetam; CLO — clonazepam; IVIG — intravenous infusion of immunoglobulins; STER — steroids; HCHL — hydroxychloroquine; N/A — no data available; d — day; w — week; m — month →

Table 1 cont. Presentation of available case reports

Publication	Age	Sex	Movement disorders	Symptoms onset day[1]	Treatment	Changes in CT/MRI of brain	Resolution of movement disorders
Rábano-Suárez P. et al. [34]	88	F	Myoclonus	21	STER	No	Total after 3d
Manganotti P. et al. [35]	50	F	Ataxia, ophthalmoplegia	16	lopinavir, ritonavir, HCHL, IVIG	No	Total after 7d
Manganotti P. et al. [36]	49	F	Ataxia, ophthalmoplegia	14	lopinavir, ritonavir, HCHL, IVIG, STER	No	N/A
Lantos JE. et al. [37]	36	M	Ataxia, ophthalmoplegia	2	HCHL, IVIG	Increase in T2 signal within third cranial nerve	Partial after 6d
Fadakar N. et al. [38]	47	M	Ataxia, dysarthria	3	lopinavir, ritonavir	Increase in T2 signal of both cerebellar hemispheres	Partial after 1m
Kopscik MR. et al. [39]	31	M	Ataxia, ophthalmoplegia	N/A	plasma, tocilizumab, IVIG	No	No improvement after 2m
Perrin P. et al. [40]	67	M	Ataxia, tremor	11	HCHL, STER	No	Total after 5d
Perrin P. et al. [40]	64	M	Ataxia, tremor	13	lopinavir, ritonavir, IVIG, STER	Leukoencephalopathy signs	Total after 16d
Povlow A. et al. [41]	30	M	Ataxia, dysarthria	1	N/A	No	Partial after 10d
Gutiérrez-Ortiz C. et al. [42]	50	M	Ataxia	3	IVIG	No	Total after 2w
Fernández-Domínguez J. et al. [43]	74	F	Ataxia, ophthalmoplegia	24	lopinavir, ritonavir, HCHL, IVIG	No	Partial after 12d
Fernando EZ. et al. [44]	38	M	Myoclonus	14	lopinavir, ritonavir, HCHL	No	Total after 7d
Shetty K. et al. [45]	41	M	Myoclonus, ataxia	10	CLO, LEV, STER	No	Significant after 8w
Blanco-Palmero VA. et al. [46]	76	M	Myoclonus	2	N/A	No	N/A
Blanco-Palmero VA. et al. [46]	88	F	Myoclonus	25	N/A	No	N/A
Giannantoni NM. et al. (2021) [47]	67	M	Myoclonus, ataxia, voice tremor, dysarthria	11	CLO, LEV, STER	No	Total after 14d
Przytuła F. et al. [48]	49	M	Myoclonus, ataxia, voice tremor, hand tremors	11	CLO, LEV, STER	No	Significant after 3w
Przytuła F. et al. [48]	62	M	Myoclonus, opsoclonus, ataxia	11	IVIG, STER	No	Significant after 2w
Saha B. et al. [49]	78	K	Myoclonus, opsoclonus, ataxia	N/A	LEV, IVIG, STER	No	Significant after 10d
Ishaq H. et al. [50]	63	M	Myoclonus, opsoclonus, ataxia	23	IVIG, STER	No	Total after 4w
Emekli AS. et al. [51]	54	M	Ataxia, hand tremors, dysarthria	14	favipiravir, IVIG, STER, metoprolol	Increase in T2 signal of both cerebellar hemispheres	Partial after 1m

[1]Day of movement disorders onset since COVID-19 first symptoms; VAL — valproic acid; LEV — levetiracetam; CLO — clonazepam; IVIG — intravenous infusion of immunoglobulins; STER — steroids; HCHL — hydroxychloroquine; N/A — no data available; d — day; w — week; m — month

Table 2. Clinical features of intubated and post-extubated patients

	Non-intubated group	Post-extubated group
Mean age (years)	57.92 (30–88)	59.87 (28–85)
Men/women	n = 37 (75.51%) / n = 12 (24.49%)	n = 39 (84.78%) / n = 7 (15.22%)
Myoclonus	n = 33 (66.35%)	n = 37 (80.43%)
Opsoclonus	n = 11 (22.45%)	n = 3 (6.52%)
Ataxia	n = 41 (83.67%)	n = 7 (15.22%)
Tremor	n = 15 (30.61%)	n = 8 (17.39%)
Myoclonus and ataxia	n = 14 (28.57%)	n = 4 (8.70%)
Myoclonus and opsoclonus	n = 0 (0.0%)	n = 1 (2.17%)
Myoclonus and opsoclonus and ataxia	n = 11 (22.45%)	n = 0 (0.0%)

The obtained data indicates that hyperkinetic movement disorders in the course of COVID-19 are an early complication. They occurred in 32.14% of non-intubated patients still during the duration of influenza-like symptoms, and in 67.86% appeared almost immediately after their disappearance: on average 3.32 days after.

The fact that a large majority of cases (75.51%) with hyperkinetic movement disorders were males is also noteworthy. This is in line with the available data, which shows that men are less tolerant of the SARS-CoV-2 infection, with higher mortality (59.0–70.3%) [70, 71]. It is worth noting that this is also confirmed by the even higher percentage of men (84.78%) in the group of mechanically ventilated patients. It has been reported that women produce a stronger immune response to infections than men. This is due to, among other things, the result of the documented action of oestrogens, progestagens, and androgens [72]. It is also worth mentioning that another neurological disorder characteristic of COVID-19, anosmia, is more common in women — 63.1% [73]. Patients presenting this symptom are characterised by lower mortality [74] and less frequently manifested influenza-like symptoms [75].

Interestingly, in the imaging studies of the central nervous system, in the vast majority of cases, no changes were visible (87.23%). Where they occurred, they did not show any specific pattern, apart from two cases of cerebellitis (Tab. 1). Previous reports indicate that ischaemic changes are the most frequently identifiable changes in the central nervous system in the course of COVID-19 [76]. In cohorts of patients presenting with neurological symptoms, MRI lesions were detected in 58.9%, of which 34.6% were pathologies characteristic of ischaemic or haemorrhagic stroke [77].

The lack of significant changes in imaging studies, and the fact that 82.22% of patients received immunomodulatory treatment achieving an improvement in their clinical condition, indicate an infection-associated, autoimmune basis of hyperkinetic movement disorders. This is consistent with the opinions of other researchers [34]. One of the arguments for the autoimmune background is the presence of opsoclonus-myoclonus syndrome. In its pathogenesis has been described the involvement of antibodies that react against cerebellar Purkinje cells [78]. Moreover, a recent study showed that a patient diagnosed with autoimmune encephalitis during SARS-CoV-2 infection presented high titres of serum and CSF IgG autoantibodies against the Purkinje cells nuclei, striatal neurons, and hippocampal neurons [79]. Together with another report of a patient with anti-GAD antibody-associated cerebellitis developed after COVID-19 [51], it can be concluded that the cerebellum participates in the development of hyperkinetic movement disorders in the course of autoimmune response to SARS-CoV-2 infection. It has been established that SARS-CoV-2 gains entry into a cell through angiotensin--converting enzyme 2 receptors, which are expressed in glial cells and neurons [80]. Therefore, it remains possible that the brainstem, cerebellar tracts, and deep cerebellar nuclei are targets of direct neural invasion and the subsequent immune response. A coexisting cause could be the susceptibility of these regions to the SARS-CoV-2 mediated cytokine storm inflammation [81].

Further reinforcement of the autoimmune theory comes from the fact that some of the ataxia cases occurred as a component of Miller-Fisher syndrome [35, 37, 42, 43]. There have also been reports of neurological syndromes classically associated with autoimmune responses, such as ADEM and optic neuritis with anti-MOG antibodies [82]. An additional argument for the autoimmune origin comes from a post-mortem case series that found neuropathological changes predominantly in the brainstem and cerebellum, compatible with autoimmune encephalitides [83]. However, it is worth noting that in some cases of hyperkinetic movement disorders, there were spontaneous remissions without the use of treatment [21], and that the toxic effects of some of the drugs used, or pure coincidence, cannot be ruled out. The development of hyperkinetic movement disorders should also consider cerebrovascular disease, which is a secondary cause in 20% of cases [84]. Although most of the analysed patients did not have any changes in imaging tests, chronic vascular changes

were shown in at least one patient [55]. In addition, small vessel disease, particularly involving deep brain structures, is the most common subtype of ischaemic lesion associated with secondary movement disorders [85].

Some reports also point out that SARS-CoV-2 infection can cause an imbalance in the intestinal microflora and cause inflammation on the gut-brain axis [86]. Moreover, the receptor for angiotensin-converting enzyme type 2 (ACE-2) associated with one of the possible routes of viral entry is highly expressed in enterocytes of the small intestine [87]. Intestinal dysbiosis and intestinal inflammation are some of the suggested links between the pathogenesis of Parkinson's Disease (PD) and other neurological disorders [88]. In addition, it has been independently reported that over-activation of the renin-angiotensin system may potentiate the microglial response and the associated oxidative stress, which may translate into increased degeneration of dopaminergic neurons [89].

Therefore, it cannot be ruled out that the primary inflammatory processes in the course of SARS-CoV-2 infection are also an element of the complex mechanism of the development of hyperkinetic movement disorders. However, there is currently no data indicating a risk of developing PD following viral upper respiratory tract infection [90]. There are also studies indicating an increase in the incidence of psychiatric disorders during the COVID-19 pandemic [91]. Some reports show that the manifested hyperkinetic movement disorders may have functional origins [92].

To exclude possible neurological symptoms caused by central nervous system hypoxia, patients undergoing mechanical ventilation were not considered in the primary analysed group. Significant hypoxia remains a known causative factor in the occurrence of myoclonus [93], as are some of the anaesthetic agents [94]. This is consistent with the obtained data, which showed that as many as 80.43% of mechanically ventilated patients presented myoclonus. Furthermore, in 69.57% of those cases, it was the only symptom of hyperkinetic movement disorders. Moreover, patients staying in the intensive care unit may develop critical polyneuropathy and myopathy, which may affect the proper assessment of hyperkinetic movement disorders [57].

Future directions

Based on this systematic review, it can be concluded that hyperkinetic movement disorders in the course of SARS-CoV-2 infection are an early symptom with a potential autoimmune background. Nevertheless, they have a good prognosis with the applied treatment. The most common manifestation of hyperkinetic movement disorders was ataxia, followed by myoclonus and various types of tremors. When caring for people suffering from COVID-19, the possibility of such complications should be taken into account. Further observations are needed to determine their frequency and the most effective methods of treatment.

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