REVIEW ARTICLE

Neurologia i Neurochirurgia Polska
Polish Journal of Neurology and Neurosurgery
2023, Volume 57, no. 1, pages: 63–76
DOI: 10.5603/PJNNS.a2023.0012
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ISSN: 0028-3843, e-ISSN: 1897-4260

LEADING TOPIC

Leading Topic Editor: Alina Kułakowska, MD, PhD, Department of Neurology, Medical University of Bialystok, Bialystok, Poland

Hyperkinetic movement disorders following SARS-CoV-2 infection and vaccination — an update

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ABSTRACT

The aim of this review was to summarise current knowledge regarding hyperkinetic movement disorders related to SARS-CoV-2 infection and vaccination in terms of phenomenology, epidemiology, pathogenesis and treatment. After a thorough review of the PubMed and Google Scholar databases (2020–2022), we identified myoclonus and ataxia sometimes accompanied by opsoclonus (AMS) as the two most frequent COVID-19 sequelae, with chorea, tremor and dystonia being very rare. The pathogenesis seems to be variable, but in the majority of AMS cases it was autoimmunological, with good response and recovery after corticosteroids or intravenous immunoglobulins infusions. Vaccination may be complicated by hyperkinetic movement disorders (e.g. tremor, dystonia), but this is very rare. Patients with Deep Brain Simulation depletion should not be postponed due to lockdowns as this may result in fatal outcomes.

Key words: ataxia, myoclonus, opsoclonus, chorea, tremor, dystonia, COVID-19, SARS-CoV-2

(*Neurol Neurochir Pol 2023; 57 (1): 63–76*)

Introduction

Post-COVID conditions are being reported worldwide under a broad range of names, including long COVID, post-acute COVID-19, long-term effects of COVID, post-acute COVID syndrome, chronic COVID, long-haul COVID, late sequelae and others, as well as the research term 'post-acute sequelae of SARS-COV-2 infection' (PASC). They may be a result of the persistance of acute phase symptoms or newly developed or recurrent symptoms after recovery. According to the WHO (World Health Organisation), post COVID-19 syndrome refers to cases with a history of probable or confirmed SARS-CoV-2 infection, usually at least three months after onset, with symptoms that last for at least two months and cannot be explained by an alternative diagnosis [1]. The most frequent complications of SARS-CoV-2 infection that may fulfill the definitions of PASC or Long-COVID include: fatigue (58%), headache (44%), attention disorder (27%), hair loss (25%), and dyspnoea (24%). Among the neurological complications, the most common are cognitive decline, sleep problems, myalgia, and anosmia/dysgeusia) [2].

Several possible pathogenetic mechanisms of neurological symptoms associated with COVID-19 (direct viral invasion, hypoxic injury, coagulopathy, inflammatory response and immune dysfunction, side effects of medications, psychological — resulting in functional symptoms after recovery from severe life-threatening condition) have been recently described [3]. Myoclonus and tremor — the predominant movement disturbances associated with COVID-19 — are non-specific symptoms that often occur in critically ill patients as a result of hypoxia or toxic-metabolic disturbances [4]. In patients severely affected by COVID-19, with multiorgan failure, requiring mechanical ventilation and treatment in the Intensive Care Unit (ICU), neurological symptoms are most likely related to hypoxia and a toxic-metabolic cause or causative medications.

On the other hand, in patients with an asymptomatic or mild infection, with no history of severe hypoxia or multiorgan failure, the cause of movement disorders accompanying

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Received: 17.01.2023 Accepted: 10.02.2023 Early publication date: 17.02.2023

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COVID-19 is most likely to be autoimmune. As in other infectious diseases, SARS-CoV-2 can result in autoimmunological reactions, probably due to the mimicry mechanism. Hyperkinetic movement disorders besides autoimmune neuropathies (e.g. Guillain-Barre syndrome) may represent the group of such conditions. Fortunately, they are not as frequent as other PASCs. As they respond well to immune therapies, all acute/subacute movement disorders following a SARS-CoV-2 infection have to be considered as being of autoimmune origin and properly treated. They usually occur a few weeks after recovery, and if treated may resolve in under two months. In this way, they do not fulfill the diagnosis of PASC.

The most frequently reported hyperkinetic movement disorder related to COVID-19 is myoclonus, often occurring in syndromes including ataxia and opsoclonus. Only a few cases of chorea, tremor and dystonia have been reported [5]. A systematic review of hyperkinetic syndromes following SARS-CoV-2 infection was presented in this journal in 2021 [6]. For this latest analysis, we reviewed the PubMed and Google Scholar databases for the period 2020–2022, searching for papers using the key words: 'ataxia', 'myoclonus', 'opsoclonus', 'chorea', 'dystonia', 'tremor', 'SARS-CoV-2', 'COVID-19', and 'vaccination' to update the literature. Key results are set out in Tables 1–3.

Ataxia/myoclonus/opsoclonus syndrome

To date, there have been 50 publications of case reports and case series of patients with myoclonus or ataxia or opsoclonus (or a combination of these) related to COVID-19 [7–56]. A total of 105 patients had been described in the medical literature up to the end of 2022. Forty-three of these 105 cases had a severe SARS-CoV-2 infection and required hospitalization in the ICU [48–56]. As the movement disorder could be a possible complication (hypoxia, medications) they were not included into analysis. Sixty two out of 105 cases developed movement disorders despite mild or moderate severity of infection — as these were possibly of autoimmune origin, these reports were included into detailed analysis.

Sixty two patients — 55 men and seven women aged 32–88 years — developed myoclonus or opsoclonus or ataxia after SARS-CoV-2 infection. The most frequent was myoclonus (57/62 patients), followed by ataxia (46/62), and opsoclonus (17/62). In two patients, myoclonus presented as an independent symptom. Thirty one subjects had ataxia-myoclonus syndrome (AMS), 14/62 had opsoclonus-myoclonus-ataxia syndrome (OMAS), and 3/62 patients had opsoclonus-myoclonus syndrome (OMS). The myoclonus type affecting COVID-19 patients was a positive one, but also action-sensitive and stimulus-sensitive in some cases. Symptoms were generalised, in some patients affecting their voice and gait. Seventeen patients presented additional signs such as dysarthria, dysphagia, and/or nystagmus as a manifestation of cerebellar syndrome. Thirteen patients presented neuropsychiatric symptoms that accompanied movement disturbances: two mild somnolence, one sopor, four confusion, four cognitive impairment, one psychomotor agitation with frontal lobe syndrome, and one psychomotor retardation.

The delay between the onset of COVID-19 infection and the onset of neurological symptoms was 2–42 days (mean 13). In the majority of cases, 43/62 patients, SARS-CoV-2 infection was mild or asymptomatic, usually with fever, cough, myalgia, general weakness and not requiring oxygen therapy. Fifteen out of 62 had a moderate course of infection and required non-invasive oxygen therapy. In four subjects, there was no precise data as to the severity of the COVID-19.

The neuroimaging studies (computed tomography — CT, magnetic resonance imaging —MRI), neurophysiological (electroencephalography, electroneurography, electromyography) did not show abnormalities in all reported cases. General examination of cerebrospinal fluid (CSF) was normal in 46 patients, isolated protein elevation was observed in six patients, isolated lymphocytic pleocytosis in one, and lymphocytic pleocytosis with elevated protein in one. Lumbar puncture was not performed in eight cases.SARS-CoV-2 RT-PCR in the CSF was negative in all tested cases. In three reports, autoantibodies were identified: against glial fibrillary acidic protein (GFAP) [16], leucine-rich glioma-inactivated 1 (LGI-1) [29], and glutamic acid decarboxylase (anti-GAD) [22]. Another study described a patient with post-COVID-19 ataxia-myoclonus syndrome in whom autoantibodies directed against Purkinje cells of the cerebellum, striatal and hippocampal neurons were identified by nerve tissue immunostaining with serum and CSF [13]. Franke et al. analysed the cerebrospinal fluid for antineuronal and antiglial antibodies among the patients with neurological symptoms after SARS-CoV-2 infection: anti-neuronal antibodies were present (IgG anti cerebellum granule cells in three cases, anti nucleus and proximal dendrites of Purkinje cells in two cases, myelinated fibres in the cerebellum in one case, neurophil of the olfactory bulb in one case, blood vessels in the brain in three cases, neurophil in the hippocampus in one case, and glia limitans and astrocytes in two cases) [56]. These studies may indicate molecular mimicry between autoantigens and SARS-CoV-2 as possible pathogenesis in such cases. A possible autoimmune cause is also indicated by FDG-PET studies in five cases of COVID-19-associated ataxia with myoclonus and/or encephalopathy which showed cortical hypometabolism and cerebellar and/or putaminal hypermetabolism [8, 13]. Cerebellar hypermetabolism in FDG-PET correlated with autoantibody target (against Purkinje cells and striatum) and clinical symptoms in a case reported by Grimaldi [13]. A combination of cortical hypometabolism and cerebellar hypermetabolism is unclear, and has been rarely reported due to infectious encephalitis [57] or in paraneoplastic cerebellar degeneration [58]. However, it may be the consequence of increased energy uptake due to the inflammatory process associated with an immune reaction in the cerebellum and cortical neurons damage or receptors/ion channels blockade due to parainfectious mechanisms [8].

Table 1. Characteristics of patients with myoclonus/opsoclonus/ataxia associated with COVID-19

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Treatment/outcome
Rabano-Suarez et al. 2020 [7]	63/M	Mild	• Myoclonus • Dysarthria • Dysphagia • Mild somnolence	9 days	Propofol, Valproic acid, Levetiracetam, Clonazepam, Methylprednisolone 1 g/day, 5 g total plasmapheresis 5 cycles, Significant improvement
	88/F	Mild	• Myoclonus	21 days	Methylprednisolone 250 mg/day for 3 days, Complete recovery
	76/M	Mild	• Myoclonus	11 days	Clonazepam, Levetiracetam (no effect), Methylprednisolone 250 mg/day for 3 days, Recovery after 3 weeks
Delorme et al. 2020 [8]	72/M	Moderate	• Myoclonus • Ataxia • Psyhomotor agitation • Frontal lobe syndrome	12 days	IVIG 2 g/kg, Significant improvement, Complete recovery after 6 weeks
Wright et al. 2020 [9]	79/M	Mild	• Opsoclonus • Ataxia • Cognitive impairment	13 days	No data/improvement of cognitive function and resolution of eye movement abnormality after 19 days, death after 27 days due to general physical decline
Dijkstra et al. 2020 [10]	44/M	Mild	• Myoclonus • Ataxia • Voice tremor • Transient ocular flutter (no evident opsoclonus) • Cognitive impairment	1 week	Methylprednisolone 1g daily for 5 days, IVIG 0,4 g/kg for 3 days/partial recovery after 15 days, full recovery within 2 months
Schellekens et at. 2020 [11]	48/M	Mild	• Myoclonus • Ataxia • Saccadic intrusions of eye movement (no opsoclonus)	13 days	Levetiracetam, Partial recovery within 2 months
Shah et al. 2020 [12]	Middle aged/M	No data	• Myoclonus • Opsoclonus • Ataxia • Ataxic dysarthria	3 weeks	Methylprednisolone 1 g/day, Sodium valproate 20 mg/kg/day, Clonazepam 2 mg/day, Levetiracetam 2 g/day, Recovery within 1 week
Grimaldi et al. 2020 [13]	72/M	Moderate	• Myoclonus • Ataxia • Ataxic dysarthria • Action tremor	17 days	IVIG 0.4 g/kg/day for 5 days, Methylprednisolone 1 g/day for 5 days/ Recovery within 2 weeks
Salari et al. 2021[14]	42/M	Mild	• Myoclonus • Ataxia • Dysarthria • Cognitive deficits	Unknown	Methylprednisolone 1 g/day, 5 g total, Complete recovery after 1 month
	52/M	Mild	• Myoclonus • Ataxia	7 days	Methylprednisolone 1 g/day, 5 g total, Significant improvement after 1 month
	38/M	No data	• Myoclonus • Ataxia	8 days	Clonazepam 2 mg/day, Levetiracetam 1,000 mg/day, Methylprednisolone 1 g/day, 5 g total, Significant improvement after 2 weeks
Chan et al. 2021 [15]	44/M	Mild	• Myoclonus • Ataxia • Voice tremor • Mild rigidity in upper extremities	12 days	Methylprednisolone 1 g/day, 5 g total, Clonazepam 0.75 mg 2 × 1, Levetiracetam 1,000 mg 2 × 1, Significant improvement after 18 days
Asan et al. 2021 [16]	44/M	Mild	• Myoclonus • Ataxia	14 days	Methylprednisolone 1 g/day, 5 g total, Significant improvement after few days, Total recovery after 3 months

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Table 1. cont. Characteristics of patients with myoclonus/opsoclonus/ataxia associated with COVID-19

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Treatment/outcome
Saha et al. 2021 [17]	78/F	Mild	• Myoclonus • Opsoclonus • Ataxia	14 days	IVIG 0.4 g/kg for 5 days, Methylprednisolone 1 g/day, Levetiracetam 1 g/day, Significant improvement after 7 days
Ishaq et al. 2021 [18]	63/M	Moderate	• Myoclonus • Opsoclonus • Ataxia • Confusion	23 days	Methylprednisolone 1 g/day, 5 g total followed by IVIG 0.4 g/kg for 5 days, Complete recovery after 4 weeks
Ram et al. 2021 [19]	79/M	Mild	• Myoclonus • Ataxia	10 days	Intravenous dexamethasone, 6 mg/day, Levetiracetam 2,000 mg/day, Complete recovery after 2 months
Otmani et al. 2021 [20]	59/M	Asymptomatic	• Myoclonus • Nystagmus	Unknown	Levetiracetam, IVIG, Methylprednisolone 1 g/day, 5 g total, Complete recovery after 21 days
Werner et al. 2021 [21]	62/M	Mild	• Ataxia • Dysarthria	16 days	Methylprednisolone 500 mg/day, 2.5 g total, Acyclovir, Significant improvement after 6 days
Emekli et al. 2021 [22]	54/M	Mild	• Ataxia • Dysarthria • Tremor • Confusion • Convergence spasm in ophtalmological examination	21 days	Methylprednisolone 1 g/day, 10 g total followed by oral steroids, IVIG 0.4 g/kg for 5 days, propranolol, Significant improvement after 1 month
Przytuła et al. 2021 [23]	49/M	Mild/ asymptomatic	• Myoclonus • Ataxia • Voice tremor	11 days	Methyloprednisolone 1 g/day, 5 g total followed by oral Prednisone 60 mg/day for 2 weeks, Significant improvement after 3 weeks
	62/M	Mild	• Myoclonus • Opsoclonus • Ataxia	11 days	IVIG 0.4 g/kg for 5 days, Methylprednisolone 1 g/day, 5 g total followed by oral prednisone (1 mg/kg/day) for 2 weeks with gradual dose reduction, Significant improvement after 2 weeks
Shetty et al. 2021 [24]	41/M	Mild	• Myoclonus • Gait ataxia	10 days	Clonazepam, Levetiracetam, Methylprednisolone 1 g/day, 5 g total, Significant improvement after 6 weeks
Giannantoni et al. 2021 [25]	67/M	Moderate	• Myoclonus • Ataxia • Ocular saccadic movements • Voice tremor • Generalised tremor	11 days	Methylprednisolone 1 g/day, 5 g total followed by oral prednisone (1 mg/kg/day) for 2 weeks with gradual dose reduction, Clonazepam, Levetiracetam, Improvement after 14 days
Sundar et al. 2021 [26]	60/M	Mild	• Myoclonus • Ataxia	14 days	Intravenous and oral steroids, Levetiracetam, Clonazepam, Complete recovery after 2 weeks
	53/M	Mild	• Myoclonus • Ataxia	7 days	Clonazepam, Methylprednisolone, followed by oral steroids, Complete recovery after 10 days
	47/M	Moderate	• Myoclonus • Opsoclonus • Ataxia • Voice tremor • Postural tremor of upper limbs • Generalised seizures	10 days	Methylprednisolone for 5 days, followed by oral steroids for a week, Clonazepam, Partial recovery

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Table 1. cont. Characteristics of patients with myoclonus/opsoclonus/ataxia associated with COVID-19

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Treatment/outcome
Emamikhah et al. 2021 [27]	51/M	Mild	• Myoclonus • Opsoclonus • Ataxia • Voice tremor	2 weeks	Clonazepam 0.5 mg 4 × 1, Levetiracetam 500 mg 2 × 1, IVIG 2 g/kg, total dose 150 g, Complete recovery after 4 weeks
	54/M	Moderate	• Myoclonus • Ataxia	4 days	Levetiracetam 2,000 mg/day, Sodium valproate 1,000 mg/day IVIG in total dose 100 g, Partial recovery after one week
	52/M	Moderate	• Myoclonus • Ataxia • Voice tremor	16 days	Sodium valproate 1,000 mg/day, Clonazepam 1 mg 4 × 1, Partial recovery after 2 months
	42/F	Mild	• Myoclonus • Ataxia • Voice tremor	10 days	Sodium valproate, Clonazepam/ no data
	44/M	Mild	• Myoclonus • Opsoclonus • Ataxia • Voice tremor	3 days	Sodium valproate, Clonazepam, IVIG, Complete recovery after 2 months
	52/M	Mild	• Myoclonus • Ataxia • Voice tremor	3 weeks	Clonazepam, IVIG in total dose of 100 g, Significant improvement after 4 weeks
	39/M	Moderate	• Generalized seizures • Myoclonus • Opsoclonus • Ataxia • Voice tremor	10 days	Levetiracetam, Sodium valproate, Clonazepam, IVIG, Dexamethasone/ no data
Foucard et al. 2021 [28]	83/M	No data	• Myoclonus • Opsoclonus • Ataxic dysarthria • Confusion	10 days	IVIG 0.4 g/kg for 5 days, Steroids 1 g/day for 5 days, Diazepam/ significant improvement after 1 week
	63/M	No data	• Myoclonus • Ataxia • Ataxic dysarthria	6 weeks	IVIG 0.4 g/kg for 5 days/significant improvement after 1 week
Smyth et al. 2021 [29]	50/M	Moderate	• Myoclonus • Opsoclonus • Confusion	7 days	Levetiracetam 1,500 mg/day, Clonazepam 1 mg/day, Methylprednisolone 1 g/day for 3 days followed by oral prednisolone, Improvement after 11 days, recurrence of symptoms, complete recovery after Methylprednisolone 1 g/day for 3 days followed by oral prednisolone
Guerra et al. 2021 [30]	50/M	Asymptomatic	• Myoclonus • Ataxia	10 days	Methylprednisolone 250 mg/d for 3 days followed by oral steroids, Complete recovery after 1 week
	80/M	Moderate	• Myoclonus • Ataxia • Dysarthria	8 days	Methylprednisolone 120 mg/d for 5 days, Mild improvement after 1 week
Urrea-Mendoza et al. 2021 [31]	32/M	Moderate	• Myoclonus • Opsoclonus • Ataxia	12 days	Clonazepam 3 mg/day, Valproic acid 3,000 mg/day, Oral methylprednisolone 40 mg/day, Significant improvement after 24 days

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Table 1. cont. Characteristics of patients with myoclonus/opsoclonus/ataxia associated with COVID-19

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Treatment/outcome
Sanguinetti et al. 2021 [32]	57/M	Mild	• Myoclonus • Opsoclonus	10 days	Clonazepam, IVIG 0.4 g/kg for 5 days, Intravenous, Methylprednisolone 80 mg/day Significant improvement
Chacko et al. 2021 [33]	53/F	Moderate	• Myoclonus • Opsoclonus • Sopor	14 days	Methylprednisolone followed by oral steroids, Rituximab, Plasmapheresis, Levetiracetam, Clobazam, Complete recovery after 32 days
Fernandes et al. 2021 [34]	58/F	Mild	• Myoclonus • Opsoclonus • Ataxia	14 days	Clonazepam, Levetiracetam, IVIG, Corticosteroids, No data on outcome
Vinod et al. 2021 [35]	52/M	Mild	• Myoclonus • Ataxia • Bilateral nystagmus • Generalised seizure	10 days	Midazolam, Levetiracetam, Clonazepam, Methylprednisolone 1 g/day, 5 g total, IVIG for 5 days, Significant improvement after 18 days
Tanu et al. 2021 [36]	52/M	Mild	• Myoclonus • Ataxia • Dysarthria • Bilateral horizontal gaze evoked nystagmus	8 days	Antibiotics, Anticoagulants, Dexamethasone 8 mg/day i.v., Valproic acid 600 mg/day, Clonazepam 1 mg/day, Significant improvement after 1 week
Talaei et al. 2021 [37]	53/F	No data	• Myoclonus • Opsoclonus • Axial rigidity	14 days	Myoclonic storm requiring intubation and ventilation, Steroids, Plasmapheresis, Significant improvement after 1 week
Serrazina et al. 2022 [38]	75/M	Moderate	• Myoclonus • Ataxia • fragmentation of ocular movements	9 days	Valproic acid 1,000 mg/day, Clonazepam 2 mg/day, Methylprednisolone for 5 days, followed by IVIG and then oral steroids, Partial recovery
Vijayaraghavan et al. 2022 [39]	64/W	Moderate	• Generalised seizure • Myoclonus • Opsoclonus • Ataxia	18 days	Methyloprednisolone 1g/day, 5 g total, Worsening of symptoms after a few days, IVIG 0.4 g/kg daily for 5 days, Rituximab 1 g IV, Complete recovery after 2 weeks
Rodriguez-Quiroga et al. 2022 [40]	60/M	Mild	• Myoclonus • Ataxia • Ocular flutter	10 days	IVIG 0.4 g/kg daily for 5 days, Methylprednisolone 1 g/day for 3 days, Levetiracetam, Full recovery in 18 days
	67/M	Mild	• Myoclonus • Ataxia • Dysarthria	11 days	Methylprednisolone 1 g/day for 3 days, Full recovery in 13 days
	36/M	Mild	• Myoclonus • Ataxia	15 days	Methylprednisolone 1 g/day for 3 days, Levetiracetam, Full recovery in 4 weeks
	44/M	Mild	• Myoclonus • Ataxia • Opsoclonus	13 days	Methylprednisolone 1 g/day for 3 days, Full recovery in 10 days
	33/M	Mild	• Myoclonus • Ataxia • Ocular flutter	8 days	Levetiracetam, Clonazepam, Full recovery in 6 weeks

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Table 1. cont. Characteristics of patients with myoclonus/opsoclonus/ataxia associated with COVID-19

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Treatment/outcome
Mohamed et al. 2022 [41]	60/M	Asymptomatic	• Myoclonus • Dysarthria • Down-gaze paralysis	Unknown	Dexamethasone 12 mg/day for 3 days, Clonazepam, Levetiracetam, Progressive recovery
Kato et al. 2022 [42]	55/F	Mild	• Myoclonus • Gait disturbances • Mild somnolence	10 days	Methylprednisolone, Complete recovery after 3 days
Godani et al. 2022 [43]	71/M	Mild	• Myoclonus • Opsoclonus • Ataxia	2 days after COVID-19 resolution	Clonazepam for 1 month with no effect, IVIG 0.4 g/kg daily for 5 days — significant improvement after 17 days
Grazzini et al. 2022 [44]	70/M	Mild	• Myoclonus • Ataxia • Voice tremor, dysarthria • Tremor • Cognitive impairment	15 days	Clonazepam, Valproic acid, IVIG 0.4 g/kg for 5 days, Complete recovery after 2 months
	63/M	Mild	• Myoclonus • Ataxia • Tremor	2 days	Diazepam, Levetiracetam, IVIG 0.4 g/kg for 5 days, Worsening of symptoms 5 days after IVIG treatment start, Significant improvement after 25 days
	56/M	Mild	• Dysmetria • Ataxia • Psychomotor retardation	15 days	Plasmapheresis, Rapid clinical improvement
Chattopadhyay et al. 2022 [45]	70/M	Mild	• Ataxia • Dysarthria • Tremor	35 days	Methylprednisolone 1 g/day for 5 days, Significant improvement after 10 days
Osawa et al. 2022 [46]	52/M	Moderate	• Myoclonus • Ataxia	16 days	Methylprednisolone 1 g/day for 3 days, Benzothiamine, Cyanocobalamin, Pyridoxine improvement after 3 days, Complete recovery after 3 months
Przytuła et al. 2022 [47]	45/M	Mild	• Myoclonus • Ataxia • Voice tremor • Four-limbs tremor	12 days	Methyloprednisolone 1 g/day for 5 days followed by oral prednisone 70 mg/day for 4 weeks with gradual dose reduction, Significant improvement after 4 weeks

Cerebellar hyperperfusion in brain single photon emission computed tomography (SPECT) has been observed in patients with OMS [59]. A patient with post COVID-19 AMS reported by Osawa et al. revealed hyperperfusion in the cerebellum and hypoperfusion in the cerebral cortex, with frontal lobe predominance [46].

Patients have been treated symptomatically with antiepileptic medications (valproic acid 11/60 patients, levetiracetam 24/62, clonazepam 27/62, diazepam 2/62, midazolam 1/62, and clobazam 1/62) and immunotherapy including steroids (methylprednisolone 40/62 patients, dexamethasone 4/62), intravenous immunoglobulins (IVIG) 25/62 patients, of which steroids combined with IVIG were administered in 15/25, immunomodulatory medications (rituximab in 1/62 patient), and plasmapheresis in 4/62 patients. In subjects treated only by symptomatic medications (benzodiazepines, antiepileptic drugs) only a partial improvement was observed in follow-up [11, 27]. In one patient, benzodiazepines and antiepileptics were used for two months without improvement, only after the use of steroids and IVIG symptoms partially subsided [38]. In the majority of cases, symptomatic treatment has been combined with immunotherapy, and in these cases a significant improvement was observed between three days after the steroids administration [42, 46] and three months after the onset of treatment. There have also been reports of two patients who did not improve with steroids, but were then administered second-line IVIG or IVIG with rituximab with recovery [18, 39]. Treatment with corticosteroids seems to be

Table 2. Characteristics of patients with chorea associated with COVID-19 and SARS-CoV-2 vaccination

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Neuroimaging	Treatment/outcome
Ashrafi et al. 2022 [60]	67/F	Moderate – with post-COVID encephalitis after 2 weeks	• Chorea on face and four limbs, with right arm dominance	3 months	MRI: bilateral hyperintensity on basal ganglia	Tetrabenazine 2 × 12.5 mg, Rapid recovery after a few days
	62/F	No data	• Four-limbs chorea with right-side dominance	15 days	MRI: unremarkable	Tetrabenazine, Significant improvement
Barberà et al. 2022 [61]	69/F	No data	• Four-limbs chorea • Right-side hemiparesis • Mixed aphasia	21 days	CT: cerebral venous sinus thrombosis, capsuloganglionic and thalamic infarcts with haemorrhagic transformation of left thalamic infarct	Anticoagulants, death
Sawczyńska et al. 2022 [62]	77/F	Moderate	• Four-limbs and orofacial chorea with left-side dominance • Confusional state	At one time with infection, 2 weeks after COVID-19 vaccination	MRI: diffuse white matter hyperintensities, cortical and subcortical atrophy	Methylprednisolone 1 g/day for five days followed by IVIG 2 g/kg over course of five days Diazepam, Remdesivir, Complete recovery in a few days
Hassan et al. 2021 [63]	58/M	Moderate	• Four-limbs chorea	4 days	MRI: chronic white matter hyperintensities	Ceftriaxone, Acyclovir, Dexamethasone, Risperidone, Omeprazole, Improvement after a few days
Cotta Ramusino et al. 2021 [64]	62/M	Mild	• Four-limbs, head and trunk chorea • Confusional state	At one time with infection	MRI - hypointense signal in dorsolateral portion of both putamina (SWI)	Haloperidol 4.5 mg/day, Tetrabenazine 50 mg/day, Dexamethasone, Significant improvement after a few days
Ghosh et al. 2021 [65]	60/M	Moderate	• Right limbs chorea	2 days	MRI: left striatal hyperintensity, Laboratory tests: acute hyperglycaemia	Intravenous fluids, Insulin recovery
Byrnes et al. 2020 [66]	36/M	Moderate	• Four-limbs chorea	At one time with infection	MRI: multifocal lesions affecting bilateral medial putamen, left cerebellum, hippocampus, punctate restricted diffusion in right basal ganglia	Midazolam, Diazepam, Methylprednisolone 500 mg/d for 5 days followed by IVIG 2 g/kg for 5 days and oral Prednisone, Improvement after a few days
Salari et al. [67]	13/M	N/A	• Right-sided hemichorea	7 days after 1st dose of COVID-19 vaccination (BBIBP-CorV (Sinopharm)	MRI: multiple white matter lesions, one of them enhanced with gadolinium	Methylprednisolone 1 g/day for 3 days followed by oral Prednisolone (50 mg/d), Tetrabenazine (25 mg/d), moderate improvement after 2 weeks, Significant improvement after 1 month
	18/M	N/A	• Left-sided hemichorea	7 days after COVID-19 vaccination (BBIBP-CorV (Sinopharm)	MRI: unremarkable	Methylprednisolone 1 g/day for 3 days followed by oral Prednisolone (50 mg/d), Tetrabenazine (25 mg/d), moderate improvement after 2 weeks

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Table 2. cont. Characteristics of patients with chorea associated with COVID-19 and SARS-CoV-2 vaccination

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Neuroimaging	Treatment/outcome
Matar et al. 2021 [68]	88/M	N/A	• Left-sided hemichorea • Mild right-sided parkinsonian syndrome	16 days after 1st dose COVID-19 vaccination (Astra Zeneca, AZD1222)	MRI: Chronic small vessel ischaemic changes	Methylprednisolone 1 g/day for 3 days, Significant improvement after 1 day
	84/M	N/A	• Left-sided hemichorea	40 days after 1st dose COVID-19 vaccination (Astra Zeneca, AZD1222)	MRI: Chronic small vessel ischaemic changes	Methylprednisolone 1 g/day for 3 days, Significant improvement after 3 days
Ryu et al. 2021 [69]	83/M	N/A	• Right-sided hemichorea	1 day after 2nd dose of COVID-19 vaccination, 21 days after 1st dose (Pfizer--BioNTech)	MRI: unremarkable Brain SPECT — perfusion pattern asymmetrically decreased in left thalamus	Haloperidol 0.75 mg 2 × 1, Significant improvement after 2 weeks
Batot et al. 2022 [70]	90/M	N/A	• Left hemichorea--hemiballismus • Dysarthria	Hours after 2nd dose of COVID-19 vaccination, 21 days after 1st dose (Pfizer--BioNTech)	MRI: unremarkable Brain FDG-PET — increased metabolism of right putamen (compared to left side)	Tetrabenazine, with a gradual increase (up to 75 mg daily) for 3 weeks, followed by Olanzapine (up to 7.5 mg daily) for 1 week, No effect Methylprednisolone 1 g/day for 5 days, Significant improvement
Shahali et al. 2022 [71]	72/M	N/A	• Right-sided hemichorea	9 days after 1st dose of COVID-19 vaccination (Astra Zeneca ChAdOx1 nCoV-19)	MRI: left thalamus acute ischaemic infarct	Nadroparin 90 U/kg/day, Sodium ozagrel 160 mg/day, Edaravone 60 mg/day, Methylprednisolone 1 g/kg, Haloperidol 0.75 mg/day, Warfarin 0.125 mg/kg/day, Improvement during 2 weeks

equally effective, but significantly cheaper and more readily available than IVIG therapy. Therefore, we suggest starting with steroids and only in a case of non-responsiveness or contraindications following this treatment with IVIG [47].

Chorea

A total of eight patients (four women and four men, aged 36–77 years, mean 61) with COVID-19 [60–66] and an additional seven patients as an adverse event after vaccination against SARS-CoV-2 [67–71] (six men and one woman aged 13–90) have been reported to develop chorea (Tab. 2). Five patients had moderate, one patient had mild, and in two cases the severity of the COVID-19 was not reported. The delay between infection and the appearance of chorea was between less than 24 hours and 90 days after SARS-CoV-2 identification, mean 16.5 days. In post-vaccination cases, symptoms occured between seven and 40 days, mean 17 days after the first dose of COVID-19 vaccine and in two cases on the first day after the second dose of vaccine. Four post-COVID-19 and six post-vaccination patients had unilateral chorea. Four subjects had generalised symptoms, two presented with impaired consciousness, and one with mixed aphasia and right-side hemiparesis — this patient was diagnosed with cerebral venous sinus thrombosis, capsuloganglionic and thalamic infarcts with haemorrhagic transformation of the left thalamic infarct.

In diagnostic tests among post-COVID-19 infection cases, head MRI was normal in three subjects, four had pathological lesions in basal ganglia — one had hypointense susceptibility weighted imaging (SWI) signal in the dorsolateral portion of both putamina, one had left striatal hyperintensity on T1-weighted imaging, one had multifocal lesions affecting the bilateral medial putamen, left cerebellum, hippocampus, punctate restricted diffusion in the right basal ganglia, and one had bilateral hyperintensity on basal ganglia. One subject had bilateral thalamic infarcts with haemorrhagic transformation of the left thalamic infarct (CT). Moreover,

Table 3. Characteristics of patients with tremor associated with COVID-19

Article	Age/ /gender	COVID-19 severity of infectious stage	Neurological symptoms	Time between acute infection and onset of neurological symptoms	Diagnostic tests	Treatment/ /outcome
Henry et al. 2020 [72]	77/M	Moderate	• Essential tremor worsening • Bilateral dysmetria	No data	• Head CT: normal	Azithromycin, Ceftriaxone, No data of outcome
Klein et al. 2020 [73]	46/M	Mild	• Four-limbs postural tremor • Wide based gait	8 days	• MRI: chronic ischaemic lesions	Propranolol, Mild improvement
Diezma-Martin et al. 2020 [74]	70/M	Mild	• Four-limbs tremor • Orthostatic tremor • Voice tremor • Wide based gait	17 days	• MRI, CSF, laboratory tests: normal	Clonazepam, Mild improvement
Passaretti et al. 2021 [75]	60/M	Asymptomatic	• Essential tremor worsening	21 days	• MRI, laboratory tests: normal	Propranolol, No improvement
Pistola et al. 2021 [76]	46/M	Mild	• Intentional tremor • Dysmetria • Gait disturbances	10 days	• Head MRI: mild leptomeningeal enhancement • Oligoclonal bands detected in CSF • EEG: normal	Oral steroids, Antibiotics, Heparin, Significant improvement after 1 month

hippocampus, cerebellum and pons lesions were noted. Head MRI of post-vaccination patients was unremarkable in six cases, with multifocal white matter lesions, of which one was enhanced with gadolinium in one patient. One patient has been diagnosed with acute left thalamic infarct due to vaccine-induced prothrombotic immune thrombocytopenia [71]. Brain SPECT (single-photon emission computed tomography) was performed in one post-vaccination subject and revealed left thalamic hypoperfusion compared to the right side. Brain FDG-PET (fluoro-deoxy-glucose positon emission tomography) performed in a patient reported by Batot et al. showed increased metabolism of the right putamen (compared to the left side). Three weeks later, after steroid treatment, a repeated brain FDG-PET showed resolution of the right putamen and increased metabolism [70]. Among all cases, lumbar puncture was performed in seven patients revealing lymphocytic pleocytosis with elevated protein in one case, isolated lymphocytic pleocytosis in one case, isolated protein elevation in two cases, and normal CSF in three cases. 1/3 CSF tested positive for SARS-CoV-2, while other microbiological analyses of CSF (bacterial and viral) were negative. Various causes of chorea have been considered in the differential diagnosis of the reported cases. The previously mentioned subject developed hyperkinetic motor symptoms as a complication of cerebral sinus thrombosis [61]. A previously healthy non-diabetic patient reported by Ghosh et al. developed hyperglycaemia, ketonuria and metabolic acidosis, and secondary right hemichorea with striatal lesions in head MRI [65]. Diabetic striatopathy secondary to COVID-19 infection has been diagnosed. A polysubstance abusing homeless patient reported by Byrnes et al. developed four-limbs chorea during SARS-CoV-2 infection. His urine test was positive for cocaine, opiates, and benzodiazepines. Head MRI showed multifocal lesions affecting the bilateral medial putamen, left cerebellum, hippocampus and one focal lesion restricted diffusion in the right basal ganglia. Improvement in the subject's choreiform movements was only observed after immunotherapy using methylprednisolone and IVIG [66]. Other causes of choreiform movements in reported cases, including endocrinological and autoimmune systemic diseases, paraneoplastic and prion disease, were excluded.

10/15 patients were treated with steroids (8/15 i.v. methylprednisolone, 2/15 dexamethasone) and 2/15 patients received a combination of steroids and IVIG. Immunotherapy alone was administered in 2/15 cases, resulting in significant improvement of symptoms after 3–14 days. Steroids or IVIG combined with symptomatic treatment (tetrabenazine 4/15, risperidone 1/15, haloperidol 2/15, diazepam 2/15, midazolam 1/15) resulted in significant improvement of symptoms within a few days to one month after treatment initiation. Causal treatment was conducted in a patient with diabetic striatopathy secondary to COVID-19 using intravenous fluids and insulin, obtaining recovery of neurological symptoms. A patient diagnosed with cerebral venous sinus thrombosis recieved anticoagulant treatment complicated by haemorrhagic transformation of the left thalamic infarct, resulting in patient death. Additional

antibiotics or antivirals were administered in 3/15 patients (remdesivir, acyclovir, ceftriaxone). Symptomatic treatment alone was used in 3/15 patients, using tetrabenasine in two cases and haloperidol in the third case, obtaining significant improvement in choreiform movements within 14 days.

Tremor

Tremor as a non-specific movement complication after COVID-19 has not been frequently reported. A total of five patients (five men, aged 46–77 years, mean 60) have been reported with tremor as the dominant sign due to a mild/moderate course of COVID-19 [72–76] (Tab. 3). Apart from these reports, tremor coexisted in 17 patients with ataxia-myoclonus syndrome (AMS), opsoclonus-myoclonus-ataxia syndrome (OMAS), and opsoclonus-myoclonus syndrome (OMS) — limb tremor four [13, 22, 44, 45], voice tremor in nine [10, 15, 23, 27], and limb tremor combined with voice tremor in four [25, 26, 44, 47] (Tab. 1). In another two cases, functional tremor was diagnosed [77, 78] and one case featured tremor as an adverse reaction to remdesivir prescribed to treat the SARS-CoV-2 infection [79]. In addition, tremor has been associated with critically ill patients with multiple organ failure due to COVID-19 requiring invasive ventilation and ICU treatment [80, 81].

In addition to the aforementioned case reports and case series specifically focused on tremor as the main symptom, several observational studies have been done to assess the incidence of neurological symptoms after SARS-CoV-2 infection. Pilotto et al. conducted an observational study on 165 patients hospitalised due to COVID-19; after a six-month follow-up, tremor as a motor complication had occurred in 15/165 (9%) patients [82]. In another observational study of 2,750 convalescents, neurological complications were noted in 71, of whom only one developed tremor [83].

Among patients with dominant tremor as a complication of COVID-19 (Tab. 3), one had asymptomatic, three mild, and one moderate COVID-19. The time elapsed between infection and the appearance of tremor was 8–21 days (mean 14) after SARS-CoV-2 onset. Two patients were diagnosed with essential tremor before the pandemic, and SARS-CoV-2 infection caused rapid tremor worsening [72, 75]. All five patients had intentional and postural limbs tremor, and symptoms were combined with orthostatic tremor in one subject, and dysmetria with gait disturbances in four patients.

Imaging with MRI/CT was normal in four cases; in one patient, mild leptomeningeal enhancement was noted in MRI. In this case, oligoclonal bands were detected in CSF. In the other patients, CSF was normal. Two patients were treated symptomatically with propranolol, one with clonazepam; two of these patients had mild improvement and there was no recovery in the third. One patient was treated with antibiotics only but outcome data was not provided. One patient was treated with steroids and antibiotics with a complete recovery after one month of rehabilitation [76]. This last case, and similarly to the AMS time when symptoms appeared in the rest of the reported patients, may suggest, at least in some cases, the autoimmunological origin and necessity of immunological treatment.

Dystonia

One patient, a 78-year-old man, developed dystonia after COVID-19 [56] and one 38-year-old male was reported to have developed cervical dystonia 24 hours after SARS-CoV-2 vaccination. The 78-year-old with severe COVID-19 required mechanical ventilation and ICU admission. He developed upper limb asymetrical (right > left) dystonia and delirium. General examination of CSF was normal and negative for SARS-CoV-2. He was positivily tested for the presence of IgG autoantibodies in CSF against neurophil olfactory bulb, cerebellum and hippocampus neurons [56]. In another two cases, functional dystonia was diagnosed — in a 46-year-old female one week after mild COVID-19 [84], and in a 22-year-old female 24 hours after SARS-CoV-2 vaccination [85]. Moreover, dystonia has been reported as a side effect after COVID-19 vaccination in a total of 113 cases (53 after Pfizer/BioNTech, 23 after Moderna, 30 after AstraZeneca, and seven after Janssen) [86].

An important consideration regarding patients with dystonia during the pandemic is the limited availability of treating neurologists — a deterioration in the quality of life was observed in 65% of the 71 dystonia patients surveyed (due to delays in botulinum toxin appointments, depressed mood, less physical activity, and less access to rehabilitation) [86]. In an observational survey of Parkinson's Disease (PD) and dystonia patients treated with deep brain stimulation (DBS), 36% of dystonia patients reported difficulties in the management of the DBS device, and 100% discontinued outpatient neurological visits due to the lockdown [87].

Limited physician availability and delayed follow-up visits in patients with generalised dystonia treated with DBS should not delay implantable pulse generator battery replacement, as the dystonic state resulting from battery depletion can be potentially life-threatening [88].

Conclusions

Hyperkinetic movement disorders, although they are infrequent sequelae of SARS-CoV-2 infection, must be followed up carefully, as many of them are potentially treatable. This seems to be true specifically for ataxia, myoclonus and opsoclonus symptoms, whereas in cases of chorea the pathophysiology is more variable (not only autoimmunological) requiring specific or symptomatic treatment.

In all cases of movement disorders suspected of a SARS-CoV-2 infection history, even those without PCR confirmation, first line treatment with corticosteroids should be offered followed by IVIG or plasmapheresis. Patients already treated with DBS should be closely monitored as any delay in the replacement of batteries can prove fatal.

Conflicts of interest: *None.*
Funding: *None.*

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