Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Electrodiagnostic findings in COVID-19 patients: A single center experience

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HIGHLIGHTS

- Neuromuscular manifestations in COVID-19 patients have been observed, especially in the prolonged hospital setting.
- We describe a series of COVID-19 patients with a neuromuscular diagnosis.
- Electrodiagnostic studies play an important role in the diagnosis and prognosis of these patients.

ABSTRACT

Objective: Neurological manifestations in patients with coronavirus disease 2019 (COVID-19) have been reported from early features of anosmia and dysgeusia to widespread involvement of the central nervous system, peripheral nervous system, as well as the neuromuscular junction and muscle. Our study objective is to evaluate the electromyography and nerve conduction study (EMG/NCS) findings among COVID-19 patients and look for possible correlations.
Methods: This is a hospital-based retrospective observational study. All COVID-19 patients between the period of 1st January 2020 to 31st December 2020 undergoing an EMG/NCS were included.
Results: Eighteen patients (12 male and 6 female) were included. Mean age was 55 ± 12 years. 11 patients required intubation for a mean period of 18.6 days (range: 3–37 days). Electrodiagnostic findings were consistent with a myopathy in a majority of these patients (82%). Five of them also had a concurrent axonal neuropathy. In the remaining patients who did not require intubation (n = 7), three patients had myopathic EMG changes and one had Guillain Barre syndrome.
Conclusion: At this time, there are no neuromuscular-specific recommendations for patients who contract COVID-19. Only time and additional data will unveil the varying nature and potential neurological sequelae of COVID-19.
Significance: Myopathic EMG changes are commonly seen in critically ill COVID-19 patients, especially with a prolonged hospital stay.

1. Introduction

The coronavirus disease 2019 (COVID-19) is a severe acute respiratory syndrome (SARS) caused by the SARS-CoV-2 coronavirus. More than 209 million confirmed cases and over 4.4 million deaths have been reported worldwide, as of August 20, 2021 (Johns Hopkins University Coronavirus Resource Center, 2021). Neurological manifestations in COVID-19 have been reported from early features of anosmia and dysgeusia to widespread involvement of the central nervous system (CNS), peripheral nervous system (PNS), as well as the neuromuscular junction and muscle (Román et al.,

Abbreviations: AIDP, Acute idiopathic demyelinating polyneuropathy; AMAN, Acute motor axonal neuropathy; AMSAN, Acute motor sensory axonal neuropathy; CIM, Critical Illness myopathy; CIN, Critical illness neuropathy; CK, Creatine Kinase; CNS, Central nervous system; COVID-19, Coronavirus disease 2019; EMG, Electromyography; GBS, Guillain Barre syndrome; NCS, Nerve conduction study; PNS, Peripheral nervous system; SNAP, Sensory nerve action potential.

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Table 1
Characteristics of COVID-19 patients with EMG/NCS findings.

		Total (n)	Intubated (n)	Non-Intubated (n)
No of patients (n)		18	11	7
Age ± SD (in years)		55 ± 12	54 ± 12	55 ± 13
Gender	**Male**	67% (12)	73% (8)	57% (4)
	Female	33% (6)	27% (3)	43% (3)
Co-morbid	**DM**	50% (9)	64% (7)	29% (2)
		67% (12)	64% (7)	71% (5)
	HTN	22% (4)	36% (4)	0
	Asthma			
EMG/NCS Diagnosis	**Myopathy**	39% (7)	36% (4)	43% (3)
	Neuropathy	6% (1)	9% (1)	0
	Myopathy + Neuropathy	28% (5)	45% (5)	0
	GBS	11% (2)	9% (1)	14% (1)
	Intraspinal canal lesion	6% (1)	0	14% (1)
	Peroneal neuropathy	6% (1)	0	14% (1)
	Ulnar neuropathy + CTS	6% (1)	0	14% (1)
Mean CK Value in IU/L*		222	246	64
Range		(28–876)	(66–876)	(28–93).
Respiratory Symptoms	**Present**	89% (16)	100% (11)	71% (5)
	Absent	11% (2)	0	29% (2)
COVID-19 Treatment	**Steroids**	83% (15)	100% (11)	57% (4)
	Tocliziumab	44% (8)	55% (6)	29% (2)
	Plasmapharesis	22% (4)	18% (2)	29% (2)
	Antivirals	11% (2)	9% (1)	14% (1)
	None	17% (3)	0	43% (3)
Outcome	**Expired**	28% (5)	46% (5)	0
	Discharged	72% (13)	54% (6)	100% (7)
	Mean mRS score	3.9	4.3	3.8

Abbreviations: CK = Creatine kinase; CTS = Carpal tunnel syndrome; DM = Diabetes mellitus; EMG/NCS = Electromyography and nerve conduction studies; HTN = Hypertension; SD = Standard deviation.
* CK values are only of the patients with myopathic EMG changes.

2020). Different neuromuscular manifestations have been reported in COVID-19 patients with myalgia being the most common symptom (Paliwal et al., 2020). Critical illness myopathy (CIM) has been frequently seen in patients with severe COVID-19 disease with a prolonged hospital stay (Cabañes-Martínez et al., 2020). Our study objective is to evaluate the electromyography and nerve conduction study (EMG/NCS) findings among COVID-19 patients presenting to our neurophysiology laboratory and look for possible correlations.

2. Methods

This is a hospital-based retrospective observational study. All COVID-19 patients between the period of 1st January 2020 to 31st December 2020 referred to the neurophysiology laboratory at the Aga Khan University Hospital Karachi, Pakistan for an EMG/NCS were eligible to be included in this study. EMG/NCS was ordered by the primary intensive care physician or a neurologist taking part in the clinical care of patients. Inclusion criteria included $\geq$ 18 years of age and a COVID-19 infection confirmed by a reverse transcriptase-polymerase chain reaction (RT-PCR) assay of a nasopharyngeal swab sample. Patients with pre-existing neuromuscular disorders were excluded. This study was approved by the Ethical Review Committee of the AKUH (Ref: 2020–5469) and the informed consent requirement was waived.

The electrodiagnostic study consisted of motor and sensory nerve conduction studies followed by concentric needle electromyography examination. Sensory NCS were conducted with antidromic stimulation. All studies were performed using a Nicolet Viking machine. Low and high pass filters were set at 2 kHz and 20 kHz, and 20 Hz and 2000 Hz for motor studies and sensory studies, respectively, with a sweep speed of 2 ms/division. The stimulus duration was of 0.05 or 0.1msec, as needed. The acceptable limb temperature for performing NCS was $\geq$ 32 °C. In the event of low limb temperatures, the patients were warmed up using a heating pad or hot water bags to maintain the required temperature, as needed.

NCS: We studied motor and sensory NCS as per the protocol. In all patients, the nerves of the upper (median) and lower extremities (sural, peroneal, and posterior tibial nerves) were examined. In cases of peripheral neuropathy, a superficial peroneal or medial and lateral plantar nerves are additionally examined depending upon whether sural nerve was absent or present, respectively, in the lower extremities. Ulnar and/or radial nerves were also additionally performed in upper extremities in the peripheral neuropathy protocol.

EMG: Needle electrode examination was performed using disposable concentric needles. The number of muscles examined was determined by the patient's clinical history and electrodiagnostic findings, however, at least seven muscles in the upper extremities and six muscles in the lower extremities (both distal and proximal muscles) were examined in every patient. Amplitude and duration of motor unit action potentials (MUAPs) along with polyphasic potentials and recruitment pattern were evaluated, in addition to the presence or absence of spontaneous activity.

Myopathy: Myopathy is defined on the basis of NCS/EMG findings. The EMG findings of short motor unit action potentials, with decreased amplitude and duration, along with normal sensory and motor NCS were seen. However in some of the patients, low compound muscle action potentials were also noted.

Guillain Barre syndrome (GBS): GBS and its variants i.e. acute idiopathic demyelinating polyneuropathy (AIDP) (Albers and Kelly, 1989), acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN) (Ho et al., 1995) are defined on the basis of electrodiagnostic criterions. (Please review Supplementary File for details.)

Intraspinal canal lesion: An intraspinal canal lesion is labelled when (i) sensory nerve action potentials (SNAPs) are normal; (ii) Motor NCS is either normal or shows decreased CMAPs in the corresponding region(s). (iii) Needle EMG examination reveals acute

Table 2
Summary of all the patients.

No.	A/G	PMH	Respiratory symptoms	Intubation (days)	Neurological symptoms	Bulk/Tone/DTR	Motor Power (MRC Grading)	Sensory Exam	Time*	Treatment	Clinical Diagnosis	EMG/NCS Findings@	Outcome (mRS)	Mean Peak CK (IU/L)
1	64 M	DM, HTN	Fever and sore throat	Yes (7)	Quadriparesis	Normal/Decreased/ Absent	UL: Proximal:3/5 Distal: 3/5 LL: Proximal: 2/5 Distal: 3/5	Normal	23	HCQ, Steroids, Tocilizumab.	CIM/CIN	Myopathy (Non-irritable)	4	424
2	65 M	DM, HTN	Fever, cough, and dyspnea	Yes (10)	Quadriparesis (prox. > distal)	Normal/Decreased/ Absent	UL: Proximal:1/5 Distal: 3/5 LL: Proximal: 2/5 Distal: 2/5	Normal	14	PP, Steroids, Tocilizumab.	CIM/CIN	Myopathy (Non-irritable) & motor axonal PN	4	117
3	44 F	DM, Asthma	Fever, cough, and dyspnea	Yes (24)	Difficult to wean off/ Quadriparesis	Normal/Decreased/ Absent	UL: Proximal: 0/5 Distal: 0/5 LL: Proximal: 1/5 Distal: 1/5	N/A	20	Steroids, Tocilizumab.	CIM/CIN	Myopathy (Irritable) & sensorimotor axonal PN	Expired	189
4	56 F	HTN, Asthma	Fever and dyspnea	Yes (15)	Difficult to wean off/ Quadriparesis	Normal/Decreased/ Absent	UL: Not assessed due to stockings. LL: Proximal: 0/5 Distal: 0/5	N/A	42	PP, Steroids, Tocilizumab.	CIM/CIN	Myopathy (Irritable)	Expired	876
5	59 M	HTN	Fever and sore throat	No	Bilateral lower limb weakness	Normal/Normal/ Absent ankle reflex; rest of the reflexes are normal (2 +)	UL: Proximal: 5/5 Distal: 5/5 LL: Proximal: 5/5 Distal: bilateral dorsiflexion: 1/5 Bilateral platerflexion: 3/5	Decreased sensations bil. lower limbs.	26	None	Radiculitis/ CPN	Acute bil. L2 & L5, S1 intraspinal canal lesion	2	94
6	59 F	HTN, Asthma	Fever and dyspnea	Yes (37)	Unable to wean off / Quadriparesis	Normal/Decreased/ Decreased (1 +)	UL: Proximal: 1/5 Distal: 1/5 LL: Proximal: 1/5 Distal: 1/5	N/A	40	Steroids	CIM	Sensorimotor axonal PN	5	75
7	38 M	None	Sore throat and cough	No	Bilateral lower limb weakness	Normal/Normal/ Decreased (1 +)	UL: Proximal: 5/5 Distal: 5/5 LL: Proximal: 3/5 Distal: 3/5	Normal	35	PP, Steroids, Tocilizumab.	CIM	Myopathy (Irritable)	3	28
8	41 M	Asthma	Fever and dyspnea	Yes (18)	Bilateral Lower limb weakness	Normal/Normal/ Normal	UL: Proximal: 5/5 Distal: 5/5 LL: Proximal: 2/5 Distal: 3/5	Normal	29	Steroids, IVIG	CIM	Myopathy (Irritable)	4	NA

(*continued on next page*)

Table 2 (*continued*)

No.	A/G	PMH	Respiratory symptoms	Intubation (days)	Neurological symptoms	Bulk/Tone/DTR	Motor Power (MRC Grading)	Sensory Exam	Time*	Treatment	Clinical Diagnosis	EMG/NCS Findings@	Outcome (mRS)	Mean Peak CK (IU/L)
9	77 M	DM, HTN, IHD	Fever and dyspnea	Yes (31)	Difficult to wean off/ Quadriparesis	Normal /Normal/ Decreased (1 +)	UL: Proximal: 3/5 Distal: 3/5 LL: Proximal: 2/5 Distal: 3/5	Normal	15	Steroids	CIM	Myopathy (Non-irritable) & sensiromotor axonal PN	Expired	NA
10	64 M	None	Dyspnea	Yes (18)	Difficult to wean off/ Quadriparesis	Normal/Normal/ Absent	UL: Proximal: 0/5 Distal: 0/5 LL: Proximal: 0/5 Distal: 0/5	N/A	16	Steroids, Tocilizumab	CIM/CIN	Myopathy (Non-irritable)	Expired	138
11	61 M	DM, HTN	Fever, cough, and dyspnea	No	Left foot drop	Normal/Normal/ Normal	UL: Proximal: 5/5 Distal: 5/5 LL: Proximal: 5/5 Distal: 3/5 (dorsi 2/5, plantar 4/5)	Dec. sensations dorsum of the foot	39	Steroids	CPN/L5 radic.	Acute bil. common peroneal MN	3	138
12	46 M	DM	Fever, cough, and dyspnea	Yes (12)	Quadriparesis	Normal/Normal/ Decreased	UL: Proximal: 1/5 Distal: 1/5 LL: Proximal: 1/5 Distal: 1/5	Decreased sensation in glove stocking pattern	120	Remdesivir, Steroids, Tocilizumab	CIM/CIN	Sensorimotor axonal PN + myopathy (Non-irritable)	5	NA
13	76 M	DM, HTN	Fever, cough, and dyspnea	Yes (3)	Quadriparesis	Normal/Decreased/ Absent	UL: Proximal: 0/5 Distal: 0/5 LL: Proximal: 0/5 Distal: 0/5	**N/A**	10	Steroids	GBS	GBS (AMSAN)	5	NA
14	46 F	None	None	No	Bil. Hand numbness	Normal/Normal/ Normal	UL: Proximal: 5/5 Distal: 4/5 LL: Proximal: 5/5 Distal: 5/5	Normal	180	None	GBS	Bil. ulnar neuropathy + CTS	2	
15	64 M	DM, HTN	None	No	Generalized weakness	Normal/Decreased/ Decreased (1 +)	UL: Proximal: 3/5 Distal: 4/5 LL: Proximal: 2/5 Distal: 4/5	Normal	45	None	CIM	Myopathy (Non-irritable)	4	68
16	47 M	DM, HTN	Fever, cough, and dyspnea	Yes (30)	Difficult to wean off/ Quadriparesis	Normal/Decreased/ Absent	UL: Proximal: 3/5 Distal: 2/5 LL: Proximal: 2/5 Distal: 1/5	Decreased sensation in glove stocking pattern	35	Steroids	CIM/CIN	Sensorimotor axonal PN + Myopathy (Irritable)	Expired	66
17	54 F	HTN	Fever, sorethroat and dyspnea	No	Quadriparesis	Normal/Decreased/ Decreased (1 +)	UL: Proximal: 2/5 Distal: 3/5 LL:	Normal	45	Remdesivir, Steroids, Tocilizumab	CIM	Myopathy (Non-irritable)	5	93

Table 2 (*continued*)

No.	A/G	PMH	Respiratory symptoms	Intubation (days)	Neurological symptoms	Bulk/Tone/DTR	Motor Power (MRC Grading)	Sensory Exam	Time*	Treatment	Clinical Diagnosis	EMG/NCS Findings@	Outcome (mRS)	Mean Peak CK (IU/L)
18	34 F	HTN, Hypothyroid, SLE	Fever, dyspnea and abdominal pain	No	Quadriparesis	Normal/Decreased/ Absent	Proximal: 1/5 Distal: 2/5 UL: Proximal: 2/5 Distal: 2/5 LL: Proximal: 1/5 Distal: 0/5	Normal	4	PP, Steroids	GBS	GBS (AMAN)	5	29

* Time from the onset of symptoms to EMG/NCSs (Electromyography and nerve conduction studies). @ Irritable myopathy means presence of abnormal spontaneous activity (e.g. positive sharp waves, fibrillations or complex, repetitive discharges) on needle EMG examination; Non-irritable means absence of abnormal spontaneous activity.Abbreviations A = Age AF = Atrial fibrillation AKI = Acute kidney injury AMAN = Acute motor axonal polyneuropathy AMSAN = Acute motor and sensory axonal polyneuropathy Bil. = Bilateral CIM = Critical illness myopathy CIN = Critical illness neuropathy CPN = Common peroneal neuropathy CTS = Carpal tunnel syndrome DM = Diabetes mellitus DTR = Deep tendon reflexes F = Female G = Gender GBS = Guillain Barre syndrome HCQ = Hydroxychloroquine HTN = Hypertension IVIG = Intravenous immune globin LL = Lower limb M = Male MN = Mononeuropathy mRS = modified Rankin scale NA = Not available PN = polyneuropathy PP = Plasmapheresis Radic. = Radiculopathy SLE = Systemic lupus erythematosus UL = Upper limb.

and/or chronic motor axon loss changes in a myotome pattern, without a distal-to-proximal gradient.

3. Results

Eighteen patients (12 male and 6 female) were included in our study (review Table 1 and 2 for details). The mean age was 55 ± 12 years. A total of 2,426 adult patients ($\geq$ 18 years) had a diagnosis of COVID-19 patients either at the time of admission or during the course of the hospital stay during the study period. Our study subjects had multiple comorbidities, with the most common comorbid condition being hypertension (67%, n = 12), followed by diabetes mellitus (50%, n = 9) and asthma (22%, n = 4). The mean duration from the diagnosis of COVID-19 infection to the performance of the electrodiagnostic study was variable, ranging between 4 to 180 days, with a mean duration of 41 days.

Eleven patients suffered from severe COVID-19 infection and required intubation for a mean period of 18.6 days (range: 3–37 days). Indication for EMG/NCS in these intubated patients was either a difficulty to wean off the ventilator support or development of flaccid quadriparesis. Electrodiagnostic findings were consistent with a myopathy in a majority of these patients (82%, n = 9). Five of them also had a concurrent axonal neuropathy. In 4 out of 9 patients with myopathic EMG changes (44%), spontaneous activity consisting of fibrillations and positive sharp waves were seen. Mean peak creatine kinase (CK) value was 246 IU/L (range: 66–876 IU/L). Unfortunately, muscle biopsy was not performed in any of the patients, which is one of the major limitations of our study. One patient had chronic peripheral neuropathy without myopathic EMG changes, and one had an acute motor and sensory axonal neuropathy (AMSAN) variant of GBS. Understandably, these patients had multiple comorbidities and had poor outcome with a 46% mortality. All of the intubated patients received systemic steroids (100%) and half of them also received tocilizumab.

In the remaining patients who did not require intubation (n = 7), three patients had myopathic EMG changes (irritable in 1), one patient each had GBS (AMAN variant), bilateral common peroneal mononeuropathies, bilateral ulnar mononeuropathies along with bilateral carpal tunnel syndrome (CTS), and an active on chronic intraspinal canal lesion affecting L2 and L5-S1 myotomes. Interestingly, none of these patients had a large-fiber peripheral neuropathy. Mean peak CK value for patients with myopathic EMG changes was 64 IU/L (range: 28–93 IU/L). Half of the non-intubated patients received steroids and two patients (29%) also received tocilizumab. No mortality was seen in this non-intubated patient group.

4. Discussion

Neurological manifestations in COVID-19 occur in one-third of all COVID-19 infections (Mao et al., 2020). A drastic increase of critical illness myopathy (CIM) has been observed in COVID-19 survivors who have been exposed to long-term mechanical ventilation. The underlying mechanism is not well understood and may involve the release of factors affecting muscle secondary to immobilization (Lönnqvist et al., 2020). It is also proposed that cytokine release causes inactivation of sodium channels, thereby causing a slowing of muscle fiber conduction velocity and a simultaneous increase in calcium permeability that leads to myonecrosis (Friedrich et al., 2015).

Cabañes-Martínez et al. published the first study reporting neuromuscular involvement in critically ill COVID-19 patients. They diagnosed 11 patients electrodiagnostically with either CIP or CIM out of 225 COVID-19 patients, with the latter being present in the majority (Cabañes-Martínez et al., 2020). Another recently

published study on six COVID-19 intubated patients afflicted with acute flaccid quadripalegia demonstrated CIM in almost all of them. Five of these patients recovered after receiving COVID-19 treatment while one succumbed to sepsis (Madia et al., 2020). In our study, it is difficult to ascertain whether the myopathic EMG changes were secondary to the critical-illness or due to direct virus-related effects. Laboratory parameters like LDH, CK, aldolase, ferritin may be elevated following post-COVID-19 myopathy. Another study revealed elevated CK in up to one-third of admitted patients (Wang et al., 2020). Manzano et al. reported a case of COVID-19 infection and myopathy in which a deltoid muscle biopsy showed raised levels of type 1 interferon, which may validate type 1 interferonopathy as a probable cause of myopathy in such individuals at a more cellular level (Manzano et al., 2020).

Growing evidence suggests that neuromuscular manifestations like GBS, rhabdomyolysis, and neuralgic amyotrophy are also on the rise (Guidon and Amato, 2020; Pergolizzi et al., 2021). In another systemic review, AIDP was reported to be the most common variant of GBS in COVID-19 patients (De Sanctis et al., 2020). There are also reports of the Miller Fischer variant of GBS being associated with COVID (Senel et al., 2020); (Ray, 2020); Lantos et al., 2020). Interestingly in our study, two patients who were diagnosed with GBS either had the AMAN and AMSAN variant, respectively. The pathogenesis of GBS in COVID-19 is still unclear. Whether COVID-19 produces antibodies against specific gangliosides still needs to be determined.

At this time, there are no neuromuscular-specific recommendations for patients who contract COVID-19. Only time and additional data will unveil the varying nature and potential neurological sequelae of COVID-19 (Guidon and Amato, 2020). Meanwhile, clinicians must keep a cautious low threshold for suspecting myopathy in patients who develop symmetrical muscle weakness in the setting of COVID-19 infection, lengthy ICU stay, or both combined. Ideally, longitudinal studies should undertake careful neurological imaging and electrophysiological examinations to understand the congruent interplay between COVID-19 induced myopathy and CIM. This can be followed by adequate access to rehabilitation facilities in patients who require rehabilitative services even after the resolution of infection.

5. Limitations

Lack of muscle biopsy and a small number of patients were the major limitations of our study. Due to the retrospective nature of this study and small numbers, a causal relationship cannot be determined. Further, drug adverse effects and other confounders, such as comorbid conditions, also could not be excluded.

6. Ethical Publication Statement

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines."

CRediT authorship contribution statement

Sajid Hameed: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Validation, Visualization, Writing - original draft, Writing - review & editing. **Ayisha Farooq Khan:** Data curation, Investigation, Methodology, Visualization, Writing - original draft, Writing - review & editing. **Sara Khan:** <Conceptualization, Data curation, Formal analysis, Methodology, Project administration, Resources, Supervision, Validation, Visualization, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clinph.2021.10.001.

References

Albers JW, Kelly JJ. Acquired inflammatory demyelinating polyneuropathies: clinical and electrodiagnostic features. Muscle Nerve 1989;12(6):435–51. https://doi.org/10.1002/(ISSN)1097-4598.

Cabañes-Martínez L, Villadóniga M, González-Rodríguez L, Araque L, Díaz-Cid A, Ruz-Caracuel I, Pian H, Sánchez-Alonso S, Fanjul S, del Álamo M, Regidor I. Neuromuscular involvement in COVID-19 critically ill patients. Clin Neurophysiol 2020;131(12):2809–16. https://doi.org/10.1016/j.clinph.2020.09.017.

De Sanctis P, Doneddu PE, Viganò L, Selmi C, Nobile-Orazio E. Guillain-Barré syndrome associated with SARS-CoV-2 infection. A systematic review. Eur J Neurol 2020;27(11):2361–70. https://doi.org/10.1111/ene.v27.1110.1111/ene.14462.

Friedrich O, Reid MB, Van den Berghe G, Vanhorebeek I, Hermans G, Rich MM, Larsson L. The sick and the weak: neuropathies/myopathies in the critically ill. Physiol Rev 2015;95(3):1025–109. https://doi.org/10.1152/physrev.00028.2014.

Guidon AC, Amato AA. COVID-19 and neuromuscular disorders. Neurology 2020;94(22):959–69. https://doi.org/10.1212/WNL.0000000000009566.

Ho TW, Mishu B, Li CY, Gao CY, Cornblath DR, Griffin JW, et al. Guillain-Barre syndrome in northern China Relationship to Campylobacter jejuni infection and anti-glycolipid antibodies. Brain 1995;118:597–605. https://doi.org/10.1093/brain/118.3.597.

Johns Hopkins University Coronavirus Resource Center. Available from: https://coronavirus.jhu.edu/ [Accessed 20 August 2021].

Lantos JE, Strauss SB, Lin E. COVID-19–associated miller fisher syndrome: MRI findings. *AJNR Am J Neuroradiol.* 2020;41:1184-1186. doi: 10.3174/ajnr.A6609

Lönnqvist P-A, Bell M, Karlsson T, Wiklund L, Höglund A-S, Larsson L. COVID-19–associated miller fisher syndrome: MRI findings. AJNR Am J Neuroradiol 2020;125(3):e334–6. https://doi.org/10.1016/j.bja.2020.05.056.

Madia F, Merico B, Primiano G, Cutuli SL, De Pascale G, Servidei S. Acute myopathic quadriplegia in patients with COVID-19 in the intensive care unit. Neurology 2020;95(11):492–4. https://doi.org/10.1212/WNL.0000000000010280.

Manzano GS, Woods JK, Amato AA. Covid-19–Associated Myopathy Caused by Type I Interferonopathy. N Engl J Med 2020;383(24):2389–90. https://doi.org/10.1056/NEJMc2031085.

Mao L, Jin H, Wang M, Hu Yu, Chen S, He Q, Chang J, Hong C, Zhou Y, Wang D, Miao X, Li Y, Hu Bo. Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China. JAMA Neurol 2020;77(6):683. https://doi.org/10.1001/jamaneurol.2020.1127.

Paliwal VK, Garg RK, Gupta A, Tejan N. Neuromuscular presentations in patients with COVID-19. Neurol Sci 2020;41(11):3039–56. https://doi.org/10.1007/s10072-020-04708-8.

Pergolizzi Jr JV, Raffa RB, Varrassi G, Magnusson P, LeQuang JA, Paladini A, et al. Potential neurological manifestations of COVID-19: a narrative review. *Postgrad Med.* 2021;1-11. doi: 10.1080/00325481.2020.1837503.

Ray A. Miller Fisher syndrome and COVID-19: is there a link? BMJ Case Rep 2020;13(8):e236419. https://doi.org/10.1136/bcr-2020-236419.

Román GC, Spencer PS, Reis J, Buguet A, Faris MEA, Katrak SM, Láinez M, Medina MT, Meshram C, Mizusawa H, Öztürk S, Wasay M. The neurology of COVID-19 revisited: A proposal from the Environmental Neurology Specialty Group of the World Federation of Neurology to implement international neurological registries. J Neurol Sci 2020;414:116884. https://doi.org/10.1016/j.jns.2020.116884.

Senel M, Abu-Rumeileh S, Michel D, Garibashvili T, Althaus K, Kassubek J, Otto M. Miller-Fisher syndrome after COVID-19: neurochemical markers as an early sign of nervous system involvement. Eur J Neurol 2020;27(11):2378–80. https://doi.org/10.1111/ene.v27.1110.1111/ene.14473.

Wang D, Hu Bo, Hu C, Zhu F, Liu X, Zhang J, Wang B, Xiang H, Cheng Z, Xiong Y, Zhao Y, Li Y, Wang X, Peng Z. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus–infected pneumonia in Wuhan China. JAMA 2020;323(11):1061. https://doi.org/10.1001/jama.2020.1585.