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.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
Electrodiagnostic findings in COVID-19 patients: A single center experience

Sajid Hameed *, Ayisha Farooq Khan, Sara Khan
Department of Neurology, Aga Khan University, Pakistan

A R T I C L E   I N F O

Article history:
Accepted 4 October 2021
Available online 13 October 2021

Keywords:
Electromyography
EMG/NCS
COVID-19
Myopathy
Coronavirus

H I G H L I G H T S
• 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.

A B S T R A C T

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.

© 2021 International Federation of Clinical Neurophysiology. Published by Elsevier B.V. All rights reserved.

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.,...
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 ≥ 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 electrodagnostic study consisted of motor and sensory nerve conduction studies followed by concentric needle electromyography examination. Sensory NCS were conducted with antidiode stimulation. All studies were performed using a Nicolet Viking machine. Low and high pass filters were set at 2 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 ≥ 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 electrodagnostic 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 electrodagnostic 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 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 ± 12 |
| Gender | Male | Female | 33% (6) | 27% (3) |
| Co-morbid | DM | 50% (9) | 64% (7) | 29% (2) |
| | HTN | 67% (12) | 64% (7) | 71% (5) |
| | Asthma | 22% (4) | 36% (4) | 0 |
| EMG/NCS Diagnosis | Myopathy | 39% (7) | 36% (4) | 43% (3) |
| | Neuropathy | 6% (1) | 9% (1) | 0 |
| | Myopathy + Neuropathy | 28% (5) | 43% (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) |
| | Tacizumab | 44% (8) | 55% (6) | 20% (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.
| 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) | Quadriplegic | 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) | Quadriplegic (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/Quadriplegic | 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/Quadriplegic | 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    | DM, HTN Fever and sore throat | No | Bilateral lower limb weakness | Normal/Normal/Absent ankle reflex; rest of the reflexes are normal (2 + ) | 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 /Quadriplegic | Normal/Decreased Decreased (1 + ) | 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 + ) | 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 | Normal | 29  | Steroids, IVIG | CIM Myopathy (Irritable) | 4  | NA |

(continued on next page)
| No. | A/G | PMH | Respiratory symptoms | Intubation (days) | Neurological symptoms | Bulk/Tone/DTR (MRC Grading) | Motor Power | Sensory Exam | Time¹ | Treatment | Clinical Diagnosis | EMG/NCS Findings² | Outcome (mRS) | Mean Peak CK (IU/L) |
|-----|-----|-----|----------------------|------------------|----------------------|-----------------------------|------------|-------------|------|-----------|-------------------|-----------------|-------------|-------------------|
| 9   | 77  | M   | Fever and dyspnea    | Yes (31)         | Difficult to wean off/Quadripareisis | Normal /Normal/ Decreased (1 + ) | UL: Proximal: 3/5 Distal: 3/5 LL: Proximal: 3/5 Distal: 3/5 | Normal      | 15         | Steroids   | CIM Myopathy (Non-irritable) & sensiromotor axonal PN | Expired         | NA          |
| 10  | 64  | M   | Dyspnea              | Yes (18)         | Difficult to wean off/Quadripareisis | Normal/NORMAL/ Absent         | N/A        | 16         | Steroids, Tocilizumab | CIM/CIN         | Myopathy (Non-irritable) | Expired         | 138         |
| 11  | 61  | M   | Fever, cough, and dyspnea | No              | Left foot drop         | Normal/Normal/ Normal         | UL: Proximal: 5/5 Distal: 5/5 LL: Proximal: 5/5 Distal: 5/5 | Dec. sensations dorsum of the foot | 39         | Steroids   | CPN/L5 radic. | Acute bil. common peroneal MN | 3              | 138         |
| 12  | 46  | M   | Fever, cough, and dyspnea | Yes (12)        | Quadripareisis         | 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   | Fever, cough, and dyspnea | Yes (3)         | Quadripareisis         | Normal/Decreased/ Absent      | N/A        | 10         | Steroids   | GBS GBS (AMSAN) | 5              | NA          |
| 14  | 46  | F   | None                  | No               | Bil. Hand numbness     | Normal/Normal/ Normal         | Normal      | 180        | None        | GBS        | Bil. ulnar neuropathy + CTS | 2              | 132         |
| 15  | 64  | M   | None                  | No               | Generalized weakness   | Normal/Decreased/ Decreased (1 + ) | Normal      | 45         | None        | CIM        | Myopathy (Non-irritable) | 4              | 68          |
| 16  | 47  | M   | Fever, cough, and dyspnea | Yes (30)        | Difficult to wean off/Quadripareisis | Normal/Decreased/ Absent      | UL: Proximal: 3/5 Distal: 4/5 LL: Proximal: 5/5 Distal: 5/5 | Decreased sensation in glove stocking pattern | 35         | Steroids   | CIM/CIN         | Sensorimotor axonal PN + Myopathy (Irritable) | Expired         | 66          |
| 17  | 54  | F   | Fever, sorethroat and dyspnea | No             | Quadripareisis         | Normal/Decreased/ Decreased (1 + ) | Normal      | 45         | Remdesivir, Steroids, Tocilizumab | CIM        | Myopathy (Non-irritable) | 5              | 93          |
Table 2 (continued)

| No. | Age (years) | Gender | Diagnosis | Sensory Exam | Motor Power | EMG/NCS Findings | CK (IU/L) | Outcome (mRS) | Treatment |
|-----|-------------|--------|-----------|--------------|-------------|------------------|----------|---------------|-----------|
| 18  | 55 ± 12     | M      | GBS (AMAN) | Absent       | Normal      | MRC Grading      | 29       | 5             | IVIG     |
|     |             |        |           |              |             |                  |          |               |           |

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 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 creatine kinase (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 (Lönqvist 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 electrodagnostically 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 affected with acute flaccid quadriplegia 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 neurolgic 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 variants, 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 services 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

Sajad 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 electrodagnostic features. Muscle Nerve 1989;12(6):435–51. https://doi.org/10.1002/1097-9498.

Cabañes-Martínez L, Villadóniga M, González-Rodríguez L, Arape L, Díaz-Cid A, Ruiz-Caracuel L, Plan H, Sánchez-Alonso S, Fanjul S, del Álamo M, Regidor L. 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.v27.11111.ene.14462.

Friedrich O, Reid MB, Van den Berge G, Vanhorebeek I, Herrmans 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.00026.2014.

Guidon AC, Amato AA. COVID-19 and neuromuscular disorders. Neurology 2020;94(22):e599–609. https://doi.org/10.1212/WNL.0000000000009566.

Ho TW, Mishu B, Li CY, Gao CY, Cornblath DR, Griffin JW, et al. Guillain-Barré 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 SR, Lin E. COVID-19–associated miller fisher syndrome: MRI findings. AJNR Am J Neuroradiol. 2020;41:1184-1186. doi: 10.3174/ajnr.A6600

Lönqvist P-A, Bell M, Karlsson T, Wiklund L, Högland A-S, Larsson L. COVID-19–associated miller fisher syndrome: MRI findings. AJNR Am J Neuroradiol 2020;41(3):e334–6. https://doi.org/10.3174/ajnr.a5056.

Madia F, Merico B, Primiano G, Cutuli SL, De Pascale G, Servidet S. Acute myopathic quadriplegia in patients with COVID-19 in the intensive care unit. Neurology 2020;95(9):1492–4. https://doi.org/10.1212/WNL.0000000000008183.

Manzano CS, Woods JK, Amato AA, COVID-19–Associated Myopathy Caused by Type 1 Interferonopathy. N Engl J Med 2020:383(24):2389–90. https://doi.org/10.1056/NEJM20201105.

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 Neurology 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. Neurosci Lett 2020;41(1):3039–56. https://doi.org/10.1016/j.neulet.2020.07.027.20200702.04708.8.

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;11:11. doi:10.1002/1941-8185.2019703.

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

Román GC, Spencer PS, Reis J, Buguet A, Faris MEA, Kraitm SM, Lainé 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):2376–80. https://doi.org/10.1111/ene.v27.11111.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.2020.17485.