Received: 20 February 2022 | Revised: 24 June 2022 | Accepted: 17 July 2022

DOI: 10.1111/1756-185X.14409

International Journal of Rheumatic Diseases WILEY

ORIGINAL ARTICLE

Prevalence of peripheral neuropathy and myopathy in patients post-COVID-19 infection

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Abstract

Background: Severe acute respiratory syndrome (SARS-CoV-2), caused by the Coronavirus 2019 (COVID-19), has become a life-threatening epidemic, affecting multiple organs, including the nervous system. Recent studies have documented that COVID-19-associated peripheral neuropathy is a common and frequent problem, with central and peripheral nervous system complications.

Objective: This work aims to evaluate the peripheral nerves and muscle involvement after COVID-19 infection, in addition to studying the prevalence rate and risk factors of their affection.

Methods: The study involved 400 patients, divided into 2 groups, with a history of COVID-19 infection with or without symptoms of neuromuscular affection, and 30 gender- and age-matched healthy volunteers were involved as controls. They were referred to the Department of Rheumatology and Rehabilitation for electro-diagnosis. All participants performed complete clinical examination and laboratory measures with an electrophysiological study.

Results: The prevalence of peripheral neuropathy and myopathy in post-COVID-19 patients was 56.3% among all patients. A significant difference was detected among patients of both groups regarding serum creatine phosphokinase level, clinical signs, and electrophysiologic findings of neuropathy and myopathy compared to the control group, with more prominent features among the symptomatic group. Histories of hospitalization, severe and long-lasting respiratory symptoms were risk factors for developing neuromuscular complications.

Conclusions: The present study could indicate that muscle involvement and peripheral nerve affection are common problems even among asymptomatic patients after COVID-19 infection, especially in the presence of any risk factors.

KEYWORDS
COVID-19, electrophysiology, myopathy, peripheral neuropathy, prevalence, risk factors

1 | INTRODUCTION

Severe acute respiratory syndrome (SARS-CoV-2), caused by COVID-19, is a life-threatening epidemic because it affects several organs, including the nervous, cardiovascular, and renal systems.[1–6]

Myopathy, neuropathy, polyradiculopathy (Guillain-Barré syndrome), stroke, cerebral perfusion abnormalities, and other neuromuscular manifestations of an ever-expanding spectrum have been reported.[7–10]

Retrograde neurotransmission through infected neurons is one of the recently discovered routes of COVID-19 entry into the central

nervous system, including its entry through the olfactory nerve and vascular endothelium infection.[4,11]

It has been suggested that the severity of COVID-19 infection is highly associated with neurological manifestations, such as cytokine storm, with rapid cytokine release that results from the host's immune response reaction to the viral infection, causing neuropathy and neurological manifestations. Thus, till now, it is unknown whether the neurological manifestations caused by COVID-19 are caused by the direct viral infection or indirect systemic inflammation in response to the virus infection.[12]

Recent studies documented that COVID-19-associated peripheral neuropathy is a common and frequent problem, with neuromuscular complications. This phenomenon is particularly common in those with comorbidities, such as diabetes mellitus, which may result from immune processes or as side effects of some medications used to manage COVID-19 symptoms, such as hydroxychloroquine, clindamycin, and steroids. To a lesser extent, prolonged hospitalization may cause entrapment neuropathy (peripheral nerve compression).[12–16]

Numerous studies reported myalgia or muscle fatigue during acute infection with COVID-19, which is likely to develop among critically ill patients and adversely affect patient outcomes.[17]

Accordingly, this study aims to evaluate the peripheral nerves and muscle involvement in (symptomatic and asymptomatic) patients post-COVID-19 infection, in addition to examining the prevalence rate and risk factors related to their affection.

2 | METHODS

This cross-sectional study was conducted on 400 patients with a history of COVID-19 infection who went to the electrophysiological unit at the Physical Medicine, Rheumatology and Rehabilitation Department. The study was approved by the Institutional Review Board (IRB) of the Faculty of Medicine, Menoufia University, with an IRB number PMRR620212, and the patients' informed consents were signed by all participants. Patients' data were collected from March 2020 to December 2021 from Menoufia University Hospital.

The current study included 400 patients with a history of COVID-19 infection, above18 years old. Both genders were divided into 2 groups; the first group included 210 patients who had symptoms of neuropathy and myopathy involving pain (burning, stabbing, or shooting pain in the affected areas), tingling, numbness, paresthesia of the limbs, weakness, myalgia, and easy fatigability. The second group included asymptomatic post-COVID-19 patients, in addition to 30 non-COVID-19 infected age- and gender-matched healthy volunteers who served as the control group.

Patients in the study groups were diagnosed having COVID-19 according to the New COVID-19 Pneumonia Prevention and Control Program (5th edition) by meeting 1 or both criteria of chest computed tomography symptoms and reverse transcription-polymerase chain reaction (RT-PCR), published by the WHO interim guidance.[18]

The present study excluded patients with previous trauma, surgery, fractures, systemic inflammatory or metabolic disorders, pregnancy, and a history of smoking or alcohol consumption that can cause neuropathy or myopathy.

All subjects had general and local clinical neurological examination of the 4 limbs, including assessment of muscle tone, power, endurance, sensations, reflexes, and pain assessment. In addition, they underwent laboratory measurements, including erythrocyte sedimentation rate, complete blood count (CBC), C-reactive protein, liver and kidney functions, hepatitis C virus antibody, and serum creatine phosphokinase (CPK). Furthermore, the electrophysiological parameters, including the study of sensory and motor conductivity of the median, ulnar, radial, tibial, peroneal, and sural nerves, were included with respect to the distal motor and peak sensory latency, amplitude, motor and sensory conduction velocity. The electromyography of 1st dorsal interosseous, biceps, triceps, gastrocnemius-soleus, tibialis posterior, and vastus lateralis was performed on both sides at rest (regarding insertional activity), at minimal activity (regarding motor unit action potential [MUAP] amplitude, and duration), and at maximal activity (regarding interference pattern) using a Nihon Kohden apparatus to assess the grade of nerve and muscle affection post-COVID-19 infection in symptomatic and asymptomatic patients vs (non-infected) healthy controls, for early detection of any muscular affection to improve management and prevent a worsening of the patients' outcomes. The assessment was performed by the same investigator.

3 | STATISTICAL STUDY

On an IBM-compatible computer, data were tabulated and analyzed using SPSS (Statistical Package for Social Science) Software version 20 (SPSS Inc., Chicago, IL, USA). The quantitative data were described as numbers and percentages, and also described as mean ± *SD*, median and interquartile range (IQR). Student's *t* test and Mann–Whitney *U* test were used to compare 2 sets of qualitative data with normal and abnormal distribution, respectively. Analysis of variance and Kruskal-Wallis tests were used to assess the difference of more than 2 groups in the normal and abnormal distribution of data, respectively. Qualitative data were analyzed using the Chi-square test. The independent risk factors for neuromuscular affection among COVID-19 patients were assessed using univariate and multivariable binary logistic regression analysis, which was presented as odds ratios (OR) with 95% confidence intervals (CI). The significance was considered when a *P* value was less than .05.[19]

4 | RESULTS

As shown in Table 1, there were insignificant differences between the 3 studied groups regarding age and gender. Moreover, both patient groups had a non-significant difference in the time-lapsed post-infection period.

TABLE 1 Demographic and clinical data of patients in the study groups

	G1 (symptomatic) N = 210	G2 (asymptomatic) N = 190	G3 (control) N = 30	*P* value
Age	37.93 ± 7.4	39.78 ± 6.9	35.7 ± 8.6	.708
Gender No. (%)				
Male	130 (62%)	120 (63%)	19 (63.3%)	.96
Female	80 (38%)	70 (37%)	11 (36.7%)	
Time of study post-infection, d				
Median IQR	92 (30–220)	87 (26–208)	-	.6
Clinical data, all patients				
Neuropathy	120 (57.2%)	40 (21%)	0 (0%)	<.001
Myopathy	50 (24%)	15 (7.8%)	0 (0%)	<.001

Note: P1 = G1 vs G2,P2 = G1 vs G3, P3 = G2 vs G3.
Abbreviation: IQR, interquartile range.

TABLE 2 Clinical and laboratory measures among subjects of the studied groups

Lab measures, and clinical symptoms and signs	G1 (symptomatic) N = 210	G2 (asymptomatic) N = 190	G3 (control) N = 30	*P* value
Serum CPK, μg/mL	754 ± 120	221 ± 65	93 ± 7.9	P1 = .001 P2 = .001 P3 = .001
Decreased/absent ankle reflexes	46 (22%)	10 (5%)	0 (0%)	P1 = .0001 P2 = .004 P3 = .19
Impaired pinprick sensation of feet/toes	55 (26%)	9 (4.7%)	0 (0%)	P1 = .0001 P2 = .001 P3=0.22
Impaired vibration of toes	67 (32%)	11 (5.8%)	0 (0%)	P1 = .001 P2 = .001 P3 = .18
Weakness of shoulder girdle muscles	19 (9%)	10 (5.3%)	0 (0%)	P1 = .14 P2 = .09 P3 = .19
Weakness of pelvic girdle muscles	31 (14.8%)	10 (5.3%)	0 (0%)	P1 = .002 P2 = .001 P3 = .19

Note: P1 = G1 vs G2, P2 = G1 vs G3, P3 = G2 vs G3.
Abbreviation: CPK, creatin phosphokinase.

Prevalence of post-COVID-19 neuromuscular affection among all patients was 56.3%, and it was 81% among symptomatic patients and 28.8% among the asymptomatic group.

In the symptomatic group, about 57.2% had neuropathy, and 24% had myopathy vs 21%, and 7.8%, respectively, in the asymptomatic group.

The elevated serum CPK level and clinical signs of neuropathy and myopathy were characteristic of the symptomatic patients group when compared to both the control and asymptomatic groups, as in Table 2.

Table 3 showed a significantly higher rate of sensorimotor demyelinating and axonal polyneuropathy of the studied nerves among the patient groups compared to the controls. Additionally, it showed a significantly higher rate of these findings among the symptomatic group than the asymptomatic one.

Table 4 showed a statistically significant presence of insertional activity, with decreased amplitude and prolonged duration of MUAP, and incomplete interference pattern of the studied muscles among patients of both groups compared to the control, with significantly higher affection among the symptomatic group than the asymptomatic.

Table 5 shows the risk factors for developing myopathy and neuropathy among patients of the studied groups that were significantly higher in the symptomatic group, such as hospitalization (42.8% of

TABLE 3 Motor nerve conductive study (MNCS) and sensory nerve conductive study (SNCS) among subjects of the studied groups

MNCS, mean ± *SD*	G1 (symptomatic) (210)	G2 (asymptomatic) (190)	G3 (control) (30)	*P* value	SNCS, mean ± *SD*	G1	G2	G3 (control)	*P* value
Median nerve, DML	7.4 ± 1.5	4.9 ± 1.2	4.2 ± 2.2	P1 = .01 P2 = .05 P3 = .01	PSL	4.8 ± 2.5	3.9 ± 1.2	2.3 ± 0.2	P1 = .4 P2 = .01 P3 = .02
Amplitude	2.1 ± 0.7	3.5 ± 1.5	4.5 ± 1.3	P1 = .05 P2 = .05 P3 = .01	Amplitude	10 ± 3.8	15 ± 3.9	20 ± 5.9	P1 = .05 P2 = .03 P3 = .01
MCV	45 ± 3.1	47 ± 1.3	49 ± 2.5	P1 = .4 P2 = .01 P3 = .1	SCV	47 ± 2.9	48 ± 2.4	49 ± 1.4	P1 = .4 P2 = .2 P3 = .1
Ulnar nerve, DML	5.2 ± 2.6	3.3 ± 1.5	2.1 ± 1.2	P1 = .03 P2 = .01 P3 = .1	PSL	5.2 ± 2.8	3.1 ± 1.4	2.1 ± 1.1	P1 = .04 P2 = .02 P3 = .1
Amplitude	3.9 ± 3.7	4 ± 1.8	6 ± 2.5	P1 = .9 P2 = .01 P3 = .05	Amplitude	11 ± 3.6	15 ± 3.9	17 ± 4.8	P1 = .04 P2 = .01 P3 = .03
MCV	42 ± 2.8	48 ± 1.1	49 ± 2.1	P1 = .03 P2 = .01 P3 = .5	SCV	46 ± 3.4	47 ± 2.5	49 ± 3.2	P1 = .9 P2 = .5 P3 = .2
Radial nerve, DML	3.4 ± 1.5	2.7 ± 1.5	1.9 ± 1.8	P1 = .8 P2 = .01 P3 = .4	PSL	5.2 ± 2.4	3.2 ± 1.6	2.2 ± 2.1	P1 = .03 P2 = .01 P3 = .2
Amplitude	2.3 ± 2.5	3.9 ± 2.4	5.9 ± 3.8	P1 = .6 P2 = .01 P3 = .02	Amplitude	11 ± 2.9	14 ± 3.3	16 ± 5.6	P1 = .6 P2 = .05 P3 = .3
Motor, MCV	47 ± 2.8	49.3 ± 2	50.2 ± 1.9	P1 = .6 P2 = .5 P3 = .8	SCV	45 ± 2.8	49.3 ± 2	50.2 ± 1.9	P1 = .05 P2 = .3 P3 = .7
Tibial nerve, DML	6.4 ± 2.4	3.9 ± 1.5	3.5 ± 1.3	P1 = .03 P2 = .01 P3 = .2	Sural nerve, PSL	6.5 ± 2.6	3.7 ± 1.6	3.3 ± 0.6	P1 = .04 P2 = .02 P3 = .6
Amplitude	3.5 ± 0.6	5 ± 3.4	7 ± 4.3	P1 = .4 P2 = .03 P3 = .01	Amplitude	4 ± 2.3	7.1 ± 2.4	7.5 ± 3.8	P1 = .05 P2 = .02 P3 = .1
MCV	40 ± 2.1	40 ± 2.9	41 ± 3.5	P1 = .4 P2 = .8 P3 = .9					

symptomatic patients were hospitalized vs 6.3% in the asymptomatic group), long-lasting respiratory symptoms (15.2% of symptomatic patients vs 2.6% of asymptomatic patients), severe respiratory symptoms (22.3% of symptomatic patients vs 3.7% of asymptomatic patients).

Univariate analysis of the suggested risk factors for post-COVID-19 neuromuscular affection included hospitalization (OR 9.7, 95% CI 1.5–23.8), long-lasting respiratory symptoms of more than 2 weeks (OR 8.9, 95% CI 2.7–10.6), the presence of severe respiratory symptoms during infection (OR 10.4, 95% CI 3.2–21.4), and multivariate regression analysis demonstrated that the presence of long-lasting, and severe respiratory symptoms were independent risk factors for the occurrence of post-COVID-19 neuromuscular complications (OR 6.2, 95% CI 1.7–9.5 and OR 7.5, 95% CI 1.1–12.4) as presented in Table 6.

5 | DISCUSSION

This article aims to evaluate the peripheral nerves and muscle involvement among patients (symptomatic and asymptomatic) post-COVID-19 infection, in addition to studying the prevalence rate and risk factors of their affection.

Several previous studies reported neuromuscular involvement among post-COVID-19 patients. Therefore, to the best of our knowledge, this is the first study conducted on a large sample of patients with variable durations after infection with COVID-19, and the first to document sensorimotor axonal and demyelinating neuropathy and myopathy even among asymptomatic post-COVID-19 patients, with clarification of the risk factors of developing neuromuscular affection among them.

The present study revealed that 56.3% of post-COVID-19 patients had neuromuscular affection among both symptomatic and asymptomatic patients. There was a significant difference among patients of both groups regarding serum CPK level, clinical signs, and electrophysiologic study findings of neuropathy and myopathy compared to the control (non-infected) group, with significantly higher differences among the symptomatic group.

We also reported multiple risk factors associated with neuromuscular affection among post-COVID-19 patients as regards hospitalization, severe, and long-lasting respiratory symptoms of more than 2 weeks.

The risk factors for developing myopathy and neuropathy among asymptomatic post-COVID-19 patients were significantly lower than that in the symptomatic group; accordingly, they had a lower prevalence of neuropathy and myopathy.

Our results are consistent with Bagnato,[20] who documented that 81% of post-COVID-19 patients had neuropathies, and myopathies with substantial weakness and functional impairment.

Moreover, Ftiha et al[12] also documented that 36.4% of patients in their study group had peripheral neuropathy after COVID-19 infection.

Similarly, Vanhorebeek[17] concluded that muscle involvement during COVID-19 infection was characterized by myalgia in 40% and muscle tiredness in 70% of patients.

TABLE 3 (Continued)

MNCS, mean ± *SD*	G1 (symptomatic) (210)	G2 (asymptomatic) (190)	G3 (control) (30)	*P* value	SNCS, mean ± *SD*	G1	G2	G3 (control)	*P* value
Peroneal nerve, DML	7.2 ± 1.7	4 ± 1.9	3 ± 1.8	P1 = .01 P2 = .05 P3 = .3					
Amplitude	1.9 ± 0.08	2.9 ± 2.5	4 ± 2.8	P1 = .7 P2 = .01 P3 = .02					
MCV	40 ± 2.4	41 ± 1.4	44 ± 2.3	P1 = .4 P2 = .02 P3 = .01					

Abbreviations: DML, distal motor latency; MCV, motor conductive velocity; PSL, peak sensory latency; SCV, sensory conductive velocity.

TABLE 4 Electromyography (EMG) among subjects of the study group

	At rest., presence of insertional activity, mean ± *SD*				At maximal activity, incomplete interference, pattern, mean ± *SD*			
	G1 (symp)	G2 (asymp)	G3 (control)		G1 (symp)	G2 (asymp)	G3 (control)	
	n = 210	*N* = 190	*N* = 30	*P* value	N = 210	N = 190	N = 30	*P* value
1st dorsal interosseous	69 ± 7.5	16 ± 3.2	0 ± 0	P1 = .001 P2 = .001 P3 = .001	57 ± 2.8	17.3 ± 2.8	0 ± 0	P1 = .001 P2 = .008 P3 = .007
Biceps brachii	57 ± 5.9	10 ± 3.3	0 ± 0	P1 = .002 P2 = .001 P3 = .02	49 ± 3.2	16 ± 1.5	0 ± 0	P1 = .001 P2 = .001 P3 = .007
Triceps brachii	49 ± 7.9	18 ± 1.4	0 ± 0	P1 = .003 P2 = .001 P3 = .01	52 ± 3.6	19 ± 1.8	0 ± 0	P1 = .001 P2 = .001 P3 = .005
Tibialis posterior	21 ± 6.8	17 ± 4.7	0 ± 0	P1 = .04 P2 = .008 P3 = .007	39 ± 3.4	18 ± 2.7	0 ± 0	P1 = .001 P2 = .001 P3 = .004
Gastrocnemius-soleus	37 ± 8.6	13 ± 9.6	0 ± 0	P1 = .001 P2 = .001 P3 = .00	33 ± 2.7	16 ± 3.7	0 ± 0	P1 = .002 P2 = .001 P3 = .007
Vastus lateralis	8 ± 5.8	16 ± 4.5	0 ± 0	P1 = .004 P2 = .001 P3 = .009	27 ± 5.2	17.7 ± 2.9	0 ± 0	P1 = .009 P2 = .004 P3 = .002
	Amplitude of MUAP (μV) among groups, mean ± *SD*				Duration of MUAP (μS) among groups, mean ± *SD*			
At minimal activity	G1	G2	G3	*P* value	G1	G2	G3	*P* value
1st dorsal interosseous	250 ± 75	520 ± 72	640 ± 67	P1 = .03 P2 = .01 P3 = .02	3.7 ± 2.8	7.3 ± 6.8	9.3 ± 5.7	P1 = .02 P2 = .01 P3 = .31
Biceps brachii	317 ± 57.9	610 ± 33	760 ± 99	P1 = .02 P2 = .01 P3 = .3	5.7 ± 3.2	6.2 ± 5.8	11.2 ± 5	P1 = .8 P2 = .03 P3 = .02
Triceps brachii	490 ± 77.9	720 ± 94	890 ± 120	P1 = .02 P2 = .01 P3 = .05	3.2 ± 3.6	7.9 ± 7.8	8.5 ± 4.9	P1 = .05 P2 = .03 P3 = .2
Tibialis posterior	219 ± 56.8	567 ± 170	794 ± 160	P1 = .04 P2 = .02 P3 = .03	2.9 ± 3.4	8.8 ± 4.7	10 ± 5.3	P1 = .02 P2 = .05 P3 = .1
Gastrocnemius-soleus	370 ± 87.6	537 ± 96	667 ± 88	P1 = .05 P2 = .01 P3 = .05	4.3 ± 2.7	6.7 ± 6.7	8.2 ± 4.9	P1 = .7 P2 = .05 P3 = .2
Vastus lateralis	658 ± 55.8	667 ± 125	789 ± 140	P1 = .7 P2 = .5 P3 = .3	7.5 ± 5.2	7.7 ± 4.9	9.1 ± 3.2	P1 = .6 P2 = .4 P3 = .7

Note: P1 = G1 vs G2, P2 = G1 vs G3, P3 = G2 vs G3, μV = microvolt, μS = microsec.
G1 symp = symptomatic group; G2 asymp = asymptomatic group; MUAP = motor unit action potential.

In the same way, Pinzon et al,[21] Guidon and Amato,[22] Li et al,[23] and Sanchez et al.[24] documented that about 19.2%, 33%, and 56% of COVID-19 infected patients had myalgia or muscle injury with elevated creatine kinase levels.

In accordance with our results, Faqihi et al,[25] Sejvar et al,[26] Jacobs et al,[27] Mehta et al,[28] Faqihi et al,[29] Paterson et al,[30] and Wu et al[31] also reported peripheral neuropathy in post-COVID-19 patients.

As all the mentioned studies agree with our results, we could suggested that neuromuscular involvement is a common complication post-COVID-19 infection, even in asymptomatic patients, and the different degrees of affection and functional impairment in different

TABLE 5 Risk factors to develop myopathy and neuropathy among patients of the studied groups

	G1 (symptomatic) N = 210	G2 (asymptomatic) N = 190	*P* value
Hospitalization	90 (42.8%)	12 (6.3%)	.001
Severe respiratory symptoms	47 (22.3%)	7 (3.7%)	.002
Long-lasting respiratory symptoms, more than 15 d	32 (15.2%)	5 (2.6%)	.004

TABLE 6 Univariate and multivariate regression analysis for the association between variables and the presence of neuropathy and myopathy among COVID-19 patients groups

	Univariate analysis			Multivariate analysis		
	Odds ratio	95% CI	*P* value	Odds ratio	95% CI	*p* value
Hospitalization	9.7	1.5–23.8	.008	1.15	0.91–7.41	.12
Old age, long-lasting respiratory symptoms, 15–40 d	8.9	2.7–10.6	.01	6.2	1.7–9.5	.02
Severe respiratory symptoms	10.4	3.2–21.4	.02	7.5	1.1–12.4	.005

studies can be explained according to the severity of COVID-19 infection and the presence of other risk factors in those patients.

In another way, Bureau et al[32] observed the low incidence of peripheral neuropathy post-COVID-19 infection in their study group. That could be explained by the fact their study group patients had a mild COVID-19 infection as they fully recovered after a short period and they were not hospitalized.

In agreement with our results, Frithiof et al[33] documented that prolonged hospitalization and severe respiratory distress symptoms are independent predictors closely related to neuromuscular complications after infection with COVID-19. They reported that 79% of COVID-19 infected patients had neuromuscular affection.

Our results revealed the presence of neuromuscular symptoms among patients for a relatively long period post-COVID-19 infection, as the median assessment time for patients in the 2 study groups was 92, and 87 days post-infection.

In accordance with our results, a study performed by Elkind et al reported a long time delay between COVID-19 viral infection onset and the appearance of neuromuscular complications.[34]

In another way, Zhao et al[35] showed that neurological disorders, such as in the cases of Guillain-Barré syndrome, are related to the early symptoms of COVID-19 infection, which arises as a para-infectious rather than a post-infectious complication in some patients.

Accordingly, the early association between COVID-19 infection and Guillain-Barré syndrome could be explained since this rare disorder is usually associated with infection or soon after infection.

Therefore, our study could not be certain about the time of appearance of neuromuscular complications, either post-infection or para-infection, as these complications were presented during the time of the study among symptomatic and asymptomatic patients of the study groups.

6 | CONCLUSION

The present study implies that muscle involvement and peripheral nerve affection are common problems even among asymptomatic post-COVID-19 patients, especially in the presence of any risk factors, such as a history of long hospitalization, severe, and long-lasting respiratory symptoms. Thus, in order to improve management and prevent a worsening of the patients' outcomes, we must be aware of the presence of any neurologic symptoms in patients after COVID-19 infection.

AKNOWELDGEMENT

We would like to thank all staff members and workers of physical medicine, rheumatology and rehabilitation for their help and support.

CONFLICT OF INTERESTS

The authors declare they have no conflicts of interest.

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How to cite this article: Saif DS, Ibrahem RA, Eltabl MA. Prevalence of peripheral neuropathy and myopathy in patients post-COVID-19 infection. *Int J Rheum Dis*. 2022;00:1-8. doi: 10.1111/1756-185X.14409