IOSR Journal of Nursing and Health Science (IOSR-JNHS)
e-ISSN: 2320–1959.p- ISSN: 2320–1940 Volume 12, Issue 2 Ser.V (Mar. – Apr. 2023), PP 57-64
www.iosrjournals.org

Electrophysiological Evaluation of Post COVID Neuropathy

SK Mahbub Alam,
MD, Associate Professor, Department of Neurology, Bangabandhu Sheikh Mujib Medical University, Dhaka-1000
Maftahul Jannat,
MD,Medical Officer, Department of Neurology, Bangabandhu Sheikh Mujib Medical University, Dhaka- 1000
Mehedi Hasan,
MD, Medical Officer, Department of Neurology, Bangabandhu Sheikh Mujib Medical University, Dhaka- 1000
Sahariar Hossain Siddik,
MBBS, Resident Neurology,Department of Neurology, Bangabandhu Sheikh Mujib Medical University, Dhaka-1000

Abstract:
Background: *The COVID-19 pandemic is caused by severe acute respiratory syndrome by corona virus-2 (SARS-COV-2). Although the predominant clinical presentation is with respiratory disease, neurological manifestation is being recognized increasingly. The aim of the study was to evaluate the nerve conduction study findings of COVID-19 patients with neuropathy. Electrophysiological evaluation of neuropathy can be used as a diagnostic as well as prognostic marker of COVID neuropathy so that patient can undergo an early therapeutic trial.*
Objective: *The objective of this study was to seeelectrophysiological findings of post COVID neuropathy.*
Methods:*Thiscross sectional observational study wasundertaken in the department of Neurology, BSMMU, Dhaka, Bangladesh. Non-randomized convenient purposive sampling was done to selected COVID neuropathy patients. Total 44 patients were selected purposively. Study subjects was taken from confirmed cases of COVID-19 infection with symptoms of neuropathy admitted at COVID unit and visited at COVID follow up Clinic of BSMMU, Dhaka, after meeting inclusion and exclusion criteria during October 2020 to March 2022. Detailed history, physical examination findings, previous medical records, investigation findings were recorded in datasheet. Electrophysiological evaluation of neuropathy was done for each patient.*
Results: *Out of 44 patients most of the pathological form of neuropathy were demyelinating (44.4%) followed by axonal (40.7%) and rest were mixed (14.8%). Among our patientswe found polyneuropathy 14 (51.9%), entrapment neuropathy 7(25.9%), mononeuropathy 3(11.1%) and multiple mononeuropathy were 3 (11.1%). We also found 4 (14.8 %) patients had pure motor neuropathy, 6 (22.2%) had pure sensory neuropathy and rest 17 (63%) had mixed sensory-motor neuropathy.*
Conclusion: *Electrophysiologically we found various type of neuropathies among our patients. Though most of the patients had mild neuropathy but moderate to seveve neuropathy in long standing hospitalized patients also reported. So electrophysiological examination could be done for symptomatic neuropathy in Covid-19 affected patient.*
Keywords:*COVID-19, Electrophysiology,Neuropathy, Bangladesh*

Date of Submission: 14-04-2023 Date of Acceptance: 27-04-2023

I. Introduction:

The Novel Coronavirus (COVID-19) outbreak originated fromthe Wuhan city, China in December 2019.[1] Since then the virus had spread to various countries across the globe including Bangladesh due to its ominously high rate of transmission. WHO had declared the illness as pandemic on March 11, 2020.[2]The identified virus is named as SARS-CoV-2 and the illness caused by the virus is named corona virus disease 2019 (COVID-19).

The primary manifestation of COVID-19 infection is pneumonia. Although the most common and important presentation is with respiratory disease, reports of neurological features are increasing involving both central nervous system and peripheral nervous system.

The neurological manifestations and complications can be divided into central and peripheral nervous system manifestation. Central nervous system manifestations are dizziness, headache, acute cerebrovascular disease, impaired consciousness, transverse myelitis, acute hemorrhagic necrotizing encephalopathy, encephalitis, epilepsy and ataxia. Among peripheral nervous system manifestationshypogeusia, hyposmia, neuralgia, GuillainBarre Syndrome and skeletal muscle injury were found in different literature .[3]

The involvementof the nervous system can be due to a direct actionof these viruses on the nervous tissue and/or to an indirectaction through the activation of immune-mediatedmechanisms. While the first action can be verified duringthe acute phase of the disease, the second can be apparentonly after days, weeks, or even months following the acutephase. Coronaviruses can invade thenervous tissues involving immune-functioning macrophages,microglia, or astrocytes[4] and cause nerve damagethrough direct infection pathways (circulatory andneuronal), hypoxia, immune injury, attack to ACE2 enzymes,and other mechanisms .[5]

Among the peripheral nervous system manifestations anosmia (a complete or partial loss of smell sensation) and ageusia (loss of taste sensation) are the most frequent neurological manifestations of COVID-19. Anosmia and ageusia are common even in mild to moderate cases.[6] The smell sensation is more severely affected than the taste sensation. In a French study, Lechien and co-workers reported 86% and 88% of mild to moderate COVID -19 patients reported anosmia and ageusia respectively.[7] Anosmia was the first manifestation of Covid-19 in many patients. The possible mechanism is the SARS-CoV-2 virus utilizes angiotensin-converting enzyme 2 receptors, presents in the olfactory epithelium, to enter into the neuronal cells, and then via the olfactory nerve, it spreads to the olfactory bulb.[8]

Guillain-Barré syndrome is a frequently encountered neurological complication of COVID-19.The first patient of Guillain-Barré syndrome in COVID-19 was described by Zhao and co-workers.[9]After this, 18 more patients of Guillain-Barré syndrome in COVID-19, have been described.Electrophysiological studies were done in 12 patients and were consistent with demyelinating disease in eight and axonal neuropathy in four patients.[10,11]An albumino-cytologic dissociation in CSF and positive GD1b-IgG antibodies indicated an inflammatory pathology.[12]

Some authors found a higher neurological involvement in severe stage of COVID-19. Helms et al found significantly higher neurological complications in severe cases (84%) in France.[13] As the disease burden become increasing day by day at Bangladesh the present study was intended to see whether there is any positive electrophysiological findings and type of electrophysiological findings among symptomatic post COVID neuropathic patients. It will also help to develop strategies for early detection and provision of therapy for neuropathy.

II. Methods:

This Cross sectional observational study was conducted in COVID unit and COVID follow up clinic of BSMMU, Dhaka, Bangladesh,from October 2020 to March 2022 after obtaining ethical clearance IRB board of BSMMU.All confirmed cases of COVID 19 infection with symptoms of neuropathy admitted at COVID unit and visited atCOVID follow up Clinic of BSMMU weretaken as study population after taking informed written consent. Total 44 patients were selected purposively. Patients with known prior neuropathy like Guillain Barre Syndrome, CIDP, diabetic neuropathy, uremic neuropathy etc. were excluded.

At first all COVID -19patients who were admitted at COVID Unit or returned to the COVID follow up clinic of BSMMU were interrogated for any neuropathic symptoms. Those who had neuropathy clinically from the onset and those who developed neuropathy after recovery were selected for the study.Detailed clinical history and physical examination were done and patient were sent for Nerve conduction study. Nerve conduction study were done by Nihon Kohden machine in Neurology department of BSMMU. Normal referrence values for nerve conduction study was taken from text book of Electromyography and neuromuscular disorders by David C. Preston and Barbara E. Shapiro.[14]Necessary medical records were evaluated.The medical records demographic, neurological & other clinical and laboratory investigations and findings of nerve conduction study of the patients were recorded in the preformed data sheet.

At the end of data collection, all the data were rechecked, coded and entered in standard statistical software using SPSS.Qualitative data were expressed as frequency and percentage.The P value <0.05 will be considered statistically significant.Comparisons between positive electrophysiological findings of post COVID neuropathy and negative electrophysiological findings of post COVID neuropathy regarding demographic and laboratory characteristics were performed using Student's t-test and chi-square tests.

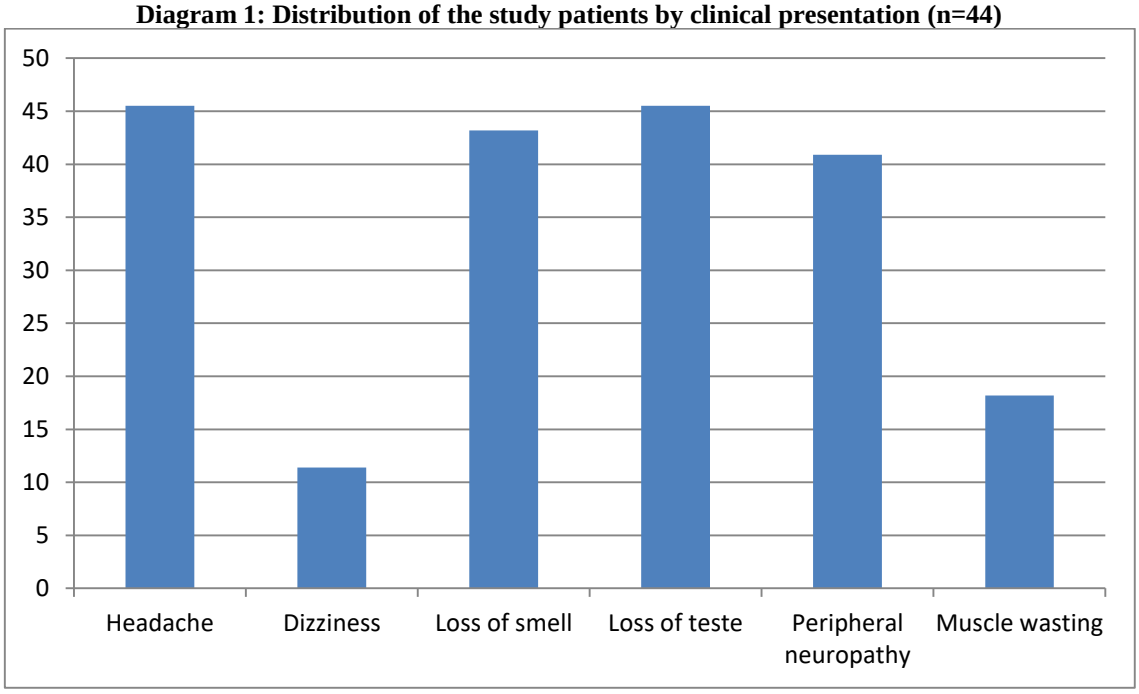
III. Results

It was observed that mean age of the patients was 48.5±17.9 years with a range of 11 – 90 years. In this study majority of the patients were male and male: female ratio was 1.8:1 Majority of the patients were service holder (47.7 %), among females majority were housewife 40.9%(Table -1).

Table-1: Sociodemographic characteristics of the study patients (n=44)

Variables	Number of patients	Percentage (%)
Age group (years)		
<40	15	34.1
41-60	17	38.6
>60	12	27.3
Mean±SD Range (min-max)	48.5±17.9 (11 – 90) years	
Sex		
Male	28	63.6
Female	16	36.4
Male: female ratio	1.8:1	
Occupation		
Service holder	21	47.7
Business	3	6.8
Housewife	18	40.9
Others	2	4.5

Diagram 1: Distribution of the study patients by clinical presentation (n=44)

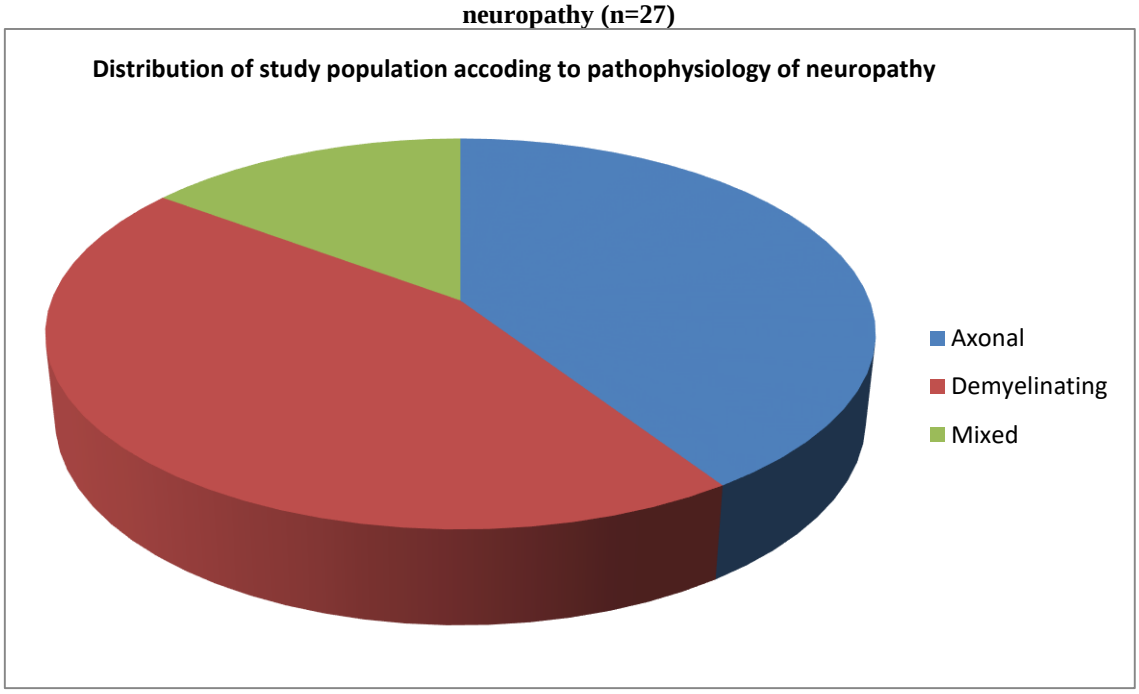


It was found that common presentations were headache (45.5%),loss of taste (45.5%), loss of smell (43.2%) followed by peripheral neuropathy (40.9%). some patients had muscle wasting (18.2%) and dizziness (11.4%) as shown in diagram 1.

In this study most of the neuropathies were mild 26 (59.1%), followed by moderate 12 (27.3%).Rest of the neuropathies 6 (13.6%) were severe.

Among all patients 17(38.6%) patients had normal electrophysiological findings and rest 27(61.4%) were abnormal.

Diagram-2: Pie chart showing distribution of the study patients according to pathophysiology of neuropathy (n=27)



In this study most of the pathological form of neuropathy were demyelinating (44.4%) followed by axonal (40.7%) and rest were mixed (14.8%) as shown in Pie chart.

Table-2: Distribution of patients according to motor nerve distal latencies.

Distal latency (upper limbs)	**Number of patients**	**Percentage (%)**	**Distal latency (lower limbs)**	**Number of patients**	**Percentage (%)**
Median, left			**Peroneal, left**		
Normal (≤4.4)	24	54.5	Normal (≤6.5)	24	54.5
Prolonged (>4.4)	6	13.6	Prolonged (>6.5)	3	6.8
Absent	14	31.8	Absent	17	38.6
Median, right			**Peroneal, right**		
Normal (≤4.4)	27	61.4	Normal (≤6.5)	21	47.7
Prolonged (>4.4)	6	13.6	Prolonged (>6.5)	1	2.3
Absent	11	25.0	Absent	22	50.0
Ulnar, left			**Tibial ,left**		
Normal (≤3.3)	27	61.4	Normal (≤6.7)	2	4.5
Prolonged (>3.3)	3	6.8	Prolonged (>6.7)	25	56.8
Absent	14	31.8	Absent	17	38.6
Ulnar, right			**Tibial ,right**		
Normal (≤3.3)	3	6.8	Normal (≤6.7)	1	2.3
Prolonged (>3.3)	30	68.2	Prolonged (>6.7)	20	45.5
Absent	11	25.0	Absent	23	52.3

Table-3: Distribution of patients according to motor nerve conduction velocities.

Conduction velocity (upper limbs)	Number of patients	Percentage (%)	Conduction velocity (lower limbs)	Number of patients	Percentage (%)
Median, Left			**Peroneal, left**		
Normal (≥49.0)	28	63.6	Normal(≥44)	15	34.1
Decreased (<49.0)	2	4.5	Decresed(<44)	12	27.3
Absent	14	31.8	Absent	17	38.6
Median,right			**Peroneal, right**		
Normal (≥49.0)	27	61.4	Normal(≥44)	14	31.8
Decreased (<49.0)	6	13.6	Decresed(<44)	8	18.2
Absent	11	25.0	Absent	22	50.0
Ulnar, left			**Tibial ,left**		
Normal (≥49.0)	29	65.9	Normal(≥41)	17	38.6
Decreased (<49.0)	1	2.3	Decresed(<41)	10	22.7
Absent	14	31.8	Absent	17	38.6
Ulnar, right			**Tibial ,right**		
Normal (≥49.0)	30	68.2	Normal(≥41)	13	29.5
Decreased (<49.0)	3	6.8	Decresed(<41)	8	18.2
Absent	11	25.0	Absent	23	52.3

Table-4: Distribution of patients according to motor nerve amplitude (CMAP)

CMAPs (upper limbs)	Number of patients	Percentage (%)	CMAPs (lower limbs)	Number of patients	Percentage (%)
Median, left			**Peroneal, left**		
Normal (≥4.0)	27	61.4	Normal≥2	25	56.8
Decreased (<4.0)	3	6.8	Decreased <2	2	4.5
Absent	14	31.8	Absent	17	38.6
Median, right			**Peroneal, right**		
Normal (≥4.0)	1	2.3	Normal≥2	2	4.5
Decreased (<4.0)	32	72.7	Decreased <2	20	45.5
Absent	11	25.0	Absent	22	50.0
Ulnar, left			**Tibial ,left**		
Normal (≥6.0)	25	56.8	Normal≥4	27	61.4
Decreased (<6.0)	5	11.4	Decreased <4	0	0.0
Absent	14	31.8	Absent	17	38.6
Ulnar, right			**Tibial ,right**		
Normal (≥6.0)	29	65.9	Normal≥4	21	47.7
Decreased (<6.0)	4	9.1	Decreased <4	0	0.0
Absent	11	25.0	Absent	23	52.3

Table-5: Distribution of the study patients by F-wave study of upper limbs

F-wave study (left)	Number of patients	Percentage (%)	F-wave study (right)	Number of patients	Percentage (%)
Normal≤31	26	59.1	Normal≤31	24	54.5
Prologed>31	1	2.3	Prologed>31	8	18.2
Absent	17	38.6	Absent	12	27.3

Table-6: Distribution of patients according to sensory nerve distal latencies

Distal latency	Number of patients	Percentage (%)	Distal latency	Number of patients	Percentage (%)
Median, left			**Median, right**		
Normal≤3.5	30	68.2	Normal≤3.5	28	63.6
Prologed>3.5	3	6.8	Prologed>3.5	6	13.6
Absent	11	25.0	Absent	10	22.7
Ulnar, left			**Ulnar, right**		
Normal≤3.1	31	70.5	Normal≤3.1	34	77.3
Prolonged>3.1	2	4.5	Prolonged>3.1	0	0.0
Absent	11	25.0	Absent	10	22.7
Sural (left)			**Sural (right)**		
Normal≤4.4	30	68.2	Normal≤4.4	22	50.0
Prolonged>4.4	0	0.0	Prolonged>4.4	1	2.3
Absent	14	31.8	Absent	21	47.7

Table-7: Distribution of patients according to sensory nerve conduction velocity

Conduction velocity	Number of patients	Percentage (%)	Conduction velocity	Number of patients	Percentage (%)
Median, left			**Median, right**		
Normal≥50	22	50.0	Normal≥50	18	40.9
Reduced<50	11	25.0	Reduced<50	16	36.4
Absent	11	25.0	Absent	10	22.7
Ulnar, left			**Ulnar, right**		
Normal≥50	19	43.2	Normal≥50	27	61.4
Reduced<50	14	31.8	Reduced<50	7	15.9
Absent	11	25.0	Absent	10	22.7
Sural (left)			**Sural (right)**		
Normal≥40	27	61.4	Normal≥40	22	50.0
Reduced<40	3	6.8	Reduced<40	1	2.3
Absent	14	31.8	Absent	21	47.7

Table-8: Distribution of patients according to sensory nerve amplitude (SNAP)

SNAP	Number of patients	Percentage (%)	SNAP	Number of patients	Percentage (%)
Median, left			**Median, right**		
Normal≥20	26	59.1	Normal≥20	23	52.3
Reduced<20	7	15.9	Reduced<20	11	25.0
Absent	11	25.0	Absent	10	22.7
Ulnar, left			**Ulnar, right**		
Normal≥17	27	61.4	Normal≥17	28	63.6
Reduced<17	6	13.6	Reduced<17	6	13.6
Absent	11	25.0	Absent	10	22.7
Sural (left)			**Sural (right)**		
Normal≥6	28	63.6	Normal≥6	22	50.0
Reduced<6	2	4.5	Reduced<6	1	2.3
Absent	14	31.8	Absent	21	47.7

In this study we found most of the patients 14 (51.9%) had polyneuropathy follwed by entrapment neuropathy 7(25.9%), mononeuropathy 3(11.1%) and multiple mononeuropathy 3 (11.1%).
In our study 4 (14.8 %) patients had pure motor neuropathy, 6 (22.2%) had pure sensory neuropathy and rest 17 (63%) had mixed sensory-motor neuropathy.

Table-09: Distribution of the study patients according to clinical and electrophysiological diagnosis

According to clinical diagnosis		
GBS		
AMAN	1	3.7
AIDP	1	3.7
Carpal tunnel syndrome		
Right	2	7.4
Left	2	7.4
Both	3	11.1
Critical illness neuropathy	2	7.4
Sensory neuropathy	6	22.2
Polyneuropathy (sensory, motor)	11	40.7

IV. Discussion

This was a cross sectional descriptivestudy carried out on patient who admitted in COVID unit and came in COVID follow up clinic of BSMMU, Dhaka, Bangladesh.In this study majority of the respondents 17 (38.6%) were belonging to age group 41-60 years. The mean age of the patients was 48.5±17.9 years with a range of 11 – 90 years. In this study majority of the patients were male and male: female ratio was 1.8:1. In another study done by Oaklander. A. L et al reported that participant'smean ages were averaged 43.3 ± 3.3 years, and among them 68.8% were female.[15]Our study has almost similar mean age but we found higher percentage of male patients 63.6%(28). Among 44 patients it was found that common presentations were headache (45.5%),loss of taste (45.5%), loss of smell (43.2%), followed by peripheral neuropathy (40.9%). Some patients had muscle wasting (18.2%) and dizziness (11.4%). In a large Iranian cohort of 10,069 patients by employing an online questionnaire-based surveyBagheri et al reported anosmia and hyposmia (48.23%), decreased taste sensation (83.38%) and headaches (48.6%) of the respondents.[16] In another study done by Ftiha et al documented that 36.4% of patients of their study group had peripheral neuropathy after COVID-19 infection.[17] In our study we also have almost similar findings of peripheral neuropathy (40.9%).In our study

11.4% patients had dizziness which has similarity with previous study done by Imran Ahmad and Farooq Azam Rathore.[3]They found 16.8% patients had dizziness. In this study we found most of the neuropathies were mild (59.1%), followed by moderate (27.3%) and rest of the neuropathies (13.6%) were severe according toNeuropathy Disability Score (NDS).[18]In our study 61.4% patients have abnormal electrophysiological findings. In a previous study done by Yan at all reported that 56.3% of post-COVID-19 patients had neuromuscular affection.[19]In our electrophysiological study we found 12(44.4%) patients had demyelinating neuropathy, 11(40.7%) had axonal neuropathy and mixed neuropathy in 4(14.80%) of cases. In previous electrophysiological studies were done in 12 patients of GBS by Ellul MA et al. were consistent with demyelinating disease in 8 and axonal neuropathy in 4 patients.[10] In our study among 2 GBS patients we found 1 axonal and 1 demyelinating neuropathy. Carpal Tunnel Syndrome was found in 7 patients, among them 4 patients had unilateral and 3 patients had bilateral involvement. In one previous study Lica Roncati et al found that there is a causal link of Carpal Tunnel Syndrome (CTS) with COVID -19 infection.[20] Though we don't know whether they have CTS before affecting COVID -19 infection.We found 2 patients suffered from critical illness neuropathy who needed long term ICU care. Out of 44 patients 6 were suffered from pure sensory neuropathy, 4 had pure motor and 11 had sensory-motor polyneuropathy.

V. Conclusion

Though COVID-19 primarily affects the respiratory and cardiovascular system, neurological involvement is not very uncommon. Some patients suffered from neurological complications without severe illness. Electrophysiologically we found various type of neuropathies among our patients. We found mild neuropathy in most of the cases but moderate to seveve neuropathy in long standing hospitalized patients. So electrophysiological examination could be done for symptomatic neuropathy in Covid 19 affected patients.

Disclosure

Author Contributions: All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Ethical issues: All patients gave informed written consent and study was approved by Institutional Review Board of Bangabandhu Sheikh Mujib Medical University.

Disclosure: The authors reports no disclosures.

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

Conflict of interest: The authors declare they have no conflicts of interest.

Source of funding: The authors received no specific funding for this work.

References:

[1]. Zhu N., Dingyu Z., Wenling W. et al. A novel coronavirus from patients with pneumonia in China, 2019. N Engl J Med 2020; 382(8):727–33.https://doi.org/10.1056/NEJMoa2001017

[2]. WHO Director-General's opening remarks at the media briefing on COVID-19 – 11 March 2020. Available at https://www.who.int/director-general/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-covid-19---11-march-2020.

[3]. Ahmad I, Rathore FA. Neurological manifestations and complications of COVID-19: A literature review. Journal of clinical neuroscience. 2020 1; 77:8-12.https://doi.org/10.1016/j.jocn.2020.05.017

[4]. Al-Obaidi MJ, Bahadoran A, Wang SM, Manikam R, Raju CS, Sekaran SD. Disruption of the blood brain barrier is vital property of neurotropic viral infection of the central nervous system. Acta virologica. 2018 1;62(1):16-27 https://doi.org/10.4149/av_2018_102

[5]. Wu Y, Xu X, Chen Z. et al. Nervous system involvement after infection with COVID-19 and other coronaviruses. Brain, behavior, and immunity. 2020:1; 87:18-22.https://doi.org/10.1016/j.bbi.2020.03.031

[6]. Moein ST, Hashemian SM, Mansourafshar B, Khorram-Tousi A, Tabarsi P, Doty RL. Smell dysfunction: a biomarker for COVID-19. InInternational forum of allergy & rhinology 2020 Vol. 10, No. 8, pp. 944-950. doi: https://doi.org/10.1002/alr.22587

[7]. Lechien JR, Chiesa-Estomba CM, De Siati DR et al. Olfactory and gustatory dysfunctions as a clinical presentation of mild-to-moderate forms of the coronavirus disease (COVID-19): a multicenter European study. European Archives of Oto-rhino-laryngology. 2020;277(8):2251-61. doi: https://doi.org/10.1007/s00405-020-05965-1.

[8]. Beltrán-Corbellini Á, Chico-García JL, Martínez-Poles J et al. Acute-onset smell and taste disorders in the context of COVID-19: a pilot multicentre polymerase chain reaction-based case–control study. European journal of neurology. 2020;27(9):1738-41. doi: https://doi.org/10.1111/ene.14273.

[9]. Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barré syndrome associated with SARS-CoV-2 infection: causality or coincidence?. The Lancet Neurology. 2020 1;19(5):383-4.https://doi.org/10.1016/s1474-4422(20)30109-5

[10]. Ellul MA, Benjamin L, Singh B et al. Neurological associations of COVID-19. The Lancet Neurology. 2020 1;19(9):767-83..doi. https://doi.org/10.1016/s1474-4422(20)30221-0

[11]. Virani A, Rabold E, Hanson T et al. Guillain-Barré syndrome associated with SARS-CoV-2 infection. IDCases. 2020. https://doi.org/10.1016/j.idcr.2020.e00771

[12]. Dinkin M, Gao V, Kahan J et al. COVID-19 presenting with ophthalmoparesis from cranial nerve palsy. Neurology. 2020;4; 28.https://doi.org/10.1212/wnl.0000000000009700

[13]. Helms J, Kremer S, Merdji H et al. Neurologic features in severe SARS-CoV-2 infection. New England Journal of Medicine. 2020 ;6; 4;382(23):2268-70. https://doi.org/10.1056/nejmc2008597
[14]. Preston DC, Shapiro BE. Electromyography and neuromuscular disorders e-book: clinical-electrophysiologic-ultrasound correlations. Elsevier Health Sciences; 2020 ;3 ;20.https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2323522/pdf/mjm0902p173.pdf
[15]. Oaklander AL, Mills AJ, Kelley M et al. Peripheral Neuropathy Evaluations of Patients With Prolonged Long COVID. Neurol Neuroimmunol Neuroinflamm. 2022 1;9(3):e1146. doi: 10.1212/NXI.0000000000001146.
[16]. Bagheri SH, Asghari A, Farhadi M et al. Coincidence of COVID-19 epidemic and olfactory dysfunction outbreak in Iran. Medical journal of the Islamic Republic of Iran. 2020; 34:62. https://doi.org/10.34171/mjiri.34.62
[17]. Ftiha F, Shalom M, Jradeh H. Neurological symptoms due to Coronavirus disease 2019. Neurology international. 2020 29;12(1):8639.
https://doi.org/10.4081%2Fni.2020.8639
[18]. Afifi L, Abdelalim AM, Ashour AS, Al-Athwari A. Correlation between clinical neuropathy scores and nerve conduction studies in patients with diabetic peripheral neuropathy. The Egyptian Journal of Neurology, Psychiatry and Neurosurgery. 2016 1;53(4): 248. DOI: 10.4103/1110-1083.202386
[19]. Yan CH, Faraji F, Prajapati DP, Boone CE, DeConde AS. International Forum of Allergy & Rhinology.Int Forum Allergy Rhinol:2020 ;10(7):806-813.
https://doi.org/10.1002/alr.22579.
[20]. Roncati L, Gianotti G, Gravina D et al. Cubital or tarsal tunnel syndrome after SARS-CoV-2 infection: A causal link? Medical Hypotheses. 2021 1; 153:110638.https://doi.org/10.1016%2Fj.mehy.2021.110638

SK Mahbub Alam. et.al. "Electrophysiological Evaluation of Post COVID Neuropathy." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)* 22(4), 2023, pp. 57-64.