Changes in the Nerve Conduction of the Paretic Limb in Post-Stroke Patients

Mohammed Moayad Mohammed[*], *Farqad Bader Hamdan*[**], *Aqeel Kareem Hatim*[***]

ABSTRACT:

BACKGROUND:

Post-stroke motor sequelae can lead to an abnormal limb posture and decreased function that can ultimately increase the susceptibility of the peripheral nerves in that limb to compression, particularly within the early stages of stroke.

OBJECTIVE:

To study nerve conduction in paretic and non-paretic extremity in patients with the first ever-one stroke and to relate these parameters with MRC (Medical Research Council) scale.

METHODS:

Twenty-three patients aged 31-62 years and duration of illness of 3 and 6 months were studied. Thirteen had right hemiparesis and 10 with left hemiparesis. MRC scale was ≥3 in 17 and ≤2 in 6 patients.

RESULTS:

The median and peroneal distal motor latencies (DML) were prolonged and the peroneal compound muscle action potential (CMAP) amplitude was reduced in the paretic as compared to the non-paretic side. The combined sensory index and the lumbrical/interosseous muscles comparison methods revealed significant differences between the paretic as compared to the non-paretic side.

Median distal sensory latency (DSL), DML, motor conduction velocity (MCV), and peroneal CMAP amplitude of the paretic limb were significantly different between those with ≥3 and those with ≤2 MRC scale. In the non-paretic upper limbs, the DSL and DML were significantly prolonged in those with ≤2 MRC when compared to those with ≥3 MRC.

CONCLUSION:

Post-stroke entrapment neuropathy may develop, along with axonal neuropathy symptoms in patients with severe paresis. The more severely affected limbs, the more severe electrophysiologic changes.

KEYWORDS: Stroke, Nerve conduction study, MRC scale.

INTRODUCTION:

Stroke is typically a disease of the descending motor tracts results in the upper motor neuron (UMN) syndrome which might consequently resulted in the affection of peripheral nerves; the lower motor neurons (LMN) that supplies the skeletal muscles [1,2]. Following the loss of central activation and development of spasticity, LMNs may become functionally depressed or may even undergo 'transsynaptic degeneration' causing muscle fibers deprived of their nerve supply [1,3].

The denervated muscle fiber undergoes atrophy and fibrous tissue and fat infiltration, a finding frequently reported in paralyzed muscles following stroke [4]. The disrupted muscle architecture rises spastic muscle echo intensity [5], which relates to the muscle thickness and compound motor action potential (CMAP) amplitude in those with longstanding stroke [6]. It has been shown that post-stroke muscle structural changes are not restricted to the paretic side [7].

In post-stroke patients, the symptoms of the affected side could be ascribed to many causes, like central post-stroke pain, complex regional pain syndrome type, pain secondary to spasticity, hemiplegic shoulder pain, and peripheral neuropathy [8].

Patients with stroke are more prone to compression neuropathy or insults to the neuronal plexuses as a result of malpositioning, traction, and using an assistive device or sustained pressure [9].

[*]Ibn Sena Teaching Hospital, Nineveh Health Directorate, Nineveh, Iraq.
[**]Department of Physiology, College of Medicine, Al-Nahrain University, Baghdad, Iraq.
[***]Department of Medicine, College of Medicine, Baghdad University, Baghdad, Iraq.

Entrapment neuropathies in stroke patients may worsen the clinical status and may be easily overlooked because of the clinical condition of the patients [10,11].

It is so common to encounter median nerve entrapment at the wrist, ulnar nerve entrapment at the elbow [12,13], and peroneal entrapment nerve neuropathy across fibular head [14], especially in the hemiparetic extremities within the acute stage of the disease but with no conduction abnormalities on the affected side [15].

On the contrary to the aforementioned studies, other denoted all electrophysiological indices of the median, ulnar, and peroneal nerves were significantly more abnormal on the nonparetic side than on the hemiparetic side or in controls [16,17].

In recent study dealt with the CMAP and motor evoked potentials, CMAP area under the curve, and the amplitudes and area under the curve of the tibialis anterior muscle were generally lower on the paretic side compared to the non-paretic side [18].

The goals of our study are to look for electrophysiologic abnormalities (if any) in the paretic extremities in patients with first-ever stroke and to define if these abnormalities are related to the severity of affection.

PATIENTS AND METHODS:

A cross-sectional study was conducted at the neurophysiology unit at Baghdad Teaching Hospital-Medical City, Baghdad for the period from Feb. 2020 till Sep. 2020. The study was approved by the Iraqi Board for Medical Specialization (order no. 931: date: 1/3/2020. Consent for participation from all subjects was ensured.

Twenty-three patients (7 females and 16 males) with an age range between 31 and 62 years (49.09±8.85 years) were referred from the neurology department with a diagnosis of the first-ever stroke. The disease duration was between 3 and 6 months (4.74±0.96 months). Patients with diabetes mellitus or a history of entrapment neuropathies, peripheral neuropathies, radiculopathies, or any other neuromuscular disease were excluded.

The patients were referred from senior neurologists after taking a medical history from the patients including the age, past medical history, and duration of the illness. Neurological examination [13 (56.5%) patients had right hemiparesis and 10 (43.5%) had left hemiparesis] was done for each patient including the examination of the sensory system, deep tendon reflexes, and plantar reflex as well as performing Phalen maneuver and testing each patient for Tinel sign at the wrist.

Grading the motor weakness was done according to the Medical Research Council (MRC) scale and accordingly, the patients were divided into two groups, those with a score of severe weakness (< 36 or ≤ 2) and those with a score of clinical weakness (< 48 or ≥ 3). Seventeen patients (74%) out of total had an MRC scale of ≥ 3 and only six (26%) with ≤ 2 [19,20].

Electrophysiological assessments

All the electrodiagnostic examinations were performed between 8:00 AM and 3:00 PM and the room temperature was monitored between (25^{o}C-28^{o}C) during the test procedures and the skin temperature between (32^{o}C-34^{o}C) was ensured using a skin thermometer.

The neurophysiological tests were done using Micromed EMG machine (model 1715, Italy) according to the methods of Preston and Shapiro [21]. Median distal sensory latency (DSL), sensory nerve action potential (SNAP) amplitude, and conduction velocity (CV) were studied by the antidromic method. Also, median and peroneal distal motor latency (DML), CV, and the CMAP amplitude and duration nerves were studied. Motor parameters were studied from abductor pollicis brevis and extensor digitorum brevis muscles, respectively.

The comparison of median and ulnar mixed palm-to-wrist DSLs, median lumbrical and ulnar interossei DMLs, median and ulnar DSLs from digit IV, and median and radial DSL from digit was also studied [21].

Statistical analysis

The SPSS statistical software version 25 (IBM corporation, USA) was used for all statistical methods. Test of normality (normal distribution) of the continuous data was done using Shapiro Wilk test. Paired t-test was applied for comparison between the paretic and nonparetic sides of the same patient. Student t-test (for normally distributed variables) and Mann Whitney U test (for non-normally distributed variables) were employed for comparison between the patients with MRC ≤ 2 and those with MRC ≥ 3. Categorical variables were expressed as counts and percentages and analyzed with the chi-square test. For all tests, a significant level of statistics was considered when the $p<0.05$.

RESULTS:

The age, sex, disease duration, rehabilitation, and MRC scale score for all patients with stroke were presented in Table 1.

Table 1: Baseline clinical data of patients with stroke.

No.	Age (years)	Sex	Stroke duration (months)	Rehabilitation	MRC scale
1	31	F	3	No	≥ 3
2	38	M	6	Yes	≥ 3
3	59	M	6	No	≤ 2
4	49	M	4	Yes	≥ 3
5	46	M	6	No	≥ 3
6	36	F	5	Yes	≥ 3
7	49	F	5	Yes	≥ 3
8	50	F	4.5	No	≥ 3
9	48	M	5	No	≥ 3
10	62	M	4.5	Yes	≤ 2
11	53	F	5.5	No	≥ 3
12	56	M	3.5	No	≥ 3
13	55	M	4.5	Yes	≥ 3
14	60	M	4.5	Yes	≤ 2
15	59	M	6	No	≤ 2
16	45	F	4	Yes	≥ 3
17	62	M	5	No	≤ 2
18	35	M	6	Yes	≥ 3
19	51	M	5	No	≤ 2
20	44	M	3	Yes	≥ 3
21	46	M	3.5	No	≥ 3
22	54	F	5.5	No	≥ 3
23	41	M	4	Yes	≥ 3

Neurophysiological data
Paretic versus non-paretic limb

The median DML was significantly prolonged (p = 0.029) on the paretic side (4.03±0.98 ms) as compared to the 3.75±0.64 ms on the non-paretic side. The median CMAP amplitude, MCV, DSL, SNAP amplitude, and SCV were not different between the two sides.

The peroneal DML was significantly prolonged (p = 0.015) in the paretic side (5.53±0.73 ms) as compared to 5.14±0.76 ms of the non-paretic side. On the contrary, the peroneal CMAP amplitude was significantly reduced (p = 0.001) in the paretic limb (2.8±1.19 mV) as compared to 3.63±1.34 mV of the non-paretic limb. The peroneal MCV was not different between the two limbs (Table 2).

Table 2: Nerve conduction studies on paretic and non-paretic sides.

Variables		Paretic (n=23)	Non-Paretic (n=23)	p-value
Median	DML (ms)	4.03±0.98	3.75±0.64	0.029
	CMAP amplitude (mV)	9.38±6.44	8.86±5.3	0.419
	MCV (m/s)	50.24±8.17	49.28±5.95	0.328
	DSL (ms)	3.64±0.6	3.64±0.57	0.771
	SNAP amplitude (μV)	42.3±16.32	41.76±26.45	0.205
	SCV (m/s)	48.26±7.44	49.13±7.57	0.259
Peroneal	DML (ms)	5.53±0.73	5.14±0.76	0.015
	CMAP amplitude (mV)	2.8±1.19	3.63±1.34	0.001
	MCV (m/s)	43.27±6.68	44.32±5.43	0.318

DML = distal motor latency; CMAP = compound muscle action potential;
MCV = motor conduction velocity.

Using the median-ulnar palm difference, the mean median DSL was significantly prolonged (p = 0.001) on the paretic side (2.79±0.49 ms) as compared to 2.46±0.41 ms on the non-paretic side whereas no difference was observed with ulnar DSL. Similarly, using the ring finger difference, the mean median DSL was significantly prolonged (p = 0.003) on paretic sides (3.64±0.55 ms) versus 3.33±0.34 ms on the non-paretic side. Meanwhile using the ulnar DSL, the difference was non-significant.

Likewise, using the thumb difference, the mean median DSL was 3.37±0.45 ms in the paretic limb which is significantly prolonged (p = 0.019) when compared to the 3.13±0.36 ms in the non-paretic limb. Meanwhile, the radial DSL was not significantly different between the two limbs. Also, using the lumbrical/ interosseous comparison method, the mean median DML was significantly prolonged (p = 0.014) when comparing 3.7±0.67 ms of the paretic limb and 3.44±0.41 ms of the non-paretic limb. The mean ulnar DML was not significantly different between the two limbs (Table 3).

Table 3:Comparison methods between paretic and non-paretic limbs.

Variables	Paretic (n=23)	Non-Paretic (n=23)	p-value
Median / palm	2.79±0.49	2.46±0.41	0.001
Ulnar / palm	2.15±0.23	0.15±0.33	1.0
Median / digit IV	3.64±0.55	3.33±0.34	0.003
Ulnar / digit IV	3.04±0.3	2.97±0.21	0.816
Median/digit I	3.37±0.45	3.13±0.36	0.019
Radial/digit I	2.69±0.3	2.63±0.37	0.31
Median / lumbrical	3.7±0.67	3.44±0.41	0.014
Ulnar / interosseus	3.04±0.25	2.96±0.34	0.079

Neurophysiological tests concerning the MRC scale

The demographic features of patients with MRC scale ≥ 3 and those ≥ 2 were presented in table 4. No significant difference was noticed in the duration of disease, sex, and rehabilitation process. Meanwhile, the age was significantly different between the two groups (p < 0001).

Table 4:Demographic features in stroke patients concerning MRC scale.

Variables	MRC scale		p-value
	≤ 2 (n=6)	≤ 3 (n=17)	
Age, years	58.83±4.07	45.65±7.37	<0.001
Gender			
Males	6(100%)	10(58.82%)	0.059
Females	0(0%)	7(41.18%)	
Rehabilitation			
No	4(66.67%)	8(47.06%)	0.408
Yes	2(33.33%)	9(52.94%)	
Disease duration, months	5.17±0.68	4.59±1.02	0.214

The paretic limbs

The mean median DML equals 4.92±1.1 ms in those with an MRC scale of ≤ 2 which is significantly prolonged (p = 0.006) as compared to 3.71±0.73 ms of those with MRC scale ≥ 3. Likewise, the mean median MCV in those with an MRC scale of ≤ 2 was significantly reduced (p = 0.036) in comparison to those with an MRC scale ≥ of 3 (44.33±4.93 m/s versus 52.32±8.15 m/s).

Furthermore, the mean median DSL was significantly prolonged (p = 0.011) in those with an MRC scale of ≤ 2 (4.3±0.83 ms) when compared to 3.48±0.44 ms of those with an MRC scale ≥ 3. Also, the mean peroneal CMAP amplitude (1.98±0.56 mV) was significantly reduced (p = 0.047) in those with an MRC scale of ≤ 2 versus 3.1±1.23 mV of those with MRC scale ≥ 3.

The median CMAP and SNAP amplitudes, median SCV, peroneal DML, and MCV were not different concerning the MRC scale (Table 5).

Table 5: Nerve conduction studies in paretic limbs according to the MRC scale.

Variable		MRC scale		P-value
		≤ 2 (n = 7)	≥ 3 (n = 16)	
Median	DML (ms)	4.92±1.1	3.71±0.73	0.006
	CMAP amplitude (mV)	4.48±0.66	11.11±6.69	0.062†
	MCV (m/s)	44.33±4.93	52.32±8.15	0.036
	DSL (ms)	4.3±0.83	3.48±0.44	0.011
	SNAP amplitude (µV)	23.5±10.41	35.76±16.79	0.183†
	SCV (m/s)	44.0±3.46	49.3±47.83	0.208†
Peroneal	DML (ms)	5.77±0.58	5.44±0.77	0.356
	CMAP amplitude (mV)	1.98±0.56	3.1±1.23	0.047
	MCV (m/s)	39.17±1.33	44.72±7.23	0.079

† Mann Whitney U test; DML = distal motor latency; CMAP = compound muscle action potential; MCV = motor conduction velocity.

Using the median-ulnar palm difference, the mean median DSL was significantly prolonged (p = 0.003) in those with MRC scale ≤ 2 (3.4±0.58 ms) as compared to 2.64±0.49 ms of those with MRC scale ≥ 3. No difference was observed with ulnar DSL. Similarly, using the ring finger difference, the mean median DSL was significantly prolonged (p = 0.014) in those with MRC scale ≤ 2 versus on paretic sides (4.23±0.74 ms) versus 3.5±0.42 ms in those with MRC scale ≥ 3. Meanwhile the ulnar DSL, the difference was non-significant.

On the contrary, using the thumb difference, neither the mean median DSL nor the radial DSL was significantly different between those with MRC scale ≤ 2 or ≥ 3.

Using the lumbrical/interosseous comparison method, the mean median DML was significantly prolonged (p = 0.001) when comparing 4.42±0.73 ms of those with MRC scale ≤ 2 and 3.45±0.43 ms of those with an MRC scale of ≥ 3. Likewise, the mean ulnar DML was significantly prolonged (p = 0.042) when comparing 3.22±0.24 ms of those with MRC scale ≤ 2 and 2.98±2.22 ms of those with MRC scale ≥ 3 (Table 6).

Table 6: Comparison methods in the paretic limbs according to the MRC scale.

Variable	MRC scale		P-Value
	≤ 2 (n = 7)	≥ 3 (n = 16)	
Median / palm	3.4±0.58	2.64±0.49	0.003
Ulnar/palm	2.3±0.13	2.09±0.23	0.054
Median / digit IV	4.23±0.74	3.5±0.42	0.014
Ulnar / digit IV	3.1±0.36	3.0±0.29	0.834
Median/digit I	3.7±0.65	3.29±0.38	0.103
Radial/digit I	2.77±0.021	2.66±0.33	0.463
Median / lumbrical	4.42±0.73	3.45±0.43	0.001
Ulnar / interosseus	3.22±0.24	2.98±2.22	0.042

† Mann Whitney U test

The non-paretic limbs

The mean median DML was significantly prolonged also (p = 0.005) in those with an MRC scale of ≤ 2 (4.18±0.67 ms) when compared to 3.6±0.57 ms of those with an MRC scale ≥ 3. Similarly, the mean median DSL was significantly prolonged (p = 0.028) in those with MRC scale ≤ 2 (3.7±0.44 ms) versus 3.14±0.48 ms of those with an MRC scale of ≥ 3.

The median CMAP and SNAP amplitudes, MCV and SCVs, and peroneal DML, CMAP amplitude, and MCV were not significantly different concerning the MRC scale (Table 7).

By using the median-ulnar palm difference, neither the mean median DSL nor the ulnar DSL was significantly different between those with MRC scale ≤ 2 or MRC scale ≥ 3. While using the ring finger difference, the mean median DSL was significantly prolonged (p = 0.066) in those with MRC scale ≤ 2 versus on paretic sides (3.62±0.43 ms) versus 3.29±0.32 ms in those with MRC scale ≥ 3. Meanwhile the ulnar DSL, the difference was non-significant.

Table 7:Nerve conduction studies in non-paretic limbs according to the MRC scale.

Variable		MRC scale		P-value
		≤ 2 (n = 7)	≥ 3 (n = 16)	
Median	DML (ms)	4.18±0.67	3.6±0.57	0.005
	CMAP amplitude (mV)	5.45±1.18	10.07±5.69	0.087†
	MCV (m/s)	46.0±4.0	50.44±6.19	0.118
	DSL (ms)	3.7±0.44	3.14±0.48	0.028
	SNAP amplitude (μV)	30.0±15.65	43.29±28.4	0.135†
	SCV (m/s)	45.12±3.5	49.81±8.17	0.192
Peroneal	DML (ms)	5.23±0.4	5.11±0.85	0.746
	CMAP amplitude (mV)	2.97±0.71	3.87±1.45	0.163
	MCV (m/s)	41.53±2.12	45.31±5.98	0.148

† Mann Whitney U test; DML = distal motor latency; CMAP = compound muscle action potential; MCV = motor conduction velocity.

Using the thumb difference, neither the mean median DSL nor the radial DSL was significantly different between those with MRC scale ≤ 2 or ≥ 3.

Using the lumbrical/interosseous comparison method, the mean median DML was significantly prolonged (p = 0.039) when comparing 3.73±0.64 ms of the those with MRC scale ≤ 2 and 3.34±0.23 ms of those with an MRC scale of ≥ 3. Likewise, the mean ulnar DML was significantly prolonged (p = 0.039) when comparing 3.2±0.21 ms of those with MRC scale ≤ 2 and 2.87±0.34 ms of those with MRC scale ≥ 3 (Table 8).

Table 8: Comparison methods in the non-paretic limbs according to the MRC scale.

Variable	MRC scale		P-value
	≤ 2 (n = 6)	≥ 3 (n = 17)	
Median / palm	2.77±0.42	2.42±0.4	0.091
Ulnar/palm	2.3±0.39	2.09±0.3	0.198
Median / digit IV	3.62±0.43	3.29±0.32	0.066
Ulnar / digit IV	3.02±0.37	2.95±0.14	0.543
Median/digit I	3.45±0.54	3.12±0.35	0.098
Radial/digit I	2.82±0.39	2.57±0.35	0.165
Median / lumbrical	3.73±0.64	3.34±0.23	0.039
Ulnar / interosseus	3.2±0.21	2.87±0.34	0.039

† Mann Whitney U test

DISCUSSION:

In our study, the significant difference in the DML of the median nerve between the paretic and non-paretic sides was also reported by other researchers [22,23]. It is thought to be due to continuous abnormal posture as well as possible edema because of a long stay in the bed particularly within the early stages of stroke. Moreover, the non-significant reduction in the median CMAP and SNAP amplitudes between the plegic side and healthy side is in harmony with the finding of Akyuz et al. [24] but contradicts the findings of Güneş et al. [25] probably because of the small sampled size of our study.

On comparing the DML and CMAP amplitude of the peroneal nerve, it was found that the difference was significant between the paretic and non-paretic sides. The findings harmonize those of other researchers [14,26].

These findings are thought to be due to spasticity of ankle plantar flexors and/or weakness of ankle dorsiflexors in hemiplegic patients, which leads to an equinovarus position of the foot. The continuous inversion position probably results in nerve traction and compression at the level of the fibular neck, causing demyelination and even axonopathy. Myelin loss results in a slowing of nerve conduction through the area involved. When compression is severe, ischemic changes occur and cause secondary axonal damage, expressed by reduction of CMAP amplitude [14,26].

Furthermore, an LMN involvement was suggested to occur in stroke patients as a sort of "dying back" neuropathy due to motor unit deafferentation [16,27]. The hypothesis was that UMNs results in a loss of synaptic input to the spinal alpha motoneurons, which become functionally inactive or undergo transsynaptic degeneration leading to disturbances of the axonal flow, axonal degeneration, dysfunction of neuromuscular transmission at the motor endplate, and reduction of functionally active motor units [27].

The combined sensory index (median versus ulnar mixed palmar response, median versus ulnar DSL recording from digit IV, and median versus radial DSL from digit I) [28,29] and median–ulnar lumbrical-interosseous comparison study [30,31] were significantly different between the paretic and non-paretic upper limbs. To the best of our knowledge, no comparable data are present in the literature.

As for the ulnar and radial DSLs, no difference was demonstrated in the present study. This is possible because the DSLs of ulnar and radial nerves reflect the conduction across distal segments of those nerves which are relatively not susceptible to entrapment.

Moreover, it was found that the median DSL and DML were significantly prolonged and the MCV was significantly slowed on the paretic side of patients with ≤ 2 MRC scale when compared with those on the paretic side of patients with ≥ 3 MRC scale. These findings were following the results of Odabas et al. [32]. These differences are possible because the more severe hemiparesis in patients with a less MRC scale leads to a more severe disabled posture on the affected limb resulting in more affected nerve conduction study parameters on that affected side.

Likewise, the peroneal CMAP amplitude was significantly reduced on the paretic side of patients with ≤ 2 MRC scale compared to that on the paretic side of patients with the MRC scale. Odabas et al [32] found a significant amplitude loss in motor and sensory responses of the peroneal nerve in the patient group with severe paresis (P = 0.03) [32]. The finding is possibly due to more severe equinovarus position and possibly more traction on the peroneal nerve in patients with a lower MRC scale than those with a higher MRC scale.

For the same possible reasons, using the two comparison methods demonstrates significantly prolonged DSLs and DMLs on paretic limbs in those with ≤ 2 MRC scale versus those with ≥ 3 MRC.

Comparing the neurophysiologic data in the non-paretic limbs, the median DSL and DML were significantly prolonged in those with ≤ 2 when compared to those with ≥ 3 MRC scale. To the best of our knowledge, no comparable data are present in the literature. The reason why this difference in non-paretic limbs was possibly related to the overuse of contralateral asymptomatic side to compensate for daily activities in patients with hemiparesis. This goes with the notion of Sato et al "the ratio of entrapment neuropathies increases in the asymptomatic sides because of overuse of unaffected extremities" [33].

Furthermore, combined sensory index and the median-ulnar lumbrical-interosseus study showed significantly prolonged DSL and DML on non-paretic limbs between those with ≤ 2 MRC scale when compared to those with ≥ 3 MRC scale. Likewise, no comparable data present in the literature.

This analysis of the non-paretic hand, which showed that there are significant findings between median DML and DSL values in those with ≤ 2 MRC as compared to those with ≥ 3 MRC, was following findings of Moghtaderi et al. [22].

CONCLUSION:

Our study showed an electrodiagnostic asymmetry in the paretic upper and lower extremities. Entrapment may develop, along with some axonal neuropathy symptoms in the affected extremities in patients with severe paresis. The changes are more severe in more severely affected limbs.

List of abbreviation

CMAP = compound muscle action potential
CV = conduction velocity
DML = distal motor latency
DSL = distal sensory latency
LMN = lower motor neuron
MRC = Medical Research Council
SNAP = sensory nerve action potential
UMN = upper motor neuron

Competing interests

The authors declare no conflict of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' contributions

All the authors have directly participated in the preparation of this manuscript and have approved the final version submitted. 'AH' clinically examined and referring stroke patients. 'MM' and 'FH' did the electrodiagnostic tests. 'FH' and 'MM' drafted the manuscript. 'AH', 'MM' and 'FH' conceived the study and participated in its design and interpretation. All the authors have read and approved the final manuscript.

Acknowledgements

We thank assistant professor Dr. Qasim Al-Mayah from the Research Medical Unit / College of Medicine/Al-Nahrain University for helping in statistical analysis.

REFERENCES:

1. Picelli A, Tamburin S, Gajofatto F, Zanette G, Praitano M, Saltuari L, et al. Association between severe upper limb spasticity and brain lesion location in stroke patients. Biomed Res Int. 2014; Article ID 162754.
2. Emami Razavi S Z, Azadvari M. Electrodiagnostic Findings in Post-Stroke Patients, Arch Neurosci. 2019;6:e83417.
3. van Kuijk AA, Pasman JW, Hendricks HT, Zwarts MJ, Geurts ACH. Predicting hand motor recovery in severe stroke: the role of motor evoked potentials in relation to early clinical assessment. Neurorehabil Neural Repair. 2009;23:45-51.
4. Scherbakov N, Sandek A, Doehner W. Stroke-related sarcopenia: specific characteristics. J Am Med Dir Assoc. 2015;16:272-276.
5. Picelli A, Bonetti P, Fontana C, Barausse M, Dambruoso F, Gajofatto F, et al. Is spastic muscle echo intensity related to the response to botulinum toxin type A in patients with stroke? A cohort study. Arch Phys Med Rehabil. 2012;93:1253-1258.
6. Picelli A, Tamburin S, Cavazza S, Scampoli C, Manca M, Cosma M, et al. Relationship between ultrasonographic, electromyographic, and clinical parameters in adult stroke patients with spastic equinus: an observational study. Arch Phys Med Rehabil. 2014;95:1564-1570.
7. Berenpas F, Martens AM, Weerdesteyn V, Geurts AC, van Alfen N. Bilateral changes in muscle architecture of physically active people with chronic stroke: a quantitative muscle ultrasound study. Clin Neurophysiol. 2017;128:115-122.
8. Nam JS, Choe YR, Yoon SY, Yi TI. Upper limb pain and paresthesia in a post-stroke patient treated with ultrasound-guided electrical twitch-obtaining intramuscular stimulation (ETOIMS) of scalene muscles. Brain Neurorehabil. 2018;11:e1
9. Laufer Y. The effect of walking aids on balance and weight-bearing patterns of patients with hemiparesis in various stance positions. Phys Ther. 2003;83:112-122.
10. Mold JW, Lawler F, Roberts M, Oklahoma Physicians Resource/Research Network Study. The health consequences of peripheral neurological deficits in an elderly cohort. J Am Geriatr Soc. 2008;56:1259-1264.
11. Kabayel L, Balci K, Turgut N, Kabayel DD. Development of entrapment neuropathies in acute stroke patients. Acta Neurol Scand. 2009;120:53-58.
12. Kang JI, Moon YJ, Jeong DK, Choi H. The effect of rhythmic neurodynamic on the upper extremity nerve conduction velocity and the function for stroke patients. J Kor Phys Ther. 2017;29:169-174.

13. İsnaç F, Aşkin A, Şengül İ, Demirdal ÜS, Tosun A. Ultrasonographic and electrophysiologic evaluation of median and ulnar nerves in chronic stroke patients with upper extremity spasticity. Somatosens Mot Res. 2019;36:144-150.
14. Tsur A. Common peroneal neuropathy in patients after the first-time stroke. Isr Med Assoc J. 2007; 9:866-869.
15. Nozoe M, Kubo H, Kanai M, YamamotoM, Shimada S, Mase K. Peripheral motor nerve conduction abnormality, muscle strength, and muscle wasting in patients with acute stroke: A pilot study. J Clin Neurosci. 2020;75:80-84.
16. Paoloni M, Volpe B, Mangone M, Ioppolo F, Santilli V. Peripheral nerve conduction abnormalities in the nonparetic side of ischemic stroke patients. J Clin Neurophysiol. 2010;27:48-51.
17. Dozono K, Hachisuka A, Wada F, Hachisuka K. Peripheral neuropathies in nonparetic upper extremities of stroke patients induced by excessive use of a walking device. J Stroke Cerebrovasc Dis. 2015;24:1841-1847.
18. Berenpas F, Weerdesteyn V, Geurts AC, van Alfen N. Long-term use of implanted peroneal functional electrical stimulation for stroke-affected gait: the effects on muscle and motor nerve. J Neuro Engineering Rehabil. 2019;16:86.
19. Hermans G, Clerckx B, Vanhullebusch T, Segers J, Vanpee G, Robbeets C, et al. Interobserver agreement of Medical Research Council sum-score and handgrip strength in the intensive care unit. Muscle Nerve. 2012;45(1):18-25.
20. Turan Z, Topaloglu M, Ozyemisci Taskiran O. Medical Research Council-sum score: a tool for evaluating muscle weakness in patients with post-intensive care syndrome. Crit Care. 2020;24:562.
21. Preston DC, Shapiro BE. Electromyography in neuromuscular disorders: fourth edition, Clinical-Electrophysiologic Correlations. Chapter 20, Elsevier, 2020; p. 107-114, 124-128.
22. Moghtaderi A, Dahmardeh M, Dabiri S. Subclinical carpal tunnel syndrome in patients with acute stroke. Ir J Neurol. 2012;11:91-95.
23. Picelli A, Tamburin S, Berto G, Chemello E, Gandolfi M, Saltuari L, et al. Electrodiagnostic and nerve ultrasonographic features in upper limb spasticity: an observational study. Functional Neurol. 2017; 32:119-122.
24. Akyüz M, Özduran I, Uysal FG, Çamoğlu S, Çakcı A. Nerve conduction examinations in hemiplegic patients. J Turkish Phys Med Rehabilit. 1998;1:425-442.
25. Güneş S, Genç A, Gök H, Gökmen D, Kutlay Ş. Effects of spasticity and hemiplegic posture on median nerve and carpal tunnel in stroke patients: Electrophysiological and ultrasonographic evaluation. J Clin Neurosci. 2020;17:P31-35
26. Chatriwala ZMM, Shende M. Nerve conduction velocity of common peroneal nerve in post stroke patients. An observational study. IOSR-JSPE. 2016;3:50-55.
27. van Kuijk AA, Pasman JW, Hendricks HT, Schelhaas JH, Zwarts MJ, Geurts AC. Supratentorial ischemic stroke: more than an upper motor neuron disorder. J Clin Neurophysiol. 2007;24:450-455.
28. Zeidman LA, Pandey DK. A carpal tunnel grading system including combined sensory index□diagnosed mild cases: Relation to presenting features and outcomes. Muscle Nerve. 2018;57:45-48.
29. Mustafa MMMA, Nassar N, Amen I, Abdelmohsen H. Combined Sensory Index test versus diagnostic ultrasonography in early detection of carpal tunnel syndrome. QJM. 2020;113(suppl. 1):hcaa064.006.
30. Lee S-H, Kim D-H, Cho H-M, Nam H-S, Park D-S. Diagnostic value of the second lumbrical-interosseous distal motor latency comparison test in severe carpal tunnel syndrome. Ann Rehabil Med. 2016;40:50-55.
31. Yılmaz F, Gündüz OH, Akyüz G. Lumbrical-interosseous recording technique versus routine electrodiagnostic methods in the diagnosis of carpal tunnel syndrome. Turk J Phys Med Rehab. 2017;63:230-238.
32. Odabas FO, Sayin R, Milanlioglu A, Tombul T, Cögen EM, Yildirim G. Electrophysiological analysis of entrapment neuropathies developed in the acute and subacute period in paretic and non-paretic extremities in patients with stroke. J Pak Med Assoc. 2012;62:649-652.
33. Sato Y, Kaji M, Tsuru T. Carpal tunnel syndrome involving unaffected limbs of stroke patients. Stroke. 1999;30:414-418.