Relation Between Spasticity And Carpal Tunnel Syndrome In Patients With Stroke

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Abstract

Background: Stroke is the most common cause of mortality and is one of the most common causes of morbidity in the world. Polyneuropathies and entrapment neuropathies are known as the complications of stroke. **The purpose** of this study was to Investigate the relation between carpal tunnel syndrome in the affected and non-affected hands in patients with stroke.**Methodology:** sixty stroke patients of both sexes, their ages were ranged from (40-65) years were assigned into three equal groups (group a, b and c): group (A) with mild spasticity (grade 1 and 1+) while group (B) with moderate spasticity (grade 2 and 3) and group (c) with severe spasticity (grade 4) according to modified ashowrth scale. Subjects were assessed using Electrodiagnostic testing (nerve conduction velocity) and clinical tests (Phalen's test, Tinel's sign, Durkan's test and the hand elevation test). **Results:** This study revealed that patients with stroke had carpal tunnel syndrome in the affected hand as a result of spasticity and in the non-affected hand due to overuse but the results were more significant in group (c). **Conclusion:** It was suggested that simultaneous different mechanisms may act in inducing carpal tunnel syndrome in both hands of hemiparetic patient.Our results confirm that, in severe hemiparetic patients, the entrapment neuropathies may be commonly seen, especially in the paretic extremities. The early rehabilitation programs against the development of entrapment neuropathies may be beneficial in stroke patients.

(Key Words: Stroke, Spasticity, Carpal tunnel syndrome, Paretic hand).

Introduction

Stroke is a medical condition in which poor blood flow to the brain results in cell death. There are two main types of stroke: ischemic, due to lack of blood flow, and hemorrhagic, due to bleeding. They result in part of the brain not functioning properly.[(1)]

The neurologic symptoms manifest within seconds because neurons need a continual supply of nutrients, including glucose and oxygen, that are provided by the blood. Therefore, if blood supply to the brain is impeded, injury and energy failure is rapid[(2)].

Spasticity is a common symptom presented amongst people with sensorimotor disabilities due to stroke. It is one of the positive signs among other motor symptoms which occur after lesions in the descending corticospinal system as it causes. It was observed that spasticity primarily affects the elbow flexors (79% of patients), the wrist flexors (66%) and the ankle planter flexors (66%)[(3)].

While negative signs of upper motor neuron symptoms include pain, fatigue, anxiety, weakness, lack of coordination and make patients more dependent either on the others or on assistive devices to perform their ADL [(3)]

Hemiparesis/ hemiplegia involving the hand and arm results in regular, repeated overuse of the non paretic hand and wrist in rough correspondence to the degree of spasticity.In the upper limbs, the most frequent pattern of arm spasticity is internal rotation and adduction of the shoulder coupled with flexion at the elbow, the wrist and the fingers and pronation of the forearm in addition to scapular retraction[(5)].

As a result of fingers flexor spasticity, fingernails digging into palmar skin resulting in several complications including nail bed infections, pain when somebody attempts to force fingers opening to gain palmar access, Skin maceration, breakdown in the palm, difficulties to wear gloves or hand splints, Limitation for grasping, manipulation and release of objects and difficulties to execute grasp patterns (lateral grasp and tip pinch) [(5)].

Stroke patients with disuse of the paretic hand maybe forced to overuse their non-paretic hand while performing activities of daily living (ADLs) which largely depend on arm function,

particularly for personal activities such as feeding, dressing and grooming ,also particularly when assistive devices are used for mobility[4].

As frequent and repetitive use of the hand or wrist is a common cause of carpal tunnel syndrome (CTS) and stroke patients are vulnerable to compression neuropathy or entrapment neuropathy due to inability to move or using crutches during rehabilitation or as a result of flexed posture of hand due to spasticity such hand and wrist overuse may cause CTS in patients with stroke[6].

Upper limb spasticity may result in flexed wrist posture by involvement of the flexor muscles and this wrist posture has the potential to compress the median nerve at the carpal tunnel. This compression might also be related with the decreasing carpal tunnel area. It means that the cross-sectional area of the median nerve decreases as time of nerve compression passes [7].

Histologically compression neuropathy causes edema, fibrosis, demyelination, and Wallerian degeneration. This is probably related to the effects of chronic ischemia with secondary atrophy of the median nerve. Also, chronic compression may progress to fibrosis of the nerve in the chronic phase following stroke [7].

Compression of the median nerve at wrist which occur as a result of spasticity of wrist and finger flexors in the paretic hand resulting in carpal tunnel syndrome ,hand pain, Awkward hand placement during reaching and impairs positioning of objects held and Weakened grip strength[5].

It is usually missed to assess the status of the peripheral nerves in such patients due to motor disability.so the purpose of this study is to: Investigate the relation between carpal tunnel syndrome in the affected and non-affected hands in patients with stroke.

Subjects

Sixty patients were selected from the Out-Patient Clinics of Neurology in Kasr Al- Aini Hospitals and Out Patient Clinic of Neurology, Faculty of Physical Therapy, Cairo University. Patients were chosen according to the following criteria :

Inclusive criteria:

1- Sixtypatients with hemiplegia /paresis subsequent to a stroke as diagnosed by CT or MRI.
2- Right or left-handedness before the stroke,
3- Sixty patients from both sexes, their ages ranged from (40-65) years.
4- Body weight of patients was ranged from(55 -95 kg) ,while their height was from (151-185 cm) and BMI was ranged from (20-30 kg/m2).
5- Normative value of MDL is (≤ 4.2m.sec) , MCV is (≥55m/s) ,SDL is (≤ 3.6 m.sec) and SCV is (≥ 55m/s).
6- Duration of illness (6 – 9 months post stroke).
7- Patients have spasticity ranging from grade 1 to grade 4 according to modified Ashworth scale
8- Patients were medically stable

Exclusive criteria:

1- patients with cervical radiculopathy that could mimic CTS or interfere with its evaluation
2- patients with proximal median neuropathy, significant polyneuropathy, or marked orthopedic abnormalities;
3- patients with contractures
4- Patients with psychological disturbance or seizures
5- systemic diseases known to cause CTS, such as diabetes mellitus, hypothyroidism, rheumatoid arthritis, or chronic renal failure;

Design of the study

Cross sectional study "assessment study".

Patients were assigned to three equal groups randomly :

Group A: Consist of twenty patients with mild spasticity (grade 1 and 1+) .

GroupB: Consist of twenty patients with moderate spasticity (grade 2 and 3) .

Group C: Consist of twenty patients with severe spasticity (grade 4) according to modified ashowrth scale .

Instrumentations:

Electrophysiological instrumentation:

Electrodiagnostic testing (electromyography and nerve conduction velocity) can objectively verify the median nerve dysfunction. Electrodiagnosis rests upon **demonstrating** impaired median nerve conduction across the carpal tunnel in context of normal conduction. Compression results in damage to the myelin sheath and manifests as delayed latencies and slowed conduction velocities[8].

The Neuropack S1 MEB-9004 NIHON KODEN, JAPANwas utilized to obtain an objective evaluation of the motor and sensory conduction velocities of the peripheral nerves. It is designed to be a compact, self-contained unit which composed of a main unit featuring high performance 2-channel amplifiers, a junction box with isolation amplifiers and an articulated arm

Methods

Position of patients and electrodes: Upon arrival:

The patient was be in a relaxed sitting position on a chair and the forearm supported on the plinth. While performing motor conduction velocity apply the earth electrode around the wrist level in motor conduction.Then applythe recording motor electrodes, negative electrode on the center of the corresponding abductor pollicis brevis muscle and the positive electrode 3 cm distal to the negative electrode. Stimulate the median nerve at wrist level (just above the wrist in the midline) and median nerve was stimulated also at forearm level (Cubital fossa just medial to biceps brachii tendon) while performing motor conduction study and recording is obtained.

Measure the distance between distal and proximal stimulation to calculate conduction velocity of the median nerve to ensure that the conduction velocity is normal or not. Nerve conduction velocity was calculated by the following formula:

$$\text{Conduction velocity (meter / second} \quad \frac{\text{Distance (cm) X10}}{\text{Proximal latency – Distal latency}}$$

While when performing antidromicsensory conductionvelocity apply earth electrode around the hand in sensory conduction velocity, then apply the recording ring electrodes at index finger with the negative electrode proximal to the distal one. Stimulate the median nerve at wrist level (just above the wrist in the midline) sensory conduction study and recording is obtained.

Statistical analysis:

Statistical analysis

Descriptive statistics and ANOVA-test were conducted for comparison of subject characteristics between groups. Chi- squared test was used for comparison of sex and affected side distribution between groups. Normal distribution of data was checked using the Shapiro-Wilk test for all variables. Levene's test for homogeneity of variances was conducted to test the homogeneity between groups. MANOVA was conducted to compare the mean values of distal latency, MCV and peak latency between the three groups. Post-hoc tests using the Tukey were carried out for subsequent multiple comparison. Paired t-test was conducted for comparison between the affected and the non affected sides. The level of significance for all statistical tests was set at $p < 0.05$. All statistical measures were performed through the statistical package for social studies (SPSS) version 22 for windows.

RESULTS

- Subject characteristics:

Table 1 showed the mean ± SD of subjects' characteristics of the study groups. There was no significant difference between the three groups in the mean age and BMI ($p > 0.05$). Also, there was no significant difference in the distribution of sexand affected side between the three groups ($p > 0.05$).

Table. Basic characteristics of all participants:

	Group A	Group B	Group C	p-value
	$\overline{X}$±SD	$\overline{X}$±SD	$\overline{X}$±SD	
Age (years)	54.5 ± 5.37	53 ± 5.87	54.15 ± 6.89	0.71*
BMI (kg/m²)	27.56 ± 3	26.37 ± 1.85	27.07 ± 2.9	0.36*
Sex distribution				
Females	10(50%)	9 (45%)	8 (40%)	0.81*
Males	10(50%)	11 (55%)	12 (60%)	
Affected side				
Dominant	12 (60%)	14(70%)	14 (70%)	0.74*
Non dominant	8 (40%)	6 (30%)	6 (30%)	

$\overline{X}$**, Mean; SD, standard deviation; p-value, level of significance; * Non-significant.**

Effect of spasticity on distal latency, MCV and peak latency:

MANOVA revealed that there was a significant effect of spasticity (group effect) on distal latency, MCV and peak latency (Wilks' Lambda = 0.25; $F(12,104) = 8.48$, $p = 0.001$). Table 2 showed descriptive statistics of distal latency, MCV and peak latency of the affected and non affected sides as well as the significant level of comparison between groups.

There was no significant difference between the three groups in distal latency of the affected and non affected sides ($p > 0.05$).

There was a significant increase in MCV of the affected and non affected sides of group A compared with that of group B and group C ($p < 0.001$), while there was no significant between group B and C ($p > 0.05$).

There was a significant decrease in peak latency of the affected and non affected sides of group A compared with that of group C ($p < 0.05$). There was no significant difference in peak latency of the affected and non affected sides between group A and B and between group B and C ($p > 0.05$).

Comparison between affected and non affected sides revealed non significant difference in distal latency, MCV and peak latency between both sides in the three group ($p > 0.05$). (table 2).

Table 4:*Mean distal latency, MCV and peak latencyof the affected and non affected sides of group A, B and C:*

	Group A	Group B	Group C	p-value		
	$\overline{X}$±SD	$\overline{X}$±SD	$\overline{X}$±SD	Avs B	AvsC	BvsC
Distal latency (msec)						
Affected side	3.68 ± 0.55	4.17 ± 0.96	4.24 ± 1.04	0.19*	0.11*	0.96*
Non affected side	3.57 ± 0.48	4.03 ± 1.15	4.32 ± 1.27	0.33*	0.06*	0.65*
	p = 0.29*	***p = 0.61****	***p = 0.81****			
MCV (m/sec)						
Affected side	57.78 ± 5.06	48.57 ± 7	46.46 ± 7.16	0.001**	0.001**	0.56*
Non affected side	58.13 ± 3.84	51.14 ± 3.86	48.39 ± 5.44	0.001**	0.001**	0.13*
	p = 0.82*	***p = 0.19****	***p = 0.22****			
Peak latency (msec)						
Affected side	3.65 ± 0.94	3.82 ± 0.6	4.85 ± 2.24	0.93*	0.03**	0.07*
Non affected side	3.74 ± 0.47	3.93 ± 1.26	4.86 ± 1.71	0.87*	0.01*	0.06*
	p = 0.67*	***p = 0.66****	***p = 0.97****			

$\overline{X}$, Mean; SD, standard deviation; p-value, level of significance; * Non-significant; **Significant

DISCUSSION

One of the most important complication of stroke in the sub-acute and chronic period is the development of entrapment neuropathies. The entrapment neuropathies represent a group of peripheral nerve disorders that are characterized by pain, paresthesia and/or loss of function of nerves as a result of chronic compression along the route of peripheral nerves (Bozkurt.,2012)[17].

Although the nonparetic extremities in patients with stroke are generally considered to be normal, peripheral neuropathies of the median nerve on the unaffected side may occur due to overuse of the ambulatory assistive devices, or overuse of the nonparetic hand (Wright et al.,2017)[18].

There are studies suggesting that CTS most commonly develops in non-functional extremities with severe motor deficits. Thus, increased usage of the non- affected extremity, and the use of aiding device for support purposes cause CTS in the non-affected extremity (Kabayel et al.,2012)[12].

The results of the current study were revealed that there was no significant difference between the three groups in the mean value of distal latency of the affected side and of the non-affected side and our results were supported by Akyuz et al, (2012) [9] and Faruk et al ,(2012) [10] whom stated that there was no significant change in in distal latency values when conducted a median and ulnar motor and sensory nerve conduction study in healthy and hemiplegic upper extremities.

While Sato et al, (2012)[11]whom conducted a study on hemiplegic stroke patients dividing them into two groups as functional hand and unused hand. They detected significant abnormalities in sensory nerve distal latencies, motor nerve distal latencies and CMAP values in the non-paretic side. Thus, they concluded that excessive use of the non-paretic hand and wrist resulted in CTS in stroke patients, especially when the paretic hand is not functional.

There was a significant difference in MCV of the affected side between the three groups . There was a significant increase in MCV of the affected side of group A (group A has mild spasticity) compared with that of group B (group B with moderate spasticity) and that of group C (group C with severe spasticity). There was a significant difference in MCV of the non affected side between the three groups .There was no significant difference in MCV of the non affected side between group B and C .

This was in agreement with Kabayel et al ,(2012)[12] whom reported that in severe hemiparetic patients, the paretic hand is involved more than the other hand. They proposed that inflammation and edema are the most important risk factors that cause median nerve compression. They explained the pathologic process of median entrapment is similar to that was described earlier in the sciatic nerve models as proliferation of fibrous tissue, demyelination and axonal loss may be seen after the first week and finally marked fibrosis was seen after 28 days.

Also, these results go with Sato et al,(2012)[11] whom conducted a study on hemiplegic stroke patients and reported that in severe hemiparetic patients, the entrapment neuropathies such as median nerve neuropathy at the wrist, ulnar nerve neuropathy at the elbow and peroneal nerve neuropathy at the fibular head may be commonly seen, especially in the paretic extremities

But in prospective study done by McMorland et al ,(2015)[13]when compared the elasticity of the median nerve at the carpal tunnel on the paretic and nonparetic sides; they concluded that posture-induced changes are acute changes; however, the postural changes in spasticity are a long-standing period after the acute phase. The median nerve might have shown increased stiffness due to compression at the carpal tunnel.

Additionally McMorland et al ,(2015),[13] revealed that peripheral nerves were shown to have a slide and stretch mechanism to accommodate changes in the nerve bed length during joint movements. Likewise, the median nerve might have adapted the changes at the nerve bed length by this slide and stretch mechanism.

There was a significant decrease in peak latency of the affected side of group A compared with that of groups B and C. There was no significant difference in peak latency of the affected side between group B and C. There was a significant decrease in peak latency of the nonaffected side of group A compared with that of groups B and C. There was no significant difference in peak latency of the affected side between group B and C.

The result of this study is supported by Akyuz et al,(2012)[9] whom conducted a median and ulnar motor and sensory nerve conduction study in healthy and hemiplegic upper extremities and revealed that there were no significant changes in motor or sensory nerve conduction velocities and in distal latency values. Some patients with mild spasticity who did not use supporting device, were detected to have CTS in the wrist on the affected side only as a complication of spasticity

In a study conducted by Faruk et al,(2012)[10], whom investigate entrapment neuropathies in stroke patients in a hospital in Turkey with Medical Research Council (MRC) score < 2/5 and in those with MRC score > 3/5reported that the frequency of paretic entrapment findings on the paretic side increased in the patient group with severe paresis, and concluding that CTS developed in the non-paretic upper extremities more frequently.

It seems that stroke may be an independent risk factor CTS after one month. Increased pressure in the carpal tunnel due to edema of the upper extremity is probably the main reason for inducing

CTS in the paretic hand. On the other hand, overuse of the other hand especially for rehabilitation is an important risk factor for increased pressure in the same tunnel of the non-paretic hand (Ali et al,2012)[14].

There are three main leading theories of causation of entrapment neuropathies. First theory is the repeated compression that may cause edema in the sub-endoneurial space and synovium along with ischemia. Moreover, localized mechanical pressure of the surrounding structures such as flexor retinaculum and Palmaris longus tendon during normal movements or changes in position of the limbs may cause local nerve damage. Finally, the third mechanism which is not relevant here is tethering of the nerve due to scar tissues (Ali et al,2012)[14].

Stroke reduces arm function and independence, having an impact on self-estimated autonomy in activities of daily living; it also causes unpleasant sensations such as heaviness, rigidity and pain (Opheim et al.,(2015) [15]and Baricich et al., (2016)[16].

In a study conducted on median and ulnar nerve, it was found that NCS measures (DML, CMAP amplitude and F-wave minimal latency) significantly differed between the affected and unaffected upper limbs in chronic stroke patients (Baricich et al., 2016)[16].

To explain those data, it was suggested that spasticity in chronic stroke patients leads to changes in the muscle structure by increasing intramuscular connective tissue and fat content, and a lower motor neuron involvement was suggested to occur in stroke patients as a sort of “dying back” neuropathy due to motor unit deafferentation .The hypothesis was that UMNS results in a loss of synaptic input to the spinal alpha motor neurons, which become functionally inactive or undergo trans synaptic 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 (Opheim et al., 2015)[15].

Conclusion:

It was suggested that simultaneous different mechanisms may act in inducing carpal tunnel syndrome in both hands of hemiparetic patient. Our results confirm that, in severe hemiparetic patients, the entrapment neuropathies may be commonly seen, especially in the paretic extremities. The early rehabilitation programs against the development of entrapment neuropathies may be beneficial in stroke patients.

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