Original Article

Electrophysciological analysis of entrapment neuropathies developed in acute and subacute period in paretic and non-paretic extremities in patients with stroke

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Abstract

Objective: To 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 ≥ 3/5.

Methods: The study comprising 40 patients from January 2008 to June 2009 in the Stroke Unit of the Department of Neurology, Yuzuncu Yil University in Van, Turkey, entailed electrophysiological analysis of median, ulnar, radial nerves, peroneal, tibial and sural nerves in paretic and nonparetic upper and lower extremities. National Institute of Health Stroke scale was used for the evaluation of neurological deficient, while nerve conduction studies were performed for the diagnosis of entrapment neuropathies (EN). The patients were divided into two groups based on their initial Medical Research Council (MRC) score: < 2/5 and > 3/5. Both groups had 20 patients each. The readings were compared in the control phase 45 to 50 days after the stroke. Paired samples test and t-test using SPSS version 15 were used for statistical analysis.

Results: Carpal tunnel syndrome (CTS) at wrist was found in 7 (35%) patients, cubital tunnel syndrome at elbow in 3 (15%) patients, and evident reduction in motor action potential values of peroneal, median and ulnar nerve in 10 (50%) patients in the control studies for conduction on our patients with Medical Research Council (MRC) score of ≤ 2/5, unlike the initial findings, in the paretic side. Among the patients, in the other group, 2 (10%) developed bilateral Carpel tunnel syndrome, and it was also detected in the healthy upper extremities in 2 (10%) more patients. In the control studies for conduction in patients with Medical Research Council score of ≥ 3/5, Carpel tunnel syndrome was detected in the healthy side in 4 (20%) patients and in the affected side in 3 (15%) patients.

Conclusion: In patients with severe paresis, if the affected extremity is not functional, symptoms of entrapment neuropathy are easy to occur.

Keywords: Stroke, Entrapment neuropathy, Carpal tunnel syndrome, Cubital tunnel syndrome. (JPMA 62: 649; 2012)

Introduction

Cerebrovascular diseases (CVD) include a relatively important disease group regarding frequency and importance among neurological diseases in adulthood.[1] Peripheral neuropathy, which may develop during CVD and particularly during an ischaemic stroke, is one of the most important complications that should be considered. Entrapment neuropathy (EN) develops as a complication in patients with stroke, especially in those who are treated in rehabilitation units, and there is not enough data available regarding the specific period when this complication begins.[2]

The objective of our study was to determine the development of EN in paretic and nonparetic extremities in acute and sub-acute periods in stroke patients with MRC score of 2 or below and in those with MRC score of

3 or above, and to investigate the frequency of development of this complication.[3]

Patients and Methods

Initially 53 patients who were hospitalized at the Stroke Unit of the Neurology Department of the Yuzuncu Yil University in Van, Turkey, from January 2008 to June 2009, were registered for the study. They had no previous history of stroke, and were treated and monitored for the first time due to ischaemic or haemorrhagic stroke. However, of the patients enrolled in the study, 4 died before control examination, and 9 patients were excluded as they did not return for control.

The study, as such, was based on 40 patients (20 male, and 20 female). Among the patients, 33 had ischaemic stroke, and 7 had haemorrhagic stroke. The initial electrodiagnostic tests, including nerve conduction test (NCT), were performed within the first 3 days of the stroke (the acute stage), and control examination were performed around 45-50 days following the stroke (the sub-acute stage). The EN developments were investigated during both the stages.

Patients hospitalised for stroke, lesion localisation and type of stroke were determined through cranial computerised tomography (CT) and/or cranial magnetic resonance imaging (MRI).

Muscle strength loss was evaluated using MRC scale. Patients were evaluated and were divided into two groups: MRC score 2/5 or below, and MRC score of 3/5 or above (Table). The two groups had 20 patients each which was a coincidence.

The first examinations of enrolled patients were performed within the first 3 days after the clinical occurrence, and control examinations were done 45 to 50 days afterwards. The National Institute of Health Stroke Scale (NIHSS) was used for the evaluation of neurological deficits in the patients the two stages of the study.[4] Inclusion criteria for the study; specified patients admitted to hospital on the same day of experiencing an ischaemic or haemorrhagic stroke; patients with no disease that could be a risk factor for developing EN, with normal range of thyroid function test, vitamin B-12 and folic acid levels; patients without history of trauma to extremities or previous experiences of transient stroke attack; and patients having symptoms of stroke for more than 24 hours, but excluding a transient attack.

Table: MRC scale for stroke assessment.

0. No contraction
1. Contraction, no active movement .
2. Active movement that cannot overcome gravity
3. Active movement that can overcome gravity
4. Active movement against gravity and resistance
5. Normal muscle strength

MRC: Medical Research Council.

Nerve conduction tests were performed to diagnose EN. The most common EN were CTS. Clinical suspicion of EN led to the application of Tinel's Sign and Phalen's Test for CTS, and Wartenberg's Sign, Froment's Sign for cubital tunnel syndrome.

Electrophysiological experiments were performed at the electro-myography (EMG) laboratory of our hospital. Clinically, paretic and non-paretic, median nerve, ulnar nerve, radial nerve sensory and motor conductions in upper extremities were examined. Peroneal nerve motor and sensory conductions, tibial nerve motor conduction and sural nerve sensory conduction were also examined. All examinations were conducted at normal room temperature.

Statistical analysis was performed using computer with paired samples test, student t-test using Statistical Package for the Social Sciences programme version 15. Statistical significances level of 5% and 1% were used. Approval from the hospital's Ethics Committee was obtained for the study.

Results

Among the 40 patients 20 (50%) were females, and 20 (50%) were males, with a mean age of 53.4 ± 15.4 years (range 22 to 76 years). The mean baseline NIHSS was 11.6 ± 2.8 in the group with MRC scores ≤ 2/5, and was 7.3 ± 3.2 in the group with MRC score ≥ 3/5 (considered as a control group). There was a significant statistical difference in the NIHSS scores between the two groups (P = 0.001). The mean baseline NIHSS was 6.7 ± 2.27 in the group with MRC scores of < 3/5 and was 2.85 ± 1.6 in the control group.

While 38 (95%) patients used their right hands in daily life, 2 (5%) used their left hands. Thirty-three (82.5%) patients had cerebral infarction and 7 (17.5%) had cerebral haemorrhage. Nineteen (47.5%) patients had right hemiparesis and 21 (52.5%) had left hemiparesis. Patients were divided into 2 groups according to their MRC scores of ≤ 2/5 and ≥ 3/5 at the first neurological examination. Initial NIHSS scores of the patients were also determined. During the control stage — 45 to 50 days later, MRC and NIHSS scores were re-evaluated. The group with initial MRC scores of ≤ 2/5, with the mean baseline NIHSS being 11.6 ± 2.8, it was determined as 7.3 ± 3.2 at control. The difference between the baseline and control NIHSS scores was found to be statistically significant (P = 0.001). This difference was considered to be due to the improvement in

the NIHSS scores of patients except their motor strength.

In terms of nerve conduction tests in the control stage, patients with initial MRC score of ≤ 2/5, CTS was located in 7 (35%) patients, cubital tunnel syndrome at elbow in 3 (15%) patients, axonal neuropathy in peroneal motor nerve in 5 (25%) patients, axonal neuropathy in median motor nerve in 3 (15%) patients, and axonal neuropathy in ulnar motor nerve in 2 (10%) patients. Development of bilateral CTS was detected in 2 (10%) patients, and CTS in the healthy upper extremities in the other 2 (10%) were detected.

In control studies for nerve conduction patients with MRC score of ≥ 3/5, CTS was detected on the healthy side in 4 (20%) patients and on the affected side in 3 (15%) patients.

Discussion

Stroke remains an important condition worldwide, since it is one of the most frequent causes of death, needs hospitalised treatment and leads to long-term disability.[5] Therefore, prevention, early diagnosis and treatment of post-stroke complications are extremely important.[6]

Another important complication of sub-acute and chronic period is the development of EN. The EN represents a group of peripheral nerve disorders that are characterised by pain, parestesis and/or loss of function of nerves as a result of chronic compression along the route of peripheral nerves.[7]

In upper extremities, the most frequently encountered EN is CTS. 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.[8,9] In our study, out of 20 patients, 7 (35%) were determined to have CTS in the affected upper extremity. None of these patients was mobilised until control days, and they did not use their upper extremities in daily life. In literature, there are publications consistent with the results of our study.[9] Twenty patients with MRC score of ≤ 2/5 were included in a study conducted by Kabayel et al.[9] It reported median nerve neuropathy in the wrist in 7 patients, ulnar nerve neuropathy in 5 patients, peroneal nerve neuropathy at the level of fibular head in 7 patients on the paretic side. However, unlike our study, Kabayel et al also studied prolongation in distal latencies and reductions in conduction velocities. Our study monitored EN development as a complication in patients with stroke in the acute stages.[9]

In our study, while the frequency of paretic entrapment and axonal neuropathy findings on the paretic side increased in the patient group with severe paresis, no axonal neuropathy symptoms were observed on the paretic side in the group of patient with mild paresis - MRC score of ≥ 3/5 who could be mobilized - concluding that CTS developed in the non-paretic upper extremities more frequently. In our study, EN development was investigated in patients in both acute and sub-acute stages.

Akyuz et al[10] conducted a median and ulnar motor and sensory nerve conduction study in healthy and hemiplegic upper extremities in 50 patients. Electroneurography studies revealed no significant reductions in compound muscle action potential (CMAP) on the plegic side compared to the healthy side. They didn't find a significant correlation between nerve conduction and clinical findings. Consistent with that study, there was a statistically significant reduction in CMAP on the affected side in the group of patients with MRC score of ≤ 2/5 in our study. Also consistent with Akyuz et al, our study detected no significant changes in motor or sensory nerve conduction velocities and in distal latency values.

In our study, during conduction controls, we detected CTS in the healthy side in 4 patients, and these were the only ones who had used supporting devices for mobilisation purposes. The 16 other patients with MRC score of ≥ 3/5 did not use supporting device, and, of these patients, only three were detected to have CTS in the wrist on the affected side. In none of these patients, the development of EN or axonal neuropathy findings were observed in the healthy side. Sato and et al[11] 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 conduction velocities, sensory nerve action potentials, 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.

After CTS, the second most frequently seen EN in the upper extremity was the cubital tunnel syndrome. In our group of patients with MRC score of ≤ 2/5, cubital tunnel syndrome was seen in 3 (15%) patients, and significant reductions were detected in the amplitude values in ulnar motor nerve control studies (P = 0.01). No significant changes were observed in conduction velocities and distal latency values, even though sub-clinical conduction studies confirmed the findings in the 3 patients. Chuman[12] reported the frequency of cubital tunnel syndrome in immobilised patients in various hospitals as 7 (23%) patients, and suggested that it could have been due to the ulnar nerve compression that is

associated with immobilisation. In long-term studies, it is noted that this rate could be higher.[12]

The most frequent EN in lower extremities is peroneal nerve neuropathy.8 In our study, peroneal axonal neuropathy was found in 3 patients, and axonal neuropathy findings in the affected side involving all motor nerves were detected. Peroneal nerve neuropathy is more often seen in males and at younger ages. Most frequently encountered reasons are long-term bed dependency and surgical interventions.[3] Other important risk factors for the development of peroneal neuropathy are external compression and entrapments.[13] Entrapment occurs as a position-dependent manner in stupor, coma or under general anaesthesia, as well as an occupational disorder during kneeling or bending.[7] Tsur studied peroneal nerve involvement symptoms in 38 patients between 12-73 days after the first stroke.[14] The development of foot drop on affected side was observed. Significant impairment in amplitudes and distal latencies of motor and sensory nerves in affected side compared to the healthy side was reported.[14] In our study, significant amplitude loss was detected in motor and sensory branches of the peroneal nerve in the patient group with severe paresis ($P = 0.03$). There were no symptoms of peroneal nerve involvement in paretic or healthy sides in the group of mobilised patients with mild paresis.

Conclusion

Entrapment neuropathy may develop, and, along with this, axonal neuropathy symptoms may occur in frequent entrapment regions of the affected extremities in patients with severe paresis. Besides, any increase in the development frequency of EN, particularly of CTS, in the non-affected extremities in patients with mild paresis should also be monitored, especially if there is a use of supporting device for mobilisation purposes.

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