

The electrophysiological recording of the carpal tunnel syndrome exaggerated in the hypertensive patients

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Objective The disturbance of blood flow (BF) in the intrinsic microvasculature of the median nerve is closely involved in the entrapment of the median nerve. The chronic hypertension (CHt) causes interference with the endoneurial microcirculation and may lead to endoneurial edema and ischemic changes in the nerves.

Methods Thirty two patients, 6 males and 26 females, aged (41.5 ± 10.2) were elected to study the change in the severity of bilateral carpal tunnel syndrome (CTS) before and after suffering of CHt. All patients included in this study were subjected to the electroneurography (ENG).

Results The effect of CHt on median nerve compression causes worse severity, where the effect of CHt is indicated by the severity of the CTS and the recording of nerve conduction studies. All the elected patients diagnosed with CHt in these studies are becoming more severe of CTS after 4 months. The severity of the syndrome from mild or moderate become severe in some cases after the recording of CHt. Very high significant difference ($P < 0.05$) of the sensory and motor nerve conduction study's parameters as distal sensory and motor latency, sensory and motor conduction velocity and amplitude between the two recordings, before and after diagnosis of CHt.

Conclusion The severity of entrapment of median N (CTS) affected worse after the patients suffering from the chronic hypertensive.

Keywords carpal tunnel syndrome, chronic hypertension, blood pressure, blood low, electroneurography

Introduction

The vascular blood supply to the median nerve primarily distally by the superficial Palmar arch and proximally by the ulnar artery at wrist but several branches existing from the median and radial arteries. The median nerve requires a continuous supply of oxygen from the surrounding blood vessels to work decently. The peripheral nerves have two functionally independent vascular systems – extrinsic and intrinsic system. The extrinsic system is composed of vessels that originate from nearby arteries and veins and is responsible for supplying blood and nutrients to the outer layer of the intrinsic system in the epineurium of the nerve.¹ The intrinsic system consists of the epineurial, perineurial and endoneurial plexuses. Epineurial edema appearing before any leakage occurred from the endoneurial vessels.²

The vessels of the epineurium layer are more effected than the vessels of the endoneurium layer to trauma of the nerve.³ The capillary of endoneurium layer play an important role in the protection of nerve fibers in the fascicle and maintains in constant environment similar to the blood–brain barrier in the brain, the blood–nerve–barrier form by tight junctions of the endoneurial capillary, and impermeable to a macromolecule, the endoneurial fluid tissue pressure in the fascicle is slightly positive.³ The disturbance of blood microcirculation and endoneurial edema related to the event of neurological signs when the peripheral nerves entrapment.⁴ The endoneurial space contains no lymphatic vessel, therefore, there may be problems when edema occurs in the endoneurial space. The compression nerve may lead to endoneurial edema. Following the fascicular pressure increases and causes interference with the endoneurial blood flow (BF).⁵ The median nerve compression of the carpal tunnel syndrome (CTS) was caused median nerve dysfunction that is due to localized interference of microvascular function and the structure of nerve fiber changes.³

The most common nerve compression syndrome is CTS. The result of median nerve entrapment was at the wrist.⁶ The blood vessels with the severe CTS increases in size and adapts to the disruption of BF caused by the nerve compression, which has been observed as a larger pulsatile signal that is not observed in healthy individuals.¹ Several workers had detected the neural dysfunction induced by breakage of these blood–nerve barrier by the endoneurial edema. The role of pressure produced by entrapment of median nerve was a controversial issue.²

This paper indicates that the CTS was affected by chronic hypertension (CHt). The severity of the CTS increased after they were affected by CHt. The increase in blood pressure leads to the intraneurial edema that destroys the nerve fibers.

Materials and Methods

The beginning of this work since 2013, lasted until the February 2017, where it was noted that more than hundred patients complained from entrapment and compression of the median nerve at the wrist was diagnosed planning of the median nerve and their treatment and recovery of the signs and symptoms. The CTS patients' return worsened severely after few months from the diagnosis of chronic hypertensive.

Thirty two patients, 6 males and 26 females, aged (41.5 ± 10.2) are elected to study the change in the severity of bilateral CTSs before the suffering of the hypertension and after the diagnosis of CHt; all patients included in this study were chosen according to the criteria as:

1. A study between the first and second nerve conduction in all elective patients was at least 10 months to 22 months, including that of at least 4 months for the detection of CHt.

2. Exclude all the patients with a history of diabetes, uremia, and blood disease as thalassemia, nutrition and metabolism disorders, no evidence of peripheral neuropathy or any of the known causes of exaggerating the severity of CTS.
3. There were no history occupational and accidental trauma, neither have a manual work that exaggerate of the severity of CTS.
4. Patients complained of bilateral CTS cleared the findings of the neurological examination and the results in the nerve conduction study of bilateral hand.

This study confirms a problem with the median nerve showing confirmatory evidence for a diagnosis of CTS in conjunction with the nerve conduction studies employed by Nicolet biomedical (Viking Quest Neurodiagnostic EMG system) to all the elected patients in to time before and after diagnosis of chronic hypertensive including the sensory action potential of bilateral median nerves (include distal motor latency, amplitude and sensory conduction velocity) and the motor action potential of bilateral median nerves (include distal motor latency, motor conduction velocity).

Statistical analysis was performed with an SPSS package (version 21) using a paired-samples T-test in comparison with the means for analysis as a retrospective study.

Result

This paper shows the effect of CHt on median nerve compression, where the effect of CHt is indicated by the severity of clinical signs in the entrapment of median nerve at CTS with the recording of nerve conduction studies changes. Patients were selected for research according to the criteria as that it has neither manual work not chronic diseases causing delay in the function of nerve trunk.

Thirty two patients, 6 males and 26 females, aged (41.5 ± 10.2) referring to nerve conduction studies by different specialist (neurologist, rheumatologist) after the finding of signs and symptoms of CTS and positive of special tests at the wrist as a Phalen's test, Tinel's sign and Finkelstein's test.

In this research, nerve conduction studies of sensory action potential and motor action potential for two times were performed. First studies in the diagnosis of CTS were management with recovery without surgical interference, return signs

of CTS after affected by CHt, and their second study from 10 to 22 months from the first study and including that of at least 4 months after the discovery of CHt. All the elected patients to these studies had become more severity of CTS after 4 months from the time of diagnosis of CHt. These severity of the syndrome changed from mild to moderate in some cases, and in some cases from moderate to very severe after the complication of CHt.

The sensory parameters (distal sensory latency DSL, sensory amplitude SA and sensory conduction velocity SNCV) of the median studies were measured in all 32 patients. The recorded parameters were prolongation in distal sensory latency (DSL), reduction of sensory amplitude (S. Amp) and sensory nerve conduction velocity (SNCV). In comparison between the first study before the hypertension and the second study after discovering of CHt, statistically; there was a significant difference of these parameters between these 2 groups ($P < 0.05$). These findings are given in Table 1.

The motor nerve conduction studies show the greatest effect of the compression of the median nerve by the chronic hypertensive. These effects were explained by recording prolongation in different distal motor latency DML and by the reduction of motor nerve conduction velocity MNCV of 32 patients in comparison between the first study before the detection of CHt and second study after the discovery of CHt. There was a significant difference of these parameters between these 2 groups ($P < 0.05$). These findings are given in Table 2.

All the elected patients in these studies became more severe of CTS after 4 months from diagnosed with CHt. These severity of the syndrome changed from mild to moderate and in some cases changed from moderate to very severe after the recording of CHt.

Discussion

The CTS patients complained by median nerve dysfunction due to localized interference with microvascular disturbance led to fluid pressure of endonurial tissue elevated. The increased pressure in the tunnel caused the initial changes by obstruction to the venous return from the funicular blocked venous outflow causing the nerve to be hypervascularized and intrafunicular edematous,⁷ and an increase in intrafunicular pressure, which imperil destroys nerve fibers by impairing the blood supply.² The risk for elevated pressure within

Table 1. The sensory parameter changes

Parameter of median nerve	Number of patients	Time of calculate	Calculation of right side Mean ± SD	Calculation of left side Mean ± SD
DSL	32	Before C.H.	2.9 ± 0.22	2.98 ± 0.28
		After C.H.	3.9 ± 0.99	3.62 ± 0.61
Sensory amplitude	32	Before C.H.	14.78 ± 5.97	16.25 ± 10.49
		After C.H.	10.44 ± 6.10	10.34 ± 4.55
SNCV	32	Before C.H.	39.87 ± 2.95	38.72 ± 3.27
		After C.H.	30.90 ± 6.32	33.75 ± 4.66
P-value for DSL			0.000	0.000
P-value for S. Amp			0.008	0.006
P-value for SNCV			0.000	0.000

C.H., Chronic hypertension; P-value, significant if more than 0.05.

Table 2. The motor parameter changes

Parameter of median nerve	Number of patients	Time of calculating	Calculation of right side Mean \pm SD	Calculation of left side Mean \pm SD
DML	32	Before	4.73 \pm 0.45	4.78 \pm 0.42
		After	6.10 \pm 1.54	5.74 \pm 1.22
MNCV	32	Before	61.03 \pm 4.56	62.38 \pm 4.12
		After	51.93 \pm 9.18	54.81 \pm 8.79
P-value for DML			0.000	0.000
P-value for MNCV			0.000	0.000

C.H., Chronic hypertension; P-value, significant if more than 0.05.

the intraneural has been linked to a nerve dysfunction.⁸ These mechanical pressures led to structural changes, which followed in the CTS, and excessive pressure can constrict BF causes nerve damage.^{9,10}

Hypertension, on the median nerve as a part of peripheral nervous system alterations in endoneural blood vessels can cause morphological and morphometric changes. Disturbance of BF and intraneural edema with mechanical compression to the surrounding tissues closely involve in the appearance of neurological symptoms when the entrapment of median nerve,^{3,4} and these were affected by endoneurial continuity and the myelin formed Schwann cell. The pressure of compression on the median nerve and the relative roles of CHt can tolerate the nerve with greater force, if applied slowly, these led to nerve ischemia. Pressure will decrease intraneural and with the magnitude of duration of CTS extent of intraneural edema. The pressure of endoneurial fluid had become elevated and caused the increase of vascular permeability of endoneurium (surrounds nerve axon fibers). Because there is no lymph draining in endoneurium layer.¹ High interstitial fluid pressure causes the capillaries to collapse and limits BF to the nerve, resulting in the symptoms of numbness, tingling and weakness.¹¹ These may lead to the damage of axon transport of the median nerve. These complications of chronic pressure lead to perinodal demyelination of median nerve, and chronic disturbance and compression of function of the nerve lead to severe atrophy of all muscles. The supply by median nerve especially abductor pollicis brevis muscle can explain these changes by the severity with the complication of CTS after suffering from CHt.

These responses vary with severity, and these were evident in the studies of conduction sensory nerve where it was highly with significant difference between the first sensory studies of patients and after recording of CHt. These significant differences explain the prolonged distal sensory latency, high amplitude and vary in the loss of conduction velocity in comparison between first and second studies. These differences of highly significant motor nerve conduction studies recorded in these patients were compared between first and second studies.

The relationship between the severity differs of electrodiagnostic studies of the median nerve and major clinical events of CTS in these patients with two studies include wasting of muscles, pain in the distribution of the median nerve. Other signs and symptoms have been assessed the subsequent highest severity of incidence after the suffering of CHt. These similar results were recorded by some authors.¹²⁻¹⁴ Most of studies have been conducted about the effect of CTS in BF of microvascular with increase the fluid pressure of endoneurial tissue.¹⁵ A few studies indicated the effect of CHt in increase with the severity of CTS.^{12,16} Some studies deal with the effect of antihypertensive drugs with the increase of severity of CTS.^{17,18} However, in this study, we recorded the severity of CTS in CHt patients with different type of antihypertensive drugs.

Conclusion

CTS by compression of median nerve increases the pressure and disruption of BF. After chronic increase of the blood pressure, these changes become very severe. The severity of CTS becomes affected worse after the patient becomes suffering from the chronic hypertensive.

Recommendations

1. In the recording of the pain or paresthesia in both hands of the CHt patients, cannot exclude the CTS of different causes, type of antihypertensive drugs or the effect of disturbance for a long time in BF of median nerve, and advice the patients to do a nerve conduction study.
2. The study must be performed with the biopsy of the median nerve at the site of the entrapment of nerve in CTS to study the micro-anatomy affected by increase the blood pressure an extrinsic and an intrinsic microcirculation together with a severity recording by the nerve conduction study.

Conflict of Interest

None. ■

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