Prevalence and Associated Factors of Slowed Forearm Median Motor and Sensory Conduction Velocity in Carpal Tunnel Syndrome among Thai Patients

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ABSTRACT

Objective: This study aimed to investigate the prevalence and relationship between the associated factors and slowed forearm median motor and sensory conduction velocity (FMMCV and FMSCV) in the carpal tunnel syndrome (CTS).

Methods: The study was conducted from June 2012 to March 2014. There was a total of 69 CTS hands ($n = 38$) and 13 non-CTS hands ($n = 10$) from 82 patients' hands ($n = 48$). The slowed FMMCV and FMSCV were determined by less than 50 m/s. Demographic and clinical characteristics, FMMCV, FMSCV, and electrodiagnostic (EDX) findings related to the slowed FMMCV and FMSCV were compared and analyzed.

Results: The prevalence of slowed FMMCV in CTS was 31.6% and slowed FMSCV was 23.7%, respectively. Correlation between EDX parameters and slowed FMMCV were significantly correlated to the absence of compound muscle action potential (CMAP) amplitude (p-value = 0.006). For the slowed FMSCV, the small sensory nerve action potential (SNAP) amplitude also demonstrated significant correlation (p-value < 0.001). Among FMMCV, the occurrence of retrograde axonal atrophy (RAA) with retrograde conduction slowing (RCS) was significantly associated with the slowed FMMCV (OR 2.100; 95% CI 0.434-3.766; p-value = 0.013). For FMSCV, an increase of small SNAP amplitudes was significantly associated with slowed FMSCV (OR 4.210; 95% CI 2.031-6.390; p-value < 0.001).

Conclusion: These results support the hypothesis that slowed FMMCV is significantly ascribed to RAA with RCS and warrants further investigation into the role of the mechanism of slowed FMSCV in CTS. Furthermore, this study supports the findings of other studies that the damage to nerve function does not occur only in segments distal to the site of injury or compression.

Keywords: Forearm median motor conduction velocity, forearm median sensory conduction velocity, carpal tunnel syndrome, retrograde axonal atrophy, retrograde conduction slowing

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INTRODUCTION

Median wrist-palm conduction study typically demonstrates a decrease in conduction velocity (CV) or prolonged conduction time in patients with carpal tunnel syndrome (CTS).[1-5] Occasionally, some patients have slowed forearm median motor CV (FMMCV) or proximal conduction slowing[4,6-9] which is ascribed to two possible mechanisms. One is a either conduction block (CB) or slowing of the fastest myelinating fibers in the carpal tunnel, measured by a ratio of compound muscle action potential (CMAP) amplitude at wrist/palm

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stimulation or wrist-palm ratio (W/P ratio) which is less than 0.5 - 0.7.[4-7,9,10] The other mechanism is retrograde axonal atrophy (RAA) with retrograde conduction slowing (RCS), which may result in an increase in electrical resistance and a decrease in CV.[5-7,10,13] Several studies demonstrated that RCS occurs in humans, and that it is the primary cause of slowed FMMCV.[6,7,14] However, those studies included CTS patients whose FMMCV were < 50 m/s.[6-8,10] Therefore, some authors have argued that RCS occurs only in patients with CTS who have slowed FMMCV, low-amplitude CMAP, or markedly decreased wrist-palm median motor CV (W-P MMCV).[15-17] However, some studies demonstrated that the reduction of FMMCV did not parallel the marked decreases of W-P MMCV in CTS, which implied focal conduction slowing over the wrist is not the primary cause of RCS.[4,5,7,8,10,14,18] Furthermore, previous studies suggested that a reduction in CMAP amplitudes is not likely to cause slowed FMMCV in CTS.[14,18,19] Recently, Chang *et al* found that there was significantly greater difference and poor consistency between direct and indirect forearm mixed nerve CV in CTS. This finding suggested both direct and indirect forearm mixed nerve CV which represented CV from different nerve fibers. Hence, there is controversy over CV from nerve fibers without damage in CTS. Finally, the role of electrodiagnostic (EDX) parameters or techniques in evaluation of slowed FMMCV in CTS and the occurrence of RAA and RCS is still uncertain.[4,5,14] There has not been any recent study on the role of slowed FMSCV in CTS, because the mixed nerve CV did not clarify the mechanism. To confirm the role of the mechanism of slowed FMMCV, and to understand the phenomenon of the mechanism of slowed FMSCV are essential. This study aimed to investigate the prevalence and relationship between the associated factors and slowed FMMCV and FMSCV in CTS among Thai patients.

MATERIALS AND METHODS

In the lower northern region of Thailand, a total of 96 patients' hands from 48 participants were recruited in this study, conducted from June 2012 to March 2014. They were obtained from the EDX laboratory of the Faculty of Medicine at Naresuan University. All participants' hands were divided in two groups. Group I was the CTS group. The patients' hands were diagnosed with CTS using clinical and EDX criteria. The second group (II) was the control group, in which patients' hands were diagnosed having no clinical and EDX evidence of CTS, who served as controls.

For the CTS group, inclusion criteria were according to both clinical diagnosis of CTS and standard EDX criteria.

The clinical diagnosis of CTS was based on one or more of the following: (i) numbness, tingling pain, or paresthesia in median nerve distribution; (ii) precipitation of these symptoms by repetitive hand activities which could be relieved by resting, rubbing, and shaking of the hand; and (iii) nocturnal awakening by the sensory symptoms. A positive Tinel's or Phalen's sign might have supported the diagnosis, but it was not necessary. All CTS hands demonstrated median neuropathy at the wrist confirmed by the presence of one or more of the following standard EDX criteria: (i) prolonged orthodromic distal motor latency (DML) to the abductor pollicis bravis (APB) > 4.2 milliseconds (ms) during stimulation over the wrist 8 cm proximal to the active electrode, (ii) a prolonged antidromic distal sensory latency (DSL) to the second or third digit > 2.8 ms during stimulation over the wrist 14 cm proximal to the active electrode, and (iii) prolonged antidromic wrist-palm sensory conduction time (W-P SCT) > 1.8 ms at a distance of 8 cm.[4,5,14,19] The slowed FMMCV and FMSCV in CTS were determined by less than 50 m/s.[6-8,10,14] According to the EDX criteria of CTS, these EDX parameters were applied to indicate the mechanism of slowed FMMCV including CB, RAA with RCS, and demyelination as follows: (i) CB was defined as the W/P ratio, a ratio of CMAP amplitude at wrist/palm stimulation < 0.7 or percentage reductions of CMAP amplitudes during stimulation over the wrist compared with the palm > 30%, (ii) RAA with RCS was performed with a small or absent CMAP amplitude combined with FMMCV < 50 m/s, and (iii) demyelination was represented

by prolonged DML and FMMCV < 50 m/s, respectively.[2-5,11,14] Individuals with a history or physical findings suggestive of a neuromuscular disorder other than CTS (e.g. polyneuropathy or hereditary neuropathy), a history of wrist surgery or trauma, and any incomplete EDX tests were excluded. According to variability of median nerve distribution, patients with Martin-Gruber anastomosis were also excluded because this anastomosis would confound interpretation of FMMCV.[5] Fig 1 a flow chart has been shown of all recruited participants' hands.

The EDX studies were performed using a Micromed electromyography machine. Surface recording and stimulation were carried out for all studies. Recording electrodes were two adhesive touch proof electrodes for motor and sensory NCSs of median and ulnar nerves. The skin temperature of the hand was maintained at or above 32°C. All of the EDX techniques used for examination were applied according to AAEM (American Association of Electrodiagnostic Medicine) minimonograph #26: the electrodiagnosis of carpal tunnel syndrome[11] and several studies.[4-8,10,14,18,19] Especially, the DSL or onset sensory latency was used to assess the sensory conduction time. All of the sensory nerve action potential (SNAP) amplitudes were demonstrated with baseline to peak characteristics. The reference values were derived from the control group consisting of 13 asymptomatic, normal EDX hands tested by the same physiatrist and in the same laboratory.

The study was approved by the Ethics Committee of the Naresuan University Institutional Review Board (IRB No. 230/57).

Data collected were analysed using SPSS, version 17.0 to calculate the descriptive data. Comparison of demographic data and all EDX parameters between CTS and normal hands were evaluated by Mann-Whitney U, Fisher's exact or Chi-square test. Normal limits of CMAP and SNAP amplitudes were applied beyond 2.5 SD (standard deviation) from the controls, as cut-off values in CTS. Spearman correlation coefficient and binary logistic regression analysis were applied to determine independent correlation and association between the EDX findings and slowed FMMCV and FMSCV in CTS. Odds ratio (OR) with a 95% confidence interval (95% CI) was

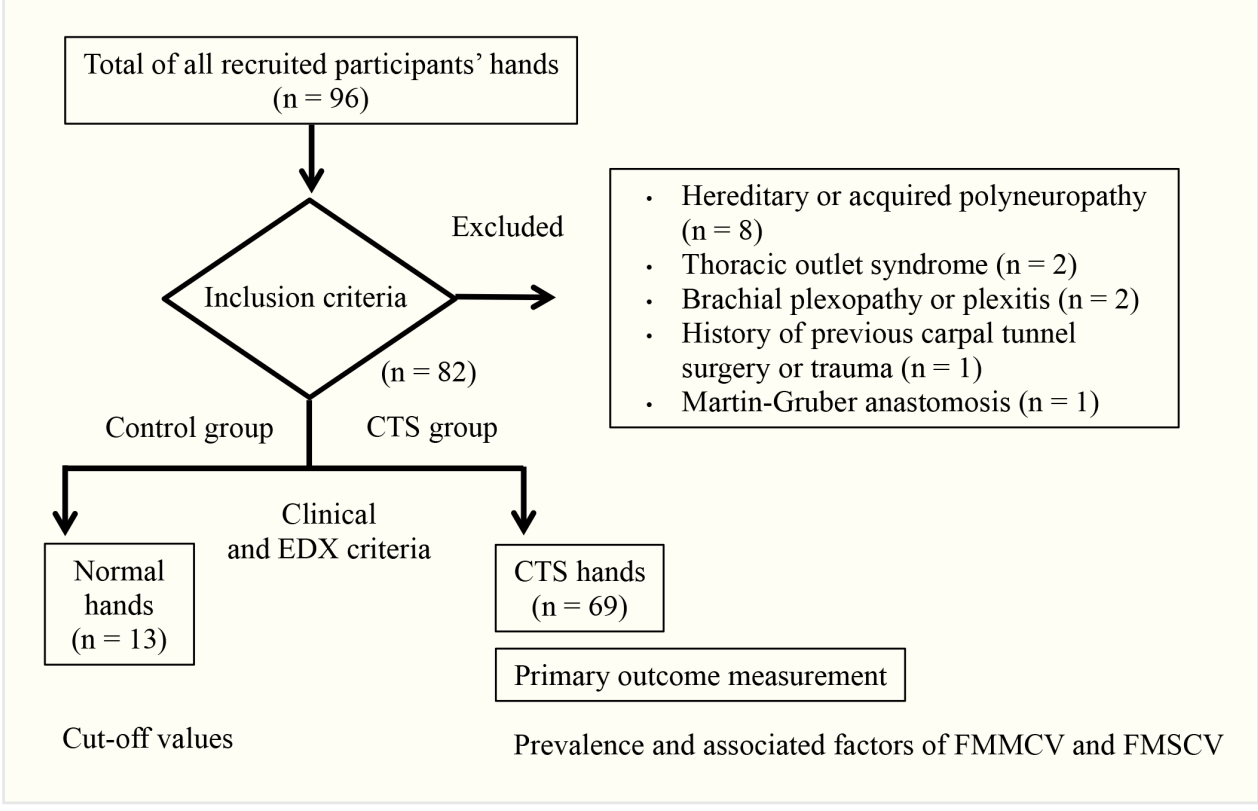

Fig 1. Flow chart of all recruited participants' hands.

TABLE 1. Comparison of the demographic characteristics and electrodiagnostic findings between the CTS patients and controls (N = 48).

	CTS (*n* = 38)	Controls (*n* = 10)	p-value
Hands (*n*)	69	13	
Age (years; mean ± SD)	56.1±12.4	53.0±7.8	0.309
Female/male	56/13	8/5	0.146
Right/left side	36/33	5/8	0.547
BMI (kg/m^2; mean ± SD)	23.5±3.6	24.2±3.5	0.572
Median motor NCS			
DML (ms)	5.7±1.6	3.4±0.3	<0.001
CMAP (mV)	5.8±3.0	9.3±4.7	0.012
W-P motor CV (m/s)	27.4±12.1	61.8±12.7	<0.001
No. of W/P ratio < 0.7	28	0	0.003
FMMCV (m/s)	52.3±28.0	53.0±4.7	0.789
Median sensory NCS			
DSL (ms)	4.0±1.1	2.5±0.2	<0.001
SNAP (μV)	28.9±13.7	46.6±16.8	0.001
W-P SCT (ms)	5.7±1.6	1.6±0.3	<0.001
W-P sensory CV (m/s)	32.6±10.5	57.4±8.2	<0.001
FMSCV (m/s)	60.0±29.6	61.5±7.6	0.575
Ulnar NCS			
FUMCV (m/s)	60.6±8.5	63.1±8.9	0.258
FUSCV (m/s)	66.3±11.7	67.9±10.2	0.521

CTS: carpal tunnel syndrome, BMI: body mass index, NCS: nerve conduction study, DML: distal motor latency, CMAP: compound muscle action potential, W-P motor CV: wrist-palm motor conduction velocity, No. of W/P ratio: a ratio of CMAP amplitude at wrist/palm stimulation, FMMCV: forearm median motor conduction velocity, DSL: distal sensory latency, SNAP: sensory nerve action potential, W-P SCT: wrist-palm sensory conduction time, W-P sensory CV: wrist-palm sensory conduction velocity, FMSCV: forearm median sensory conduction velocity, FUMCV: forearm ulnar motor conduction velocity, FUSCV: forearm ulnar sensory conduction velocity

TABLE 2. Correlation between electrodiagnostic parameters and slowed forearm median motor conduction velocity in CTS hands (N = 69).

Parameters	r_s	95% CI		p-value
		Lower	Upper	
DML ≥ 4.3 ms	-0.084	-0.326	0.157	0.495
W-P MMCV < 50 m/s	0.013	-0.228	0.254	0.916
No. of W/P ratio < 0.7	0.059	-0.182	0.301	0.630
Absence of CMAP (mV)	0.331	0.096	0.592	0.006

DML: distal motor latency, W-P MMCV: wrist-palm median motor conduction velocity, No. of W/P ratio: a ratio of CMAP amplitude at wrist/palm stimulation, CMAP: compound muscle action potential, r_s: Spearman correlation coefficient, 95% CI: 95% confidence interval

used to measure the strength of the association. A *p*-value < 0.05 was considered statistically significant.

RESULTS

Eighty-two hands ($n = 48$) which had been clinically diagnosed with CTS, were recruited to the study. There were 10 normal subjects with a mean age of 53.0 ± 7.8 years who served as controls and 38 CTS patients with a mean age of 56.1 ± 12.4 years. For the CTS group, a total of 69 CTS hands ($n = 38$) were determined from the EDX parameters. Thirteen non-CTS hands ($n = 10$) had normal NCSs and served as controls. Sixty-two hands ($n = 31$) were demonstrated bilateral CTS and 7 hands ($n = 7$) were shown to be unilateral CTS, respectively.

Comparison between the CTS and control group showed no significant differences for age, gender, side of hands, body mass index (BMI), and forearm conduction velocity of the ulnar

TABLE 3. Correlation between electrodiagnostic parameters and slowed forearm median sensory conduction velocity in CTS hands (N = 69).

Parameters	r_s	95% CI		p-value
		Lower	Upper	
W-P SCT ≥ 1.9 ms	0.100	-0.142	0.342	0.415
DSL ≥ 2.9 ms	0.003	-0.238	0.244	0.983
W-P MSCV < 50 m/s	-0.125	-0.368	0.117	0.307
SNAP < 4.6 μV	0.659	0.525	1.057	<0.001

W-P SCT: wrist-palm sensory conduction time, DSL: distal sensory latency, W-P MSCV: wrist-palm median sensory conduction velocity, SNAP: sensory nerve action potential, r_s: Spearman correlation coefficient, 95% CI: 95% confidence interval

TABLE 4. Binary logistic regression of electrodiagnostic findings related to the slowed forearm median motor conduction velocity in CTS hands (N = 69).

Findings	B	SE	Sig	Exp (B)	95% CI	
					Lower	Upper
CB	1.289	0.518	0.624	0.254	-0.761	1.268
Demyelination	0.632	0.666	0.490	-0.460	-1.764	0.845
RAA with RCS	8.167	0.850	0.013	2.100	0.434	3.766

B: B estimate, SE: standard errors, sig: significant *p*, Exp (B): odds ratio, 95% CI: 95% confidence interval for Exp (B), CB: conduction block, RAA: retrograde axonal atrophy, RCS: retrograde conduction slowing

TABLE 5. Binary logistic regression of electrodiagnostic findings related to the slowed forearm median sensory conduction velocity in CTS hands (N = 69).

Findings	B	SE	Sig	Exp (B)	95% CI	
					Lower	Upper
W-P SCT ≥ 1.9 ms	2.455	1.116	0.421	0.898	-1.289	3.085
DSL ≥ 2.9 ms	1.016	0.738	0.983	0.016	-1.431	1.462
W-P MSCV < 50 m/s	0.354	1.039	0.318	-1.038	-3.075	0.999
SNAP < 4.6 μV	67.375	1.112	<0.001	4.210	2.031	6.390

B: B estimate, SE: standard errors, sig: significant *p*, Exp (B): odds ratio, 95% CI: 95% confidence interval for Exp (B), W-P SCT: wrist-palm sensory conduction time, DSL: distal sensory latency, SNAP: sensory nerve action potential

nerves. In particular, the means of both FMMCV and FMSCV between the CTS and control groups showed no significant differences (*p*-values = 0.789 and 0.575, respectively). Compared with the controls, the CTS group had significantly prolonged DML, DSL, and W-P SCT, with small CMAP and sensory nerve action potential (SNAP) amplitude, decreased W/P ratio, and slowed W-P motor and sensory CV (W-P MMCV and W-P MSCV). The cut-off values were 0.0 mV (or absence) for CMAP and < 4.6 μV for SNAP amplitudes, which were applied beyond 2.5 SD from the controls. These demographic characteristics and EDX findings have been summarized in Table 1. Slowed FMMCV in CTS was 31.6% (*n* = 12) and slowed FMSCV was 23.7% (*n* = 9), respectively. In the CTS group, 5 patients had bilaterally slowed FMMCV, and 2 patients had both slowed FMMCV and FMSCV. In the control group, there was no occurrence of slowed FMMCV or FMSCV in the study (Table 1).

The correlation between EDX parameters and slowed FMMCV in Table 2 showed that the absence of CMAP was significantly correlated (*p*-value = 0.006), but there were no significant correlations of prolonged DML, slowed W-P MMCV, and decreased W/P ratio (or CB) (*p*-values = 0.495, 0.916, and 0.630). Table 3 showed that the small SNAP amplitude was significantly correlated to slowed FMSCV (*p*-value < 0.001), but there were no significant correlations of either prolonged W-P SCT or DSL, and slowed W-P MSCV (*p*-values = 0.415, 0.983, and 0.307).

The binary logistic regression analysis of the EDX findings related to the slowed FMMCV and FMSCV in CTS, showed an increase of RAA with RCS significantly associated with the occurrence of slowed FMMCV (OR 2.100; 95% CI 0.434-3.766; *p*-value = 0.013). For FMSCV, an increase of small SNAP amplitude was significantly associated with the occurrence of slowed FMSCV (OR 4.210; 95% CI 2.031-6.390; *p*-value < 0.001) as summarized in Table 4 and 5.

DISCUSSION

In clinical practice, the slowed FMMCV is a common EDX finding in CTS. However, there is controversy about the pathological mechanism in humans.[5-7,15-17] Several studies have demonstrated that the occurrence of RCS is the primary cause of slowed FMMCV in the CTS.[4-8,10,14,18] Compared with the controls, the results showed that the prevalence of slowed FMMCV and FMSCV were only found in the CTS group (31.6% and 23.7 %, respectively). The prevalence of slowed FMMCV was slightly less than other previous studies (prevalence of 33.3 - 36.6%).[5,7] However, there was not enough data to compare the prevalence of slowed FMSCV with the other studies. In the control group, there was no occurrence of slowed FMMCV or FMSCV (Table 1). This result suggests that there is an association of the damage of nerve fibers between forearm and wrist segments. It again supports that the RCS really occurs in the proximal nerve fibers after distal compression in CTS.[4,5,14,18]

Table 2 showed that the absence of CMAP amplitude was significantly correlated to slowed FMMCV (*p*-value = 0.006), but there were no correlations of prolonged DML, slowed W-P MMCV, and decreased W/P ratio (or CB). These findings demonstrate that the increase of severe axonal loss occurred significantly in slowed FMMCV. This supports the occurrence of RAA with RCS.[7,18, 20-22] Although the CB demonstrated that there was no significant correlation to slowed FMMCV, it also could not rule out demyelination. However, this study did not aim to analyze the subgroups of CB, including probable CB (W/P ratio between 0.5 and 0.7) and defined CB (W/P ratio less than 0.5),[5] because the criterion of CB (W/P ratio less than 0.7) in the study was more sensitive than the probable CB, so the subgroup analysis of CB would not change the results. The small numbers of the sample size would increase the statistical type I and II errors. Nevertheless, focal CB over the wrist is not the primary cause of slowed FMMCV similar to the other studies.[4,5,7,14] This finding suggests that focal conduction abnormality over the wrist is less likely to be associated with the slowed FMMCV.

For the binary logistic regression analysis of the EDX findings related to the slowed FMMCV, there was no significant association of slowed FMMCV with CB and demyelination. However,

the increase of RAA with RCS was significantly associated with the slowed FMMCV (OR 2.100; 95% CI 0.434-3.766; *p*-value = 0.013). This result again supports that the RAA with RCS is more significantly associated with slowed FMMCV than CB and demyelination.[4,5,14,18] Although, the RAA with RCS is essential for clinical assessment and planning of treatment, diagnosis of the RAA with RCS in slowed FMMCV is very difficult because it has no more specific clinical manifestation than the CTS. Therefore, all clinicians and physiatrists should concern about is the EDX evidence of slowed FMMCV in the CTS patients, because the RAA with RCS was strongly associated with retrograde neuroaxonal degeneration in the forearm. The early surgical exploration and decompression should be considered to relieve and improve the compression neuropathy.[9,10,13]

The results of slowed FMSCV in this study showed that an increase of small SNAP amplitude was significantly associated with the occurrence of slowed FMSCV (OR 4.210; 95% CI 2.031-6.390; *p*-value < 0.001). These findings suggest that the RCS of sensory fibers is more significantly associated with axonal loss than demyelination. However, there was not enough data to clarify the real role of the mechanism of this phenomenon.

A limitation of this study was there was no gold standard of the EDX criteria to diagnose the RCS, RAA, and both mechanisms of proximal nerve injury. This study reported the EDX evidence, about using electropathogenesis to diagnose theses mechanisms as in the other studies. Although, the nerve biopsy is the extreme gold standard technique to obtain the definitive diagnosis of these conditions, it is not recommend in human study or clinical practice. It is a very invasive technique and so more useful in animal studies than human studies or clinical practice.[13,23,24] Another limitation was the small numbers of CTS patients in each subgroup of CTS patients for statistical analysis. However, there was not enough data to support the role of the mechanism of slowed FMSCV in CTS.

In conclusion, these results support the hypothesis that the slowed FMMCV can be significantly ascribed to the RAA with RCS and warrants further investigation into the role of the mechanism of slowed FMSCV in CTS. Furthermore, this study supports the findings of other studies that the damage of nerve function does not occur only in segments distal to the site of injury or compression.

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