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Original Research Article

THE HIDDEN CONNECTIONS: NEUROPHYSIOLOGICAL VARIABLES AND CO-MORBIDITIES IN CARPAL TUNNEL SYNDROME AMONG KAMRUP RURAL RESIDENTS

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Abstract

Background: Present study was envisaged to analyse neurophysiological variables in clinically diagnosed Carpal Tunnel Syndrome (CTS) patients and categorize injury into demyelinating, axonal or mixed insult on its basis. The study further evaluated co-morbid conditions (diabetes, hypothyroidism and hepatosteatosis) in CTS patients and its impact on variation of neurophysiological variables in the Kamrup Rural population. **Materials and Methods:** This was a prospective cross-sectional study in which 496 hands i.e. (248 patients) of clinically diagnosed Carpal Tunnel Syndrome (CTS) patients from Kamrup Rural were studied. Nerve Conduction Study (NCS) was conducted on median sensory & median motor nerve. Various Neurophysiological variables i.e. Sensory Nerve Action Potential (SNAP), Compound muscle action potential (CMAP), Distal Latency (sensory), distal latency (motor), Nerve Conduction Velocity (sensory), Nerve Conduction Velocity (motor) were recorded. CTS patients with comorbid conditions of diabetes, hypothyroidism, hepatosteatosis from Kamrup Rural were studied for variation of neurophysiological variables, along with CTS patients without above stated co-morbid conditions. **Result:** Out of 469 hands with CTS from Kamrup Rural, 76.54% were females and 23.46% were males. The mean duration of CTS symptoms exhibited was 6.52 ± 5.18 months. Combined abnormalities in both median sensory and median motor nerve conduction were observed in 330 hands (70.36%). Isolated abnormalities in median sensory nerve conduction were found in 128 hands (28.44%), while only 6 hands (1.2%) had abnormalities exclusively in the median motor nerve. CTS patients with predisposing diabetes disease were 25%, with hepatosteatosis were 9% and with hypothyroidism were 16% in the Kamrup Rural population. **Conclusion:** Nerve conduction studies are reliable and versatile method for diagnosis of CTS when used with clinical history. The study emphasis the importance of variation of neurophysiological variables in predisposing diseases i.e. diabetes, hypothyroidism, rheumatoid arthritis and categorization of insult to median nerve i.e. sensory, motor, axonal, demyelinating or mixed in the Kamrup Rural population.

INTRODUCTION

Carpal Tunnel Syndrome (CTS) is a prevalent form of focal entrapment neuropathy characterized by the compression of the median nerve as it passes through the carpal tunnel in the wrist. This chronic condition can lead to significant disability, manifesting primarily as numbness, tingling, and pain in the hand, particularly affecting the thumb, index, middle, and part of the ring finger. The incidence of CTS is notably higher in women, with a reported rate of approximately 506 cases per 100,000 individuals, and it presents in a gender ratio of 5:1, predominantly affecting the dominant hand first.[1,2] The first documented case of CTS was reported by Brian in 1947, highlighting its association with repetitive hand movements. The diagnosis of CTS is primarily based on clinical symptoms, which include numbness, burning sensations, and pain localized to the lateral aspect of the palm and fingers. Electrodiagnostic studies, particularly nerve conduction studies (NCS), are crucial for confirming the diagnosis, as they reveal abnormal electrophysiological findings that are indicative of median nerve dysfunction.[3-8] Clinical tests such as Tinel's and Phalen's tests may also yield positive results, serving as additional

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indicators of the syndrome.[9-11] Early diagnosis of CTS is essential to prevent progressive median nerve damage. Professions that involve repetitive hand movements, such as typing or assembly line work, are associated with a higher prevalence of CTS.[12-14] Neurophysiological assessments through NCS provide valuable insights into the condition by measuring variables such as Compound Muscle Action Potential (CMAP), Distal Latency (both sensory and motor), and Nerve Conduction Velocity (NCV).[15-17] The neurophysiological diagnosis of CTS was first established by Simpson in 1956, who demonstrated a slowing of median nerve conduction at the wrist.[18-21] It is important to note that both sensory and motor conduction may be affected, although in some cases, only one may show abnormalities.[22] Comorbid conditions significantly influence the development and severity of CTS. For instance, diabetes mellitus increases susceptibility to nerve compression,[1] while hypothyroidism and hepatosteatosis can reduce the available space within the carpal tunnel.[5,23-27] These factors—reduced tunnel space and increased pressure—contribute to the onset of CTS. The present study aims to investigate the neurophysiological patterns, severity grading, and types of nerve insult in CTS patients from the rural Kamrup region of Assam, India. This study involves the collection of various neurophysiological parameters, including CMAP, Distal Latency (sensory and motor), and Nerve Conduction Velocity (sensory and motor). By evaluating the demographic and electrophysiological profiles of these patients, the research seeks to analyze the impact of chronic comorbid diseases—specifically diabetes mellitus, hypothyroidism, and hepatosteatosis—on neurophysiological variables in individuals affected by CTS. These comorbidities were selected due to their frequent occurrence among CTS patients, highlighting the need for a comprehensive understanding of their effects on the condition. In conclusion, CTS is a significant health concern that necessitates thorough evaluation and management.[3] Understanding the interplay between neurophysiological variables and comorbid conditions is crucial for developing effective treatment strategies and improving patient outcomes.[4,5] The findings from this study may contribute valuable insights into the management of CTS, particularly in rural populations where healthcare resources may be limited.[6]

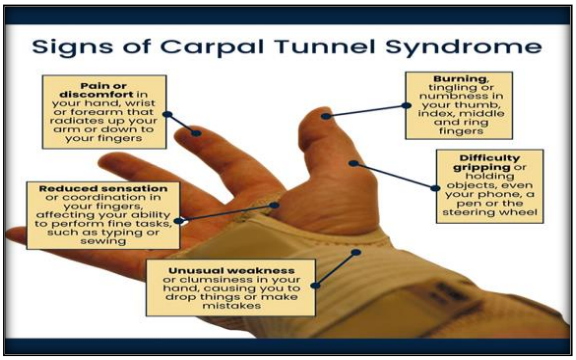

Figure 1: Showing signs of Carpal Tunnel syndrome

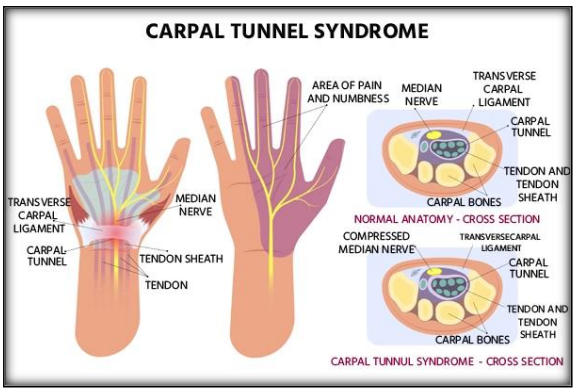

Figure 2: Depiction of Carpal Tunnel Syndrome

MATERIALS AND METHODS

The present study is a prospective cross-sectional investigation conducted in the Department of Physiology in Neurophysiology lab in Gauhati Medical College and Hospital. The study included patients with clinically confirmed carpal tunnel syndrome (CTS) who presented to the outpatient departments of, Neurology, and Orthopaedics of Gauhati Medical College and Hospital. A total of 248 CTS patients were enrolled, with clinical diagnosis following the criteria outlined by Vogt et al. These criteria included: (1) pain or paraesthesia in the hand, either related to activity or nocturnal; (2) sensory impairment in the median nerve distribution or reduced two-point discrimination; (3) isolated atrophy of the abductor pollicis brevis (APB) muscle; and (4) positive Tinel's sign or Phalen's sign. A diagnosis of CTS was suspected if a patient met the first criterion and at least one additional criterion from the list.

Clinical and laboratory features were recorded using a standardized form, and a total of 480 hands were studied for both clinical presentation and nerve conduction analysis. The nerve conduction studies (NCS) were conducted using an Medicaid NCV machine with Neurostim software to analyse the data. The specific NCS tests included measurements of (1) median distal latency (motor and sensory), (2) median sensory and compound motor action potentials (SNAP/CMAP), and (3) median nerve conduction velocity (motor and sensory). For these assessments, disc recording electrodes were used in

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mixed nerve studies, while ring electrodes were utilized in sensory studies. A ground electrode was placed between the stimulating and recording electrodes. Percutaneous supramaximal response was achieved using the same machine, with pulse durations of 0.05/0.1 milliseconds for sensory stimulation and 0.2/0.5 milliseconds for motor stimulation. Filters of 20 Hz and 2 kHz were applied during the testing.

To assess the severity of CTS, the criteria established by Hermann and Logigian (2002) were used.[7] The severity of CTS was classified as follows: (1) mild, characterized by prolongation of distal motor and sensory latency; (2) mild to moderate, indicated by latency prolongation with mild SNAP reduction; (3) moderate, signified by latency prolongation with moderate SNAP or CMAP reduction; and (4) severe, defined by either an unrecordable SNAP or a significant reduction in CMAP.[8-10]

This study also examined CTS patients with chronic comorbid conditions such as diabetes, hypothyroidism, and hepatosteatosis, in comparison to those without comorbidities.

Inclusion criteria consisted of patients aged 15 to 75 years with a clinical diagnosis of CTS. Exclusion criteria included those who refused to provide informed consent and individuals with electronically activated implants, such as cardiac pacemakers. The study was conducted in accordance with the Declaration of Helsinki and received approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrolment.

Statistical analyses were performed using SPSS version 26 (IBM Corp, Armonk, NY, USA). A Shapiro-Wilk test was conducted to assess data normality, revealing a significant deviation from normality ($p < 0.01$). Consequently, non-parametric tests were used, with results summarized as medians and interquartile ranges. The Mann-Whitney U test was employed to compare two groups, and a p-value < 0.05 was considered statistically significant, while p-values < 0.001 were considered highly significant. Pearson's correlation coefficient was used to evaluate associations between neurophysiological variables and comorbidity markers such as HbA1C, TSH, AST & ALT.

RESULTS

A prospective cross-sectional study was conducted on 248 patients diagnosed with carpal tunnel syndrome (CTS) in Gauhati Medical College and Hospital. Of the 496 hands analyzed, 469 exhibited symptoms of CTS. The mean age of the patients was 53.06 ± 14.58 years, with a significant predominance of female patients, resulting in a female-to-male ratio of 3.56:1. Among the 469 symptomatic hands, 359 (76.54%) were from female patients, while 110 (23.46%) were from male patients. The mean duration of symptoms was 6.52 ± 5.18 months, with the majority of patients (52.6%) reporting symptom duration between 6 and 12 months (129.70~130 patients).

In terms of occupation, 51% of the patients were housewives, followed by individuals engaged in dairy farms (23%), daily wage labourers (11%), and those in teaching, clerical, or other white-collar jobs (15%).

Among the 248 patients, 66 (25.5%) had diabetes, 39 (15.2%) had hypothyroidism, and 24 (9.2%) had hepatosteatosis, while 124 patients (50%) had no underlying comorbidities.

The mean HbA1C level in the diabetic patients was 7.1 ± 3.7%, while the mean AST (SGOT) (Aspartate Transaminase) and ALT (Alanine Transaminase) (SGPT) in affected individuals was 20.23 ± 12.68mg/dl and 19.78 ± 13.34mg/dl. For the patients with hypothyroidism, the mean TSH level was 5.06 ± 0.8 µIU/ml.

Regarding age distribution, the 51–60-year age group comprised the largest number of patients (79, or 30.2), followed by the 41–50-year age group (70 patients, or 29.3%). Overall, the 40–60-year age range accounted for 60% of the total patient population, indicating that middle-aged individuals are most susceptible to CTS.

In terms of neurophysiological findings, combined abnormalities in both median sensory and median motor nerve conduction were observed in 330 hands (70.36%). Isolated abnormalities in median sensory nerve conduction were found in 128 hands (28.44%), while only 6 hands (1.2%) had abnormalities exclusively in the median motor nerve.

Symptomatically, the most common presentation involved tingling, numbness, and pain in the thumb, index, and middle fingers, affecting 269 hands (57.3%). Symptoms localized to the distal ends of the index, middle, and ring fingers were reported in 135 hands (28.78), while 33 hands (7.03%) were limited to the index and middle fingers, and 29 hands (6.18%) were affected only in the thumb. There were 3 patients who complained of no pain but was having CTS.

These findings highlight the significant gender disparity, the predominant involvement of middle-aged individuals, and the importance of both sensory and motor nerve conduction studies in diagnosing CTS.[12]

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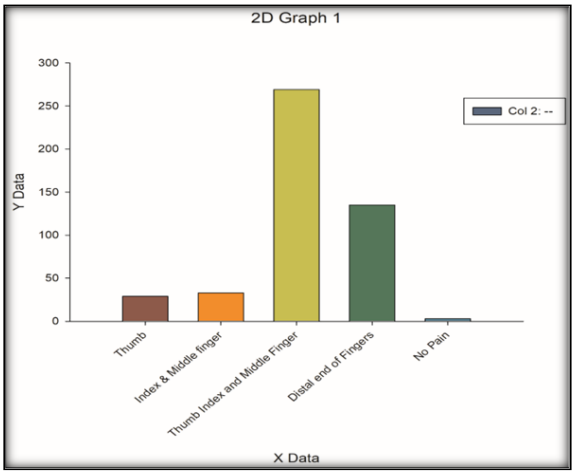

Figure:3 Showing Numbness, Tingling, Pain site in affected Carpal Tunnel Syndrome Hands.

In the present study, involving 469 symptomatic CTS hands, neurophysiological variables showed significant changes: sensory nerve action potential (SNAP) was decreased in 239 hands (50.959%), while compound muscle action potential (CMAP) decreased in 52 hands (11.1%). Additionally, an increase in distal latency (motor) in the median nerve was observed in 121 hands (24.73%), and an increase in distal latency (sensory) in the median nerve was noted in 116 hands (24.73%). A decrease in nerve conduction velocity (sensory) was recorded in 75 hands (15.99%), whereas a decrease in nerve conduction velocity (motor) was observed in 29 hands (6.18%).

In the current study, neurophysiological variables were assessed in CTS hands without any comorbid diseases (such as diabetes, hypothyroidism, or rheumatoid arthritis) and in 129 CTS hands with comorbid diabetes, accounting for 30.1% of the total. The duration of diabetes among these patients ranged from 1 to 5 years, with a mean duration of 2.37 ± 1.17 years, indicating a relatively short duration. For the CTS hands with diabetes, the median motor amplitude was recorded at 9.40 (4.75 – 12.90) mV, while the median sensory amplitude was 43.80 (26.35 - 50.10) µV. The distal latency for sensory nerves was measured at 2.59 (2.42-3.08) milliseconds, and for motor nerves, it was 4.19 (3.52 - 4.79) milliseconds. A statistically significant difference ($p < 0.001$) was found in the neurophysiological variables between CTS hands without any comorbidity and those with diabetes, as determined by the Mann-Whitney U test. Out of the 129 diabetic CTS hands studied, 38 hands were classified as Grade I (demyelinating injury), which accounts for 27.2% of the severity scale. Additionally, 12 hands were categorized as Grade II (demyelinating and axonal injury), representing 9.2%, and 4 hand was classified as Grade III (demyelinating and axonal injury), which is 1.02%. The majority, 98 hands, were categorized as Grade IV (axonal injury), making up 72.3%. In comparison, among the 213 CTS hands without any comorbid diseases, the severity scale showed that 142 hands were in Grade I (68.9%), 44 hands in Grade II (22.6%), 28 hands in Grade III (11.9%), and 20 hands in Grade IV (8.6%). Furthermore, the study identified that out of a total of 469 CTS hands, 81 hands (20.8%) had comorbid hypothyroidism. A comparison of neurophysiological variables between CTS hands with hypothyroidism and those without any comorbidities was conducted. In CTS hands with hypothyroidism, the median motor distal latency was recorded at 3.76 (3.33-4.48) milliseconds, while the median motor amplitude was measured at 13.10 (10.40-15.70) mV. The median motor nerve conduction velocity was found to be 50.07 (43.34 - 53.74) m/s. For sensory parameters, the sensory median latency was reported at 3.41 (2.58 – 3.83) milliseconds, with a median sensory amplitude of 28.10 (16.75 – 36.30) µV and a median sensory nerve conduction velocity of 38.49 (29.37 -45.47) m/s.

There was a statistically significant difference in the neurophysiological variables observed between CTS hands with hypothyroidism and those without any comorbidities (such as diabetes, hypothyroidism & Hepatosteatosis), as determined by the Mann-Whitney Test. Among the 81 CTS hands with hypothyroidism, severity grading revealed that 21 hands were classified as Grade I (demyelinating injury), accounting for 26.01%, 9 hands were in Grade II (demyelinating and axonal injury), representing 11.33%, 14 hands were categorized as Grade III (demyelinating and axonal injury), which is 17.61%, and 40 hands were classified as Grade IV (axonal injury), making up 49.23 %. In the study, 48 hands (9.79%) had hepatosteatosis out of the total 469 CTS hands. A comparison of neurophysiological variables was conducted between CTS hands with patients with hepatosteatosis and those without comorbidities. The median motor latency in CTS hands with hepatosteatosis was recorded at 3.83 (3.44-4.79) milliseconds, while the median motor amplitude was measured at 13.80 (10.80 – 18.10) mV, and the median motor nerve conduction velocity was observed to be 51.94 (45.33 – 57.14) m/s. For sensory parameters in CTS hands with comorbid hepatosteatosis the results were as follows: the median sensory latency was 3.10 (2.46- 3.85) milliseconds, the median sensory amplitude was recorded at 33.18 (19.05 – 41.60) µV, and the median sensory nerve conduction velocity was found to be 38.29 (32.75 – 45.09) m/s. A statistically significant difference was also noted in the neurophysiological variables between CTS hands with hypothyroidism and those without any comorbidities, according to the Mann-Whitney Test.

It was observed that when grading of CTS hands as per severity scale was done in CTS hands with hepatosteatosis, 22 hands were categorized in Grade I (demyelinating injury) which accounts for 46.12%, 8 hands were in Grade II (demyelinating and axonal injury) representing 14.76%, 4 hands were classified as Grade III (demyelinating and axonal injury) which is 7.8%, and 16 hands were categorized as Grade IV (axonal injury) making up 35.79%. The study

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emphasized the preponderance of axonal insult (median nerve) in CTS hands with hepatosteatosis.[28-31]

In the present study, the Pearson Correlation Test was employed to examine the relationship between neurophysiological variables and comorbidity markers, specifically HbA1C, TSH, and Liver enzymes (to establish Hepatosteatosis). In diabetic CTS patients, higher HbA1C levels were positively correlated with median motor latency and negatively correlated with median motor amplitude. Additionally, a negative correlation was found between HbA1C and sensory nerve conduction velocity (NCV). For CTS patients with hypothyroidism, TSH levels exhibited a positive correlation with both distal motor and sensory latencies, while showing a negative correlation with both motor and sensory NCV. In CTS patients with raised liver enzymes with hepatosteatosis, a negative correlation was observed with both motor and sensory NCV, as well as with sensory amplitude. These findings highlight the significant relationships between neurophysiological parameters and various comorbid conditions in CTS patients.

Table 1: Depicting Neurophysiological variables affected in CTS hands.

Parameter affected in CTS hands (n=469)	Number (Hands)	Percent (Hands)
Decrease SNAP	239	50.959
Decrease NCV (Motor)	29	6.183
Decrease NCV (Sensory)	75	15.991
Decrease CMAP	52	11.087
Increase DL (Motor)	121	25.799
Increase DL (Sensory)	116	24.733
Increase difference of DL (Sensory-Motor)	138	29.424

Table 2: Nerve conduction study in CTS hands with co-morbidity (diabetes) and CTS hands without Diabetes.

	CTS Hands with Diabetes		CTS Hands without Diabetes		p-value
	N	Median (25-75)	N	Median (25-75)	
Motor Latency (ms)	129	4.19 (3.52-4.79)	213	3.68(3.02-4.12)	<0.001
Motor amplitude (mv)	129	9.4(4.75-12.9)	213	14.4 (10.87-16.75)	<0.001
Motor NCV (m/s)	129	50.01(43.34-53.74)	213	52 (48-55.44)	<0.001
Sensory latency (ms)	41	2.59(2.42-3.08)	198	3.06 (2.41-3.76)	0.28
Sensory amplitude(µV)	41	43.8(26.35-50.1)	198	30.5 (17.38-41.3)	0.0015
Sensory NCV(m/s)	41	46(37.29-48.05)	198	40.02 (31.23-47.46)	0.068
Difference between myelinated and non-myelinated (motor) latency	129	0.84(0-1.72)	213	1.37 (0.85-1.87)	<0.001
Difference between myelinated and non myelinated (sensory) latency	129	0.780-1.72)	213	0.90 (0.5-1.76)	0.021

Table 3: Nerve conduction study in CTS hands with co-morbidity (Hypothyroidism) and CTS hands without Hypothyroidism.

	CTS Hands with Hypothyroidism		CTS Hands without Hypothyroidism (without co morbidities)		p-value
	N	Median (25-75)	N	Median (25-75)	
Motor Latency(ms)	81	3.76(3.33-4.48)	210	3.67(3.02-4.12)	0.040
Motor amplitude (mv)	81	13.1(10.4-15.7)	210	14.4 (10.87-16.75)	0.066
Motor NCV (m/s)	81	50.07(43.34-53.74)	210	52.1(48-55.44)	0.009
Sensory latency (ms)	39	3.41(2.58-3.83)	197	3.24 (2.41-3.755)	0.234
Sensory amplitude(µV)	39	28.1(16.75-36.3)	197	30.35(17.38-41.3)	0.162
Sensory NCV(m/s)	39	38.49(29.37-45.47)	197	40.26 (31.23-47.46)	0.375
Difference between myelinated and non-myelinated (motor) latency	81	1.59(1.15-2.09)	210	1.37(0.85-1.87)	0.007
Difference between myelinated and non myelinated (sensory) latency	79	0.8(0-1.55)	210	0.90(0.5-1.76)	<0.001

Table 4: Nerve conduction study in Carpal Tunnel Syndrome hands with co-Morbidity (Fatty Liver) and Carpal Tunnel Syndrome hands without any co-morbidity

	CTS Hands with Fatty Liver (Hepatosteatosis)		CTS Hands without Fatty Liver (Hepatosteatosis)(without Co morbidities)		p-value
	N	Median (25-75)	N	Median (25-75)	
Motor Latency (ms)	48	3.83(3.44-4.79)	212	3.84(3.02-4.12)	0.015
Motor amplitude (mv)	48	13.8 (10.8-18.1)	212	14.5(10.87-16.75)	0.926
Motor NCV (m/s)	48	51.94 (45.53-57.14)	212	52.8 (48-55.44)	0.967
Sensory latency (ms)	32	3.10 (2.46-3.85)	197	3.08(2.41-3.76)	0.446
Sensory amplitude(µV)	32	33.18 (19.05-41.6)	197	30.29(17.375-41.3)	0.934
Sensory NCV(m/s)	32	38.29(32.75-45.09)	197	40.03 (31.23-47.46)	0.388

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Difference between myelinated and non-myelinated (motor) latency	47	2.04(1.28-2.35)	212	1.36 (0.85-1.87)	<0.001
Difference between myelinated and non myelinated (sensory) latency	48	0.90(0-1.2)	212	0.89 (0.5-1.76)	0.072

Table 5: Showing Co-relation between Neurophysiological variables and co-morbidity markers (HbA1C, TSH, Liver enzymes)

	HBA1C		TSH		AST		ALT	
	r value	p value	r value	p value	r value	p value	r value	p value
Motor Latency (ms)	0.180	0.054	0. 215	0.068	0.044	0.706	0.048	0.698
Motor amplitude (mv)	0.026	0.799	0.076	0.530	0.088	0.438	0.102	0.388
Motor NCV (m/s)	0.005	0.968	0.098	0.446	0.161	0.148	0.059	0.634
Sensory latency (ms)	0.104	0.552	0.130	0.450	0.118	0.458	0.215	0.206
Sensory amplitude(µV)	0.058	0.739	0.334	0.046	0.130	0.401	0.380	0.021
Sensory NCV(m/s)	0.126	0.468	0.264	0.118	0.215	0.165	0.215	0.210
Difference between myelinated and non-myelinated (motor) latency	0.018	0.882	0.192	0.105	0.228	0.039	0.100	0.404
Difference between myelinated and non myelinated (sensory) latency	0.020	0.858	0.024	0. 864	0.040	0.733	0.119	0.328

DISCUSSION

Carpal Tunnel Syndrome (CTS) is a common entrapment neuropathy characterized by compression of the median nerve at the wrist.[3] Establishing an accurate diagnosis of CTS relies on a combination of clinical evaluation and neurophysiological assessment to determine the extent of median nerve impairment. In the present study, neurophysiological variables, including Sensory Nerve Action Potential (SNAP), Compound Muscle Action Potential (CMAP), distal latencies, and nerve conduction velocities, were utilized to grade the severity of CTS in a cohort of 248 patients.[22] The study population consisted of 181 females (75.4%) and 59 males (24.5%), with a predominance of females, consistent with the known higher incidence of CTS in women. Of the 469 hands analyzed, 449 were symptomatic, emphasizing the importance of nerve conduction studies in diagnosing CTS, particularly in mild cases where clinical signs and provocative tests may be inconclusive. Among the 240 CTS patients, comorbidities were identified, including diabetes in 61 patients (25.4%), hypothyroidism in 38 patients (15.8%), and hepatosteatosis in 22 patients (9.2%). The remaining 119 patients (49.5%) had no associated comorbidities. The study focused on comparing neurophysiological variables between CTS hands with hypothyroidism and those without comorbidities.[23] The results showed significantly higher median motor and sensory latencies in CTS hands with hypothyroidism compared to those without comorbidities, consistent with previous studies. Additionally, median motor and sensory amplitudes were significantly lower in CTS hands with hypothyroidism. These findings suggest that hypothyroidism can contribute to both demyelinating and axonal injury of the median nerve in CTS patients.[18] When CTS hands with hypothyroidism were categorized based on severity, 38 hands (50%) exhibited Grade IV axonal injury, 18 hands (24%) showed Grade II and III demyelinating and axonal injury, and 19 hands (25.33%) presented with Grade I demyelinating injury. The results obtained draws our attention towards the dominant phenomenon of axonal damage to the median nerve in CTS patients who have hypothyroidism. The observed impairment of both sensory and motor components of the median nerve, with a greater degree of axonal damage, is consistent with the findings of Meshram et al., who reported a mixed type of neuropathy characterized by a decrease in SNAP amplitude and median nerve conduction velocity.[21] The study also compared 121 CTS hands with diabetes to 209 hands without any comorbidities (diabetes, hypothyroidism, or hepatosteatosis). CTS hands with diabetes showed significantly higher median motor distal latency and lower median motor amplitude and nerve conduction velocity compared to those without comorbidities. However, sensory amplitude and nerve conduction velocity were significantly higher in CTS hands with diabetes,[20] while sensory distal latency was lower. This observation is attributed to the predominance of Grade IV axonal injury in diabetic CTS hands, while CTS hands without comorbidities exhibited more Grade I demyelinating injury. The study suggests that diabetic CTS hands tend to exhibit more profound electrophysiological abnormalities, with prominent demyelinating distortions in the early stages and axonal damage becoming more prominent as the disease progresses.[22] Diabetes is a known risk factor for CTS, with a higher incidence in patients with diabetes compared to the general population. When categorizing diabetic CTS hands based on severity, 84 hands (69.4%) showed Grade IV axonal injury, 5 hands (4%) exhibited Grade II and III axonal and demyelinating injury, and 32 hands (26.4%) presented with Grade I demyelinating injury. These

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results indicate that diabetes leads to more advanced forms of CTS, with prominent abnormalities in SNAP and CMAP (axonotmesis).[8] Similar findings were reported by Park et al., who observed significant decreases in CMAP and nerve conduction velocity in the median nerve of CTS hands with comorbid diabetes.[27] In the present study, 44 CTS hands (9.79%) were associated with hepatosteatosis. A comparison of neurophysiological variables between CTS hands with hepatosteatosis and those without comorbidities revealed significantly higher median motor distal latency and lower median motor amplitude and nerve conduction velocity in the hepatosteatosis group. However, for the sensory component, CTS hands with hepatosteatosis showed significantly lower sensory nerve conduction velocity, while sensory distal latency was slightly higher compared to CTS hands without comorbidities. Interestingly, sensory amplitude in the median nerve was greater in CTS hands with hepatosteatosis than in those without comorbidities. When categorizing CTS hands with hepatosteatosis based on severity, 15 hands (34.09%) exhibited Grade IV axonal injury, 20 hands (45.45%) showed Grade I demyelinating injury, and 9 hands (20.45%) presented with Grade II and III demyelinating and axonal injury. These findings suggest that CTS hands with hepatosteatosis tend to exhibit more electrophysiological abnormalities in the motor component of the median nerve, although demyelinating changes were observed in the sensory component during the early stages of the disease.[32] Similar results were reported by Mahmoud et al., who found decreased sensory and motor nerve conduction velocities, reduced SNAP amplitude, and increased distal latencies in CTS hands with comorbid hepatosteatosis.[33] The results in the present study emphasizes the importance of nerve conduction studies in diagnosing and grading the severity of CTS, particularly in the presence of comorbidities such as diabetes, hypothyroidism, and hepatosteatosis. The study highlights the significant impact of these conditions on the neurophysiological parameters of the median nerve, with varying degrees of demyelinating and axonal injury. Understanding the specific patterns of nerve dysfunction in CTS patients with comorbidities can aid in tailoring treatment strategies and predicting prognosis.

CONCLUSION

Carpal Tunnel Syndrome (CTS) patients often experience sensory motor distortions, which can be exacerbated by comorbid conditions such as hypothyroidism, diabetes, and hepatosteatosis. The presence of these comorbidities tends to amplify the neurophysiological distortions observed in CTS patients. This study underscores the importance of combining electrophysiological evaluations with clinical signs and symptoms to create a comprehensive diagnostic tool for assessing the full spectrum of CTS, particularly in patients with additional health issues. Neurophysiological variables serve as critical indicators for grading the extent of nerve damage and identifying the type of injury—whether axonal or demyelinating, and affecting sensory or motor functions—in CTS patients. Furthermore, this study explores the correlation between neurophysiological variables and changes in HbA1C, TSH, and rheumatoid factor levels. The findings advocate for an integrative approach that combines variations in neurophysiological metrics, clinical manifestations, and the grading of median nerve insult to enhance the prognosis of CTS in patients both with and without comorbidities. By understanding these relationships, healthcare providers can better tailor treatment strategies and improve patient outcomes in those suffering from CTS alongside other chronic conditions.

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