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Sleep profile and its clinical correlates in patients with carpal tunnel syndrome

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Abstract

Background & Objective: Nocturnal pain and paresthesias are common in carpal tunnel syndrome (CTS). However, there is limited literature on the impact of CTS on sleep profile. The aim of this study was to systematically analyse sleep profile in CTS and to correlate with clinical and electrophysiological features. *Methods:* Prospective evaluation of 44 patients diagnosed to have CTS was carried out using Visual Analog Scale (VAS), Functional Severity Scale (FSS) and Symptom Severity Scale (SSS) components of Boston Carpal Tunnel Questionnaire (BCTQ), Historic and objective scale (HiOb), and Quick Disabilities of the Arm, Shoulder and Hand (QuickDASH). Sleep was assessed using Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), Insomnia Severity Index (ISI) and overnight polysomnography (PSG). *Results:* Cases had significantly higher mean PSQI, ESS, ISI and STOP-BANG scores as compared to controls. PSQI, ESS, and ISI correlated significantly with FSS, SSS and QuickDASH. PSQI and ISI correlated with VAS. No significant correlation was found between sleep questionnaires and electrophysiological severity. PSG, carried out in 16 cases, showed reduced total sleep time and sleep efficacy, reduced duration of N2 and rapid eye movement (REM) sleep, and higher wake after sleep onset (WASO), wake index, and stage shift index ($p<0.05$). Sleep efficacy, sleep onset latency, WASO and wake percentage were better in those with clinical and electrophysiological severe grades of CTS.
Conclusion: CTS causes poor sleep quality, and fragmented and reduced sleep, which is under-recognised, but causes significant distress to the patients. Longitudinal alterations in sleep profile including the impact of medical/surgical intervention need to be delineated.

Keywords: Boston carpal tunnel questionnaire, carpal tunnel syndrome, sleep quality, historic and objective scale, insomnia severity index, visual analogue scale

INTRODUCTION

Carpal tunnel syndrome (CTS) is an entrapment neuropathy that is caused by compression of the median nerve at the level of carpal tunnel in the wrist.[1] Repetitive and forceful hand movements and biomechanical stresses over the wrist lead to focal demyelination of the median nerve and subsequently to axonal loss.[2] A number of medical and non-medical risk factors play a role in developing CTS and they include female gender, obesity, pregnancy, diabetes and hypothyroidism among others.[3] Though, CTS is a focal neuropathy with limited pathology, it causes significant psychological distress and absenteeism from work.[4] Nocturnal pain and unpleasant paraesthesias are the cardinal symptoms of CTS.[1] Since the symptoms typically tend to occur or be worse at night, they awaken the patient from sleep, and patients experience reduced sleep quality due to disturbed sleep.

Profiling of staff members working in a university hospital who were on sick leave due to CTS revealed that more than half the subjects (60%) had disturbed sleep due to various symptoms related to CTS.[5] A few studies noted poor sleep quality, non-refreshing sleep, increased

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latency to sleep-onset and fragmented sleep among patients with CTS.[6-9] But these studies assessed only limited facets of sleep and did not use comprehensive structured sleep questionnaires and objective measures like polysomnography (PSG). No correlation was made with the various clinical or electrophysiological characteristics of CTS in these studies. Thus, the data regarding the sleep profile in CTS are rather limited. The present study was carried out based on hypothesis that CTS significantly affects sleep which can be assessed using structured sleep questionnaires and PSG. In addition, it was hypothesised that the extent of sleep alteration would correlate with the clinical and electrophysiological severity of CTS. We aimed to describe the sleep profile among patients with CTS using structured sleep questionnaires and PSG; and to correlate the same with the clinical and electrophysiological features of CTS.

METHODS

Study design

This was a hospital-based cross-sectional case-control study where we included 44 adult subjects diagnosed to have CTS by the American Association of Electrodiagnostic Medicine (AAEM) criteria[10], seen between June 2019 and May 2021 in a single neurology unit at the National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore. Patients were excluded from the study if, the CTS was a part of a generalized polyneuropathy or mononeuritis multiplex, they had a history of prior intervention by surgery or injection in carpal tunnel, or if they had systemic inflammatory conditions like rheumatoid arthritis, traumatic causes, substance abuse and pregnancy.

Study tools

Detailed clinical and electrophysiological evaluation was carried out for all the cases by two of the investigators (JG and MN). The clinical severity of CTS was assessed using the (i) Visual Analog Scale (VAS) for pain, (ii) Boston Carpal Tunnel Questionnaire (BCTQ), (iii) Historic and objective scale (HiOb), and (iv) Quick Disabilities of the Arm, Shoulder and Hand (QuickDASH) Questionnaire. The electrophysiological grading was carried out based on previously described criteria.[11]

Sleep quality was assessed using standard questionnaires: (i) Epworth Sleepiness Scale (ESS), (ii) Pittsburgh Sleep Quality Index (PSQI), (iii) Insomnia severity index (ISI), and (iv) Obstructive sleep apnea (OSA) questionnaire (STOP-BANG).

Overnight PSG was carried out in the sleep laboratory, Electrophysiology section, Department of Neurology, NIMHANS (DOMINOTM, SOMNOmedics GmbH, Germany). The study subjects were asked to wash their hair in order to remove oil or dirt and to dry their hair thoroughly on the day of the recording. They were advised not to apply any hair products prior to the PSG. They were asked to avoid tea, coffee and afternoon nap on the day of recording. All the patients were shown the sleep lab in advance and given adequate time to familiarize themselves with the surroundings. The patients were allowed to sleep and the recording was continued until their spontaneous awakening in the morning. The American Academy of Sleep Medicine (AASM) scoring manual (version 2.4) was followed for rules, terminology and specifications regarding sleep staging and recording respiratory events, limb movements and other sleep related events.[12]

Selection of controls

We recruited age- and gender-matched healthy individuals as controls prospectively from among the relatives of patients admitted to neurology ward of NIMHANS, and hospital staff including resident doctors for the administration of sleep questionnaires. The control subjects did not have any symptoms of CTS. Additionally, archived PSG data of healthy controls were used for comparing the PSG parameters of patients. Since PSG was only in a subset of 16 cases, similar number of archived records were used as controls. It was possible to match the controls for age, but not for gender. These controls did not have any recorded symptoms of CTS.

Statistical analysis

The cases and controls were assigned a numerical code to anonymise the data and to ensure confidentiality. All the data were entered into a pre-designed proforma and then into an excel sheet for analysis. Categorical data were expressed as number (percentage). Continuous variables were reported as mean ± standard deviation (SD). Analysis was carried out to compare the various clinical and electrophysiological parameters with

the quality of life and sleep profile. Comparison between continuous variables and case-type (case or control) was done by independent t-test, while that between categorial variables by chi squared test. While comparing parameters of PSG, due to small sample size in each group, we applied the non-parametric Mann Whitney U Test (Wilcoxon Rank Sum Test), wherein we also reported the median and interquartile range. A p value of less than 0.05 was considered to be statistically significant. We did not apply correction for multiple testing. The variables, age and body mass index (BMI), were not compared separately between cases and controls since they were a component of the STOP-BANG scale.

The study was approved by the Institute Ethics Committee (NIMH/DO/IEC (BS & NS DIV)/2019-20, dated 07/06/2019). A written informed consent was obtained from all the patients prior to study entry.

RESULTS

The clinical and electrophysiological characteristics of the cases included in the present study are summarised in Table 1.

The mean age of cases and controls was 42.64 ± 8.6 years and 42.41 ± 8.5 years respectively. There were 36 women and eight men in the patient and control groups each. The global PSQI score as well as the individual components of the PSQI were worse in patients with CTS as compared to healthy controls. Cases had statistically significant higher mean ESS, ISI and STOP-BANG scores as compared to the controls (Table 2). Statistically significant moderate correlation coefficients were noted between PSQI and VAS (r = 0.434, p = 0.003), PSQI and FSS (r = 0.431, p = 0.004), PSQI and SSS (r = 0.507, p = 0.001), PSQI and HiOb (r = 0.47, p = 0.001) as well as between PSQI and QuickDASH (r = 0.568, p = 0.001).

Table 1: Clinical and electrophysiological characteristics of the present cohort of carpal tunnel syndrome

Parameters	Sleep questionnaires (n=44)	Polysomnography (n=16)
Mean age of cohort (years)	43.0 ± 8.6	42.25 ± 8.7
Gender (male: female)	8 : 36	3 : 13
Mean BMI (kg/m^2)	26.38 ± 3.4	26.23 ± 4.0
Mean neck circumference (cm)	33.90 ± 4.3	33.73 ± 4.2
Mean duration of symptoms (months)	23.13 ± 25.3	25 ± 33.6
Clinical categorisation		
VAS (nil/ mild/ moderate/ severe)	5/ 29/ 3/ 7	1/ 10/ 1/ 4
BCTQ - FSS (extreme & severe/ moderate/ mild & minimal)	18/ 17/ 9	8/ 5/ 3
BCTQ - FSS (mean ± SD)	2.49 ± 0.8	2.91 ± 0.8
BCTQ - SSS (extreme & severe/ moderate/ mild & minimal)	12/ 17/ 15	5/ 5/ 6
BCTQ - SSS (mean ± SD)	2.78 ± 0.8	2.48 ± 0.9
HiOb staging (I/ II/ III/ IV/ V)	0/ 13/ 21/ 8/ 2	0/ 4/ 8/ 4/ 0
HiOb (mean ± SD)	3.00 ± 0.8	3.00 ± 0.7
QuickDASH (mean ± SD)	43.01 ± 18.6	46.31 ± 18.2
Electrophysiological grading (I/ II/ III/ IV/ V/ VI)	12/ 4/ 6/ 3/ 14/ 5	2/ 2/ 3/ 1/ 7/ 1
Sleep questionnaires		
Mean ESS	4.54 ± 3.7	5.50 ± 4.0
Mean PSQI	6.32 ± 2.6	5.87 ± 2.4
Mean ISI	10.20 ± 5.2	10.25 ± 5.5
STOP-BANG grading for OSA risk (high/ intermediate/ low)	1/ 8/ 35	1/ 2/ 13

'BCTQ': Boston Carpal Tunnel Questionnaire, 'BMI': Body Mass Index, 'FSS': Functional severity scale, 'ESS': Epworth Sleepiness Scale, 'HiOb': Historic and objective scale, 'ISI': Insomnia severity index, 'OSA': Obstructive sleep apnea, 'PSQI': Pittsburgh Sleep Quality Index, 'QuickDASH': Quick Disabilities of the Arm, Shoulder and Hand Questionnaire, 'SSS': Symptom severity scale, 'STOP': Snoring, tiredness, observed apnea, and high blood pressure, 'BANG': BMI, Age, Neck circumference, Gender, 'VAS': Visual Analog Scale.

Table 2: Comparison of Pittsburgh Sleep Quality Index in the cases of carpal tunnel syndrome and controls

Parameters	Cases (n = 44)	Controls (n = 44)	P-value*
PSQI			
Component 1 (Subjective sleep quality)	1.45 ± 0.7	0.20 ± 0.4	**0.0001**
Component 2 (Sleep latency)	1.05 ± 1.1	0.39 ± 0.5	**0.0002**
Component 3 (Sleep duration)	1.07 ± 0.7	0.59 ± 0.5	**0.0004**
Component 4 (Habitual sleep efficiency)	0.5 ± 0.7	0.05 ± 0.2	**0.0001**
Component 5 (Sleep disturbances)	1.11 ± 0.4	0.45 ± 0.5	**0.0001**
Component 6 (Sleeping medications use)	0.11 ± 0.5	0	0.0882
Component 7 (Daytime dysfunction)	1.02 ± 0.6	0	**0.0001**
Global PSQI score	6.32 ± 2.6	1.66 ± 1.4	**0.0001**
ISI	10.20 ± 5.2	0.64 ± 1.3	**< 0.0001**
ESS	4.75 ± 3.7	1.55 ± 1.6	**< 0.0001**
STOP-BANG	1.41 ± 1.4	0.25 ± 0.6	**< 0.0001**

'ESS': Epworth Sleepiness Scale, 'ISI': Insomnia Severity Index, 'PSQI': Pittsburgh Sleep Quality Index, 'STOP': Snoring, tiredness, observed apnea, and high blood pressure, 'BANG': BMI, Age, Neck circumference, and Gender.
* Based on independent samples t test

The ESS score also showed moderate correlation coefficient, which were statistically significantly with FSS (r = 0.358, p = 0.017), SSS (r = 0.416, p = 0.005), HiOb (r = 0.464, p = 0.002), and QuickDASH (r = 0.561, p = 0.001). The ISI also correlated significantly with VAS (r = 0.411, p = 0.006), FSS (r = 0.429, p = 0.004), SSS (r = 0.56, p = 0.001), and QuickDASH (r = 0.614, p = 0.001) but not with HiOb. STOP-BANG correlated significantly with SSS (r = 0.315, p = 0.037) and QuickDASH (r = 0.437, p = 0.003) but not with VAS (r = 0.185, p = 0.23), FSS (r = 0.239, p = 0.119), and HiOb (r = 0.561, p = 0.072). No statistically significant correlation was found between the sleep questionnaires and electrophysiological severity.

Sixteen cases in the present study underwent overnight PSG study. The mean (±SD) age of the 16 cases, was 42.25 ± 8.7 years and that of 16 controls was 42.31 ± 10.6 years (p = 0.98). The cases included 13 (81.3%) women and 3 (18.75%) men, while the control group included 6 (37.5%) women and 10 (62.5%) men (p = 0.03). There was evidence of fragmented and reduced sleep among cases as compared to controls in the form of (i) reduced total sleep time and sleep efficacy, (ii) reduced duration of second stage of sleep (N2) and rapid eye movement (REM) sleep, and (iii) higher wake after sleep onset (WASO), wake index, and stage shift index (p < 0.05) (Table 3).

The total number of isolated and periodic limb movements (PLM) was higher in cases as compared to controls, but did not reach statistical significance. Isolated limb movement index and PLM index was comparable between cases and controls. Apneas and hypopneas were analyzed in 12 cases and 14 controls since in the rest of the subjects, the respiratory events were not recorded satisfactorily due to technical issues. While there were no statistically significant differences between cases and controls for obstructive or central apneas, the frequency of hypopneas or hypopnea index were significantly higher in the patient group. The number of central apneas and central apnea index was comparable between cases and controls. Also, the overall desaturation index was higher in cases as compared to controls (p = 0.0012). Cases spent longer sleep time in the supine position than lateral decubitus position as compared to controls (not-significant). The average blood oxygen saturation level in cases and controls were not different.

The FSS correlated significantly with sleep efficacy (r = 0.535, p = 0.033), wake percentage (r = -0.532, p = 0.034), and WASO (r = -0.565, p = 0.022). No significant correlation was found between PSG parameters and other clinical severity scales. Except for with sleep efficacy, a negative correlation was found between electrophysiological severity grading of CTS and sleep onset latency, WASO and wake percentage.

Table 3: Observations in overnight polysomnography in patients of carpal tunnel syndrome and controls in the present study

Parameters (Median, IQR)	Cases (n=16)	Controls (n=16)	P value^
TST (minutes)	271 (138)	387 (79.8)	**<0.001**
Sleep efficiency (%)	63.4 (27.4)	84.4 (7.12)	**<0.001**
Sleep efficiency ≤ 85%	13	8	0.135
Latency to sleep stages (minutes)			
Sleep onset latency	21.8 (16.7)	12.5 (19.6)	**<0.001**
Latency to N1	26.7 (23.3)	21.5 (38.9)	**<0.001**
Latency to N2	23.6 (16.4)	24.4 (30.4)	**<0.001**
Latency to N3	35.6 (30.5)	49.6 (70.7)	**<0.001**
Latency to REM	131 (116)	92.2 (75.5)	**<0.001**
Percentage of sleep stages			
Percentage of N1	4.75 (7.3)	5.05 (11.1)	**<0.001**
Percentage of N2	34.8 (14.7)	53.4 (16.1)	**<0.001**
Percentage of N3	13.0 (16.8)	17.8 (24.7)	**<0.001**
Percentage of REM	4.85 (7.8)	16.5 (13.4)	**<0.001**
WASO	148.0 (162)	65.2 (47.2)	**<0.001**
Wake percentage	36.2 (27.5)	15.1 (7.7)	**<0.001**
Wake index*			**<0.001**
Stage shift index*	10.8 (6.95)	7.1 (2.5)	**<0.001**
Limb movements			
Total limb movement	88.5 (170.0)	58.5 (77.0)	0.175
Total limb movement index	10.6 (18.3)	10.1 (12.9)	0.534
Isolated limb movements	54.5 (55.5)	18.5 (40.0)	0.054
Isolated limb movements index	6.6 (7.3)	3.1 (6.0)	0.169
Number of PLMS	31.5 (80.8)	34.5 (43.2)	0.581
PLMS index	3.9 (9.3)	4.95 (8.1)	0.924
Respiratory Events			
Number of apneas**	4.5 (13.2)	1 (7)	0.1565
Apnea index**	1 (3.5)	0.2 (1.15)	**0.04**
Number of obstructive apneas**	4.5 (14)	0.5 (2.5)	0.903
Obstructive apnea index**	1 (3.95)	0.05 (0.425)	**0.004**
Number of hypopneas**	67.5 (97.2)	15.5 (25.5)	**<0.001**
Hypopnea index**	17.0 (29.4)	2.9 (3.4)	**<0.001**
Apnea with hypoapnea	86 (105)	28 (23.2)	**<0.001**
Apnea Hypoapnea index	19.6 (28.9)	3.6 (3.42)	**<0.001**
Body position during sleep			
Mean % of sleep time in supine position***	99.8 (7.35)	83.6 (37.1)	**<0.001**
Mean % of sleep time in lateral decubitus position***	0.2 (7.35)	16.4 (37.2)	0.5922
Oxygen saturation			
Mean oxygen saturation****	96.5 (2.25)	97 (1.6)	**<0.001**
Desaturation index****	3.95 (21.6)	2.7 (4.5)	**<0.001**
Minimum SpO2****	89.5 (5.5)	90 (4)	**<0.001**

'N': Non-Rapid Eye Movement Sleep, 'PLMS': Periodic limb movements of sleep, 'REM': Rapid Eye Movement sleep, 'TRT': Total Recording Time, 'TST': Total Sleep Time, 'WASO': Wake After Sleep Onset.
* Data available in 10 controls.
** Data available in 12 patients and 14 controls.
*** Data available in 9 controls.
**** Data available in 13 controls.
^ Based on Mann Whitney U test

DISCUSSION

The effect of various pain syndromes on quality and duration of sleep has been reported.[13,14] Pain has an arousal-enhancing influence that prevents the initiation and maintenance of sleep. Sleep can also influence the pain perception. Having fewer hours of sleep or poor-quality sleep is associated with increased pain perception. Thus, the relationship between sleep and pain is bidirectional.[15,16] While nocturnal sensory disturbances such as pain and paresthesias are a characteristic feature of CTS which awakens patients from sleep, the impact of CTS on sleep has not been studied adequately. The present study overcomes the limitations of previous studies that assessed sleep in CTS, but fragmentarily (Table 4). Taking together the parameters of all the sleep questionnaires in the present study, poor subjective sleep quality with reduced overall sleep duration, increased sleep latency, frequent awakenings, and increased day time sleepiness was found in patients with CTS. Poor sleep was reflected in higher scores in both the global and individual components of the PSQI in patients as compared to healthy controls (6.32 ± 2.6 vs 1.66 ± 1.4). Other studies reported an even higher PSQI in patients with CTS, though no comparison with healthy controls was made.[8,17,18] The present study found significant correlation between PSQI and symptom severity as has been noted in a few previous studies.[8,18] Bilgin-Topcuoglu *et al*., did not find any difference in the mean ESS between patients with or without CTS, however only patients with obstructive sleep apnea (OSA) were studied.[19] In contrast, in the present study, the mean ESS score was higher in patients as compared to healthy controls and this correlated with symptom severity. Poor sleep quality might have led to excessive daytime sleepiness in these patients. The present study also identified the positive correlation of ISI with SSS and FSS. Tulipan *et al*., reported that the mean ISI correlated with the QuickDASH scores.[20]

Literature on objective documentation of altered sleep profile in CTS by PSG is sparse (Table 4). Apart from the study by Lehtinen *et al*., where a static-charge sensitive bed was used, two other studies, by Rubin et al., and McMahon *et al*., used actigraphy to record sleep efficiency and awakening in patients with CTS.[6,9,21] The salient feature of the present study is that the PSG findings of patients with CTS were compared with that of healthy subjects which has not been carried out any previous study so far. The findings in the present study indicated that the sleep quality was poor, the total sleep time was reduced and there was fragmented sleep with poor sleep continuity in patients with CTS as compared to healthy controls. In a cohort of OSA, the sleep onset latency, sleep efficiency and various sleep stages were comparable in patients with and without CTS.[19]

In the present study, the parameters of sleep questionnaires did not correlate with electrophysiological severity of CTS. However, PSG analysis showed that there was paradoxically better sleep efficiency and reduced awakening among patients with worse FSS scores and higher electrophysiological grade of severity. Sleep is expected to be interrupted by positive sensory symptoms, and in severe CTS, sensory function may be impaired to such an extent that numbness minimizes the experience of paresthesias.[22] This may be reflected as paradoxical improvement in sleep quality. Rubin *et al*., also reported no correlation of median nerve sensory or motor latency with the ISI.[21] Lehtinen *et al*., performed median and ulnar nerve conduction studies when patients woke up at night due to pain or numbness, and failed to find any differences in sensory conduction velocities and distal motor latencies as compared to the baseline.[6] Therefore, a direct correlation between clinical sleep severity and electrophysiological severity cannot be established. There are no studies in the literature to our knowledge that have established a positive correlation between PSQI, ISI, and electrophysiological severity.

Besides documenting the sleep architecture, the present study also offered an opportunity to test the contribution of body position and hypoxia to CTS. Previously McCabe *et al*., hypothesized that body position in sleep is an important factor in the pathophysiology of CTS. The wrist is more likely to be in flexed or extended when body is in the lateral decubitus position, and this contributes to increased pressure in the carpal tunnel.[23-25] But the main drawback was that this hypothesis was based on epidemiological associations and was not confirmed by electrophysiological studies or PSG. In the present study, however, the duration of time spent in the supine and lateral decubitus position, as recorded by PSG, was comparable in patients and controls. Bilgin-Topcuoglu *et al*., also did not find any difference in polysomnographically documented sleeping position in patients with and without CTS.[19] In contrast, Roth-Bettlach *et al*., reported less frequent night-time paresthesias in subjects who slept in the lateral decubitus

Table 4: Summary of studies on sleep profiling in patients with carpal tunnel syndrome

Author, Year	Number	Sleep assessment tools	Parameters	Key findings
Lehtinen *et al*., 1996.[6]	N = 34 M: F = 13:21	Set of questions related to sleep	Sleep quality, fragmentary sleep, nocturnal awakening, waking early, sleep latency, insomnia, use of sleeping pills	Poor sleep quality, fragmentary sleep, excessive daytime sleepiness, and more snore in CTS as compared to controls.
	N = 6	Polygraphic sleep study = static-charge sensitive bed (Biomatt) Actigraphy	Gross body movement, respiratory movements, hand movement.	Improved body movement and less nocturnal awakening noted after carpal tunnel release. No significant change in electrophysiological parameters noted during night awakening.
Patel *et al*., 2012.[7]	N = 62 M: F = 16:46	Short insomnia instrument, ISI	ISI parameters, preferred sleep position.	All items of ISI higher in CTS.
Tanik *et al*., 2016.[18]	N = 366	PSQI, VAS	PSQI and VAS parameters	Sleep quality correlated with VAS but not with electrophysiological severity.
Tulipan *et al*., 2017.[20]	N = 398 M: F = 163:235	ISI, QuickDASH	ISI and QuickDASH parameters	Improved ISI and QuickDASH post CTR.
Bilgin-Topcuoglu *et al*., 2017.[19]	N = 80 with OSA M: F = 71:9	ESS	ESS parameters	Prevalence of CTS in patients with OSA = 27.5%. BCTQ did not correlate with severity of OSA or CTS. No difference in ESS between OSA patients with and without CTS.
		PSG	Sleep architecture, respiratory parameters, body movements, blood oxygen saturation.	No difference in PSG parameters between OSA patients with and without CTS.
Niedermeier *et al*., 2020.[17]	N = 61 M: F = 14:47	QuickDASH, PSQI	QuickDASH and PSQI parameters	Improved sleep quality post CTR.
Rubin *et al*., 2020.[21]	N = 17 M: F = 8: 13	ISI, Sleep log	ISI parameters Time going to bed, sleep onset latency, Number of awakenings, time of final awakening, perceived sleep quality	Insomnia in 90% patients, fragmentary sleep but no problem with falling asleep or waking up early. No correlation between results of the sleep log or ISI and median nerve sensory/ motor latency.
		Actigraphy	Sleep quality, continuity, sleep latency, total sleep duration, sleep efficiency.	No relationship between actigraphy sleep measures and median nerve motor/ sensory latency.
McMahon *et al*., 2020.[9]	N = 20 M: F = 5:15	PROMIS Questionnaire	PROMIS 29, PROMIS upper extremity, PROMIS Sleep Disturbance, and PROMIS Sleep-Related Impairment questionnaires	No statistically significant difference in any domains of the questionnaire before and after CTR.

		Actigraphy	Movement index, fragmentation index, sleep fragmentation index, number and duration of awakenings, sleep efficiency, and total sleep time	Decreased sleep fragmentation and duration of awakening post carpal tunnel release, but not significant. Reduced sleep activity count significantly.
Current study	N = 44 M: F = 8: 36	PSQI, ESS, ISI, STOP-BANG	PSQI, ESS, ISI, STOP-BANG parameters	Scores in patients of CTS significantly higher as compared to healthy controls. Significant correlation with clinical severity scales (FSS, SSS, and QuickDASH). No correlation with electrophysiological severity.
		Overnight PSG	Sleep architecture, respiratory parameters, body movements, blood oxygen saturation.	Total sleep time, sleep efficacy significantly reduced. Wake percentage, wake after sleep onset, and stage shift index significantly higher in patients. Hypoapnea index and desaturation index higher in patients.

'BCTQ': Boston carpal tunnel questionnaire, 'CTR': Carpal tunnel release, 'ESS': Epworth Sleepiness Scale, FSS'-Functional severity scale, 'ISI': Insomnia Severity Index, 'PROMs': Patient-reported outcomes measures, 'PSG': Polysomnography, 'PSQI': Pittsburgh Sleep Quality Index, 'QuickDASH': Quick Disabilities of the Arm, Shoulder and Hand Questionnaire, 'STOP': Snoring, tiredness, observed apnea, and high blood pressure, 'BANG': BMI, Age, Neck circumference, and Gender, 'HiOb'- Historic and objective scale, 'ISI': Insomnia severity index, 'SSS': Symptom severity scale.

position.[26] It is possible that a change in sleeping posture occurs after the development of CTS, wherein a patient adapts and avoids sleeping on the lateral decubitus position to minimize the paresthesias.

In the present study patients, of CTS had higher number of hypoapneas and hypoapnea index as compared to controls. The desaturation index was also significantly higher in patients. A previous study demonstrated an association between OSA and CTS[19]; intermittent hypoxia during the night was hypothesized to contribute to the pathophysiology of CTS in these patients. The finding of increased hypoapnea and desaturation in the present study may support the contribution of hypoxia in the development of CTS.

Our study had a few limitations. Sleep quality could be affected by eg socio-demographic, psychological (depression, anxiety, stress), or biological (blood pressure, diabetes, BMI) factors. However, this limitation was circumvented to a large extent by adopting case-control study design. We selected age-gender matched controls in order to ensure to a large extent an uniform distribution of these factors between cases and controls. Also, the cases did not have major systemic co-morbidities and therefore the confounding factors related to them were largely avoided. Moreover, none of the cases in the present study had psychological factors like depression, anxiety or stress (data not shown). Hence, we do not think this limitation would have a considerable bearing on the findings of this study.

Another limitation of our study was that the objective assessment of sleep using PSG could be performed only in a subset of cases, and the comparison controls available were from archived data; this control data was also not gender matched with the cases. Hence, for the PSG component of the analyses, we could not rule out residual confounding. However, such comparison is not usually reported in literature, and our PSG findings corroborated well with the PSQI based sleep quality.

The study provides robust data regarding sleep profile in patients with CTS as well as their clinical and electrophysiological correlations, which is the major strength of this study. All the patients were recruited from a single Neurology unit and comprehensive assessment of sleep was carried out. Case ascertainment was rigorous, as only clinically symptomatic patients with electrophysiologically confirmed CTS were eligible. All the assessments including

electrophysiological testing and PSG were carried out using a single system by a single investigator.

In conclusion, we show that parameters of CTS were significantly associated with poor sleep quality, ascertained both by a valid PSQI questionnaire, as well as objectively by PSG in subset of patients. However, due to limitation of the study design, we cannot comment if the association is causal in nature. Hence, it is prudent that patients of CTS may be assessed for quality of sleep and counselled/ treated accordingly.

DISCLOSURE

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REFERENCES

1. Ferry S, Hannaford P, Warskyj M, *et al*. Carpal tunnel syndrome: a nested case-control study of risk factors in women. *Am J Epidemiol* 2000;151(6):566-74. doi: 10.1093/oxfordjournals.aje.a010244.
2. Werner RA, Andary M. Carpal tunnel syndrome: pathophysiology and clinical neurophysiology. *Clin Neurophysiol* 2002;113(9):1373-81. doi: 10.1016/s1388-2457(02)00169-4.
3. Roquelaure Y, Garlantézec R, Rousseau V, *et al*. Carpal tunnel syndrome and exposure to work-related biomechanical stressors and chemicals: Findings from the Constances cohort. *PLoS One* 2020;15(6):e0235051. doi: 10.1371/journal.pone.0235051.
4. Damms NA, McCallum LM, Sarrigiannis PG, *et al*. Pain as a determinant of health-related quality of life in patients with carpal tunnel syndrome; a case-controlled study. *Postgrad Med* 2020;132(1):52-5. doi: 10.1080/00325481.2019.1694840.
5. de Azevedo JW, de Oliveira AB, Nascimento Vd, *et al*. Profile of patients on sick leave with carpal tunnel syndrome. *Acta Ortop Bras* 2015;23(5):244-6. doi: 10.1590/1413-785220152305145784.
6. Lehtinen I, Kirjavainen T, Hurme M, *et al*. Sleep-related disorders in carpal tunnel syndrome. *Acta Neurol Scand* 1996;93(5):360-5. doi: 10.1111/j.1600-0404.1996.tb00010.x.
7. Patel JN, McCabe SJ, Myers J. Characteristics of sleep disturbance in patients with carpal tunnel syndrome. *Hand* (N Y). 2012;7(1):55-8. doi: 10.1007/s11552-011-9373-1.
8. Patel A, Culbertson MD, Patel A, *et al*. The negative effect of carpal tunnel syndrome on sleep quality. *Sleep Disord* 2014;2014:962746. doi: 10.1155/2014/962746.
9. McMahon HA, Heineman JT, DeGeorge BR Jr. Patient-generated actigraphy data as a novel outcomes instrument in carpal tunnel syndrome. *Ann Plast Surg* 2020;84(6S Suppl 5):S441-S445. doi: 10.1097/SAP.0000000000002269.
10. Jablecki CK, Andary MT, Floeter MK, *et al*. American Association of Electrodiagnostic Medicine; American Academy of Neurology; American Academy of Physical Medicine and Rehabilitation. Practice parameter: Electrodiagnostic studies in carpal tunnel syndrome. Report of the American Association of Electrodiagnostic Medicine, American Academy of Neurology, and the American Academy of Physical Medicine and Rehabilitation. *Neurology* 2002;58(11):1589-92. doi: 10.1212/wnl.58.11.1589.
11. Bland JD. A neurophysiological grading scale for carpal tunnel syndrome. *Muscle Nerve* 2000;23(8):1280-3. doi: 10.1002/1097-4598(200008)23:8<1280::aid-mus20>3.0.co;2-y.
12. Berry RB, Brooks R, Gamaldo C, *et al*. AASM Scoring Manual Updates for 2017 (Version 2.4). *J Clin Sleep Med* 2017;13(5):665-6. doi: 10.5664/jcsm.6576.
13. Marty M, Rozenberg S, Duplan B, *et al*. Section Rachis de la Société Française de Rhumatologie. Quality of sleep in patients with chronic low back pain: a case-control study. *Eur Spine J* 2008;17(6):839-44. doi: 10.1007/s00586-008-0660-7.
14. Marin R, Cyhan T, Miklos W. Sleep disturbance in patients with chronic low back pain. *Am J Phys Med Rehabil* 2006;85(5):430-5. doi: 10.1097/01.phm.0000214259.06380.79.
15. Haack M, Simpson N, Sethna N, *et al*. Sleep deficiency and chronic pain: potential underlying mechanisms and clinical implications. *Neuropsychopharmacology* 2020; 45(1):205-16. doi: 10.1038/s41386-019-0439-z.
16. Lavigne GJ. Effect of sleep restriction on pain perception: towards greater attention! *Pain* 2010;148(1):6-7. doi: 10.1016/j.pain.2009.10.013.
17. Niedermeier SR, Pettit RJ, Frantz TL, *et al*. Early postoperative improvement in sleep and pain after carpal tunnel release. *Hand* (N Y). 2020;15(3):311-4. doi: 10.1177/1558944718793984.
18. Tanik N, Sarp U, Ucar M, *et al*. Pain, depression and sleep disorders in patients with diabetic and nondiabetic carpal tunnel syndrome: a vicious cycle. *Arq Neuropsiquiatr* 2016;74(3):207-11. doi: 10.1590/0004-282X20160020.
19. Bilgin-Topçuoğlu Ö, Oruç Ö, Saraç S, *et al*. Carpal tunnel syndrome in obstructive sleep apnea patients. *Noro Psikiyatr Ars* 2017;54(4):307-11. doi: 10.5152/npa.2016.15907.
20. Tulipan JE, Kim N, Abboudi J, *et al*. Prospective evaluation of sleep improvement following carpal tunnel release surgery. *J Hand Surg Am* 2017;42(5):390.e1-390.e6. doi: 10.1016/j.jhsa.2017.02.009.
21. Rubin G, Orbach H, Rinott M, *et al*. Relationship between electrodiagnostic findings and sleep disturbance in carpal tunnel syndrome: A controlled objective and subjective study. *J Int Med Res* 2020;48(2):300060519862673. doi: 10.1177/0300060519862673.
22. MacDermid JC, Wessel J. Clinical diagnosis of carpal tunnel syndrome: a systematic review. *J Hand Ther* 2004;17(2):309-19. doi: 10.1197/j.jht.2004.02.015.
23. McCabe SJ, Uebele AL, Pihur V, *et al*. Epidemiologic

associations of carpal tunnel syndrome and sleep position: Is there a case for causation? *Hand* (N Y) 2007; 2(3):127-34. doi: 10.1007/s11552-007-9035-5.
24. McCabe SJ, Xue Y. Evaluation of sleep position as a potential cause of carpal tunnel syndrome: preferred sleep position on the side is associated with age and gender. *Hand* (N Y) 2010;5(4):361-3. doi: 10.1007/s11552-010-9271-y.
25. McCabe SJ, Gupta A, Tate DE, *et al*. Preferred sleep position on the side is associated with carpal tunnel syndrome. *Hand* (NY) 2011;6(2):132-7. doi: 10.1007/s11552-010-9308-2.
26. Roth-Bettlach CL, Hasak JM, Krauss EM, *et al*. Preferences in sleep position correlate with nighttime paresthesias in healthy people without carpal tunnel syndrome. *Hand* (N Y) 2019;14(2):163-71. doi: 10.1177/1558944717735942.