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Are body mass index and the systemic immune-inflammation index risk factors for carpal tunnel syndrome?

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ABSTRACT

Objectives: Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy of the upper extremity that affects activities of daily living. In our study, we aimed to reveal the relationship between CTS and BMI, and to evaluate symptom severity and functionality in these patients by using the Boston CTS questionnaire.

Methods: In this study, 300 patients with CTS and 100 healthy individuals without CTS whose EMG was performed in our neurology clinic electrophysiology laboratory between June 2021 and December 2021, were included. BMI, SII index, and Boston CTS questionnaire findings were compared between patients diagnosed as having mild, moderate, and severe CTS (according to electrophysiologic evaluations) and a control group consisting of healthy individuals.

Results: In our study, a statistically significant difference was observed between the CTS and control groups in terms of mean age ($p < 0.001$). When the increased BMI and SII parameters were compared, a statistically significant difference was observed between the control and CTS groups ($p < 0.001$ for both). In the multivariate logistic regression analysis, it was observed that the risk of CTS increased 1.566 times as BMI increased, and the risk of CTS increased 1.005 times as the SII index increased ($p < 0.001$ for both).

Conclusions: We observed that increased BMI and advanced age were risk factors for CTS. In our study, in which the relationship between the SII index and CTS was evaluated for the first time, according to our findings, inflammation was thought to play a role in the pathophysiology of CTS.

Keywords: Carpal tunnel syndrome, body mass index, nerve conduction study, inflammation, systemic immune-inflammation index

Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy of the upper extremity, which causes functional disability in clinical practice and affects daily life activities [1]. It presents with localized paresthesia in the hands, especially in the palms and 1st-3rd fingers, and increasing pain at night, which develops as a result of compression of the median nerve at the wrist level. It has been reported to be more common in females and the 3rd and 5th decades [2]. Apart from this, advanced age, pregnancy, connective tissue diseases, hypothyroidism, diabetes mellitus, occupational diseases are other common predisposing factors [2]. In the electrophysiology laboratory, the diagnosis of CTS is made

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through motor and sensory nerve conduction studies of the median nerve. The motor and sensory conduction velocities of the median nerve and motor distal latency are evaluated, and severity classification is made according to the level of symptoms [3, 4].

Considering the predisposing factors, it is obvious that multifactorial processes are dominant in the etiopathogenesis of CTS. Although the underlying mechanisms are not fully understood, it is thought that the common result of all these processes is compression in the channel where the median nerve travels and an increase in pressure secondary to the increase in fat tissue in individuals with obesity. Axonal damage to the median nerve develops as a result of compression of the median nerve in the carpal tunnel, and ischemia in the median nerve develops as a result of damage to the vascular structures in the perineum. Repetitive compression leads to tenosynovial thickening, the pressure becomes continuous, and the median nerve is pressed in the canal [5]. Studies in the literature on whether inflammation affects these processes are limited. We aimed to reveal the relationship between the systemic immune-inflammation index (SII), which was shown as an inflammation marker in various diseases recently, and the severity of CTS [6, 7]. We believe our study is the first to investigate this relationship because we have not come across a similar study in the literature. It is known that individuals with obesity are prone to CTS, and it has been shown in various studies that there is a relationship between an increase in body mass index (BMI) and the development of CTS [1, 8]. It has been reported that the development of CTS in individuals with obesity may be associated with an increase in adipose tissue or an increase in hydrostatic pressure in the canal where the median nerve travels [9].

In our study, we aimed to reveal the relationship between CTS and BMI and the SII in patients with CTS detected in our electrophysiology laboratory and evaluate severity and functionality in these patients using the Boston CTS questionnaire.

METHODS

In this study, 300 patients with CTS and 100 healthy individuals without CTS who were evaluated in our electrophysiology laboratory between June 2021 and December 2021 were included. The nerve conduction studies and clinical diagnostic findings of the patients with clinical and electrophysiologic diagnoses of CTS and a control group were recorded. The study was approved by the local ethics committee (2021/025). Informed consent forms were obtained from the patients with CTS and the control group.

Electrophysiologic examinations of the patients were performed using a Nihon Kohden Corp. device by the same person, in the same place. Standardization was achieved by paying attention to the body temperature and room temperature of the patients.

Median nerve motor and sensory conduction studies were performed on patients who were admitted to the electromyography (EMG) laboratory with entrapment neuropathy. In addition, ulnar nerve motor and sensory conduction studies and concentric needle EMG were included in the study to exclude polyneuropathy and radiculopathy.

In the median nerve motor nerve conduction study, the superficial electrodes and the recording electrode were placed on the abductor pollicis brevis (APB) muscle, and then the recording was made by stimulating the median nerve 5 cm proximal to the active electrode on the wrist, along the fold between the hyper- and hypothenar muscles in the middle of the wrist, and by stimulating the median nerve in the elbow bend near the brachial artery pulse, as the second stimulation point. Motor distal latency, motor conduction velocity, and compound muscle action potential (CMAP) were recorded. Distal latency $\geq$ 3.98 ms, CMAP $\leq$ 4 mV, or conduction velocity $\leq$ 49.7 m/s was considered abnormal. The ulnar nerve was stimulated 5 cm proximal to the recording electrode and 4 cm distal to the elbow for motor conduction. It was considered abnormal if the ulnar nerve distal latency was above 3.3 ms, its velocity was below 49.9 m/s, or the CMAP amplitude was below 7.0 mV.

For the sensory nerve conduction study of the median nerve, recordings were made on 1st, 2nd, and 3rd fingers after stimulating from the wrist, palm-wrist segment, and wrist-elbow segment, respectively. For the sensory nerve conduction study of the ulnar nerve, a recording was made on the 5th finger. According to the normal levels of our laboratory, the velocity of the sensory potential obtained from the negative peak is considered abnormal if it is < 32.92 m/s for the 1st finger, < 39.4 m/s for the 2nd finger, < 39.65 m/s for the

3rd finger, < 35.2 m/s for palm-wrist, < 49.0 m/s for wrist-elbow, and < 37.3 m/s for 5th finger [10].

For the diagnosis of CTS; median motor nerve distal latency (mMDL), median motor nerve conduction velocity (mMNCV), median nerve motor compound muscle action potential (mCMAP), second finger median sensory nerve conduction velocity (mSNCV), median sensory nerve distal latency (mSDL), median sensory nerve action potential amplitude (mSNAP), ulnar motor nerve distal latency (uMDL), ulnar motor nerve conduction velocity (uMNCV), ulnar motor nerve compound muscle action potential (uCMAP), fifth finger median sensory nerve conduction velocity (uSNCV), ulnar sensory nerve distal latency (uSDL), and ulnar sensory nerve action potential amplitude (uSNAP) were evaluated and recorded.

Using concentric needle electrodes, needle EMG was usually applied to the abductor pollicis brevis muscle and if necessary, according to the findings, it was applied to the pronotor teres and flexor pollicis longus muscles, which were more proximal muscles. When necessary, needle EMG was performed on abductor digiti minimi and extensor indicis proprius muscles to exclude radiculopathy from polyneuropathy [4].

Bandpass filters were set at 20-20,000 Hz for motor nerve conductions, 20-2000 Hz for sensory nerve conductions, and 10-10,000 Hz for needle EMG. Body temperature was measured and corrected to 31°C according to the conversion table; 1 m/s was added to motor and sensory nerve conduction velocities in participants aged over 60 years.

Participants included in the study were grouped according to their electrophysiologic findings as mild CTS, moderate CTS, severe CTS, and the control group. Both hands were studied in all participants and patients were classified electrophysiologically according to the following criteria. Mild CTS: Decreased sensory conduction velocity and amplitude in the median nerve finger-wrist segment and palm-wrist segment. Moderate CTS: Prolongation of the distal latency of the median motor nerve (> 4.0 msec) and decrease in the median nerve sensory conduction velocity and amplitude. Severe CTS: Failure to obtain uCMAP, decreased median motor nerve amplitude, prolongation of the distal latency of the median motor nerve, or failure to obtain mCMAP [11].

Patients with hereditary polyneuropathy, diabetic polyneuropathy, cervical radiculopathy, brachial plexopathy, malignancy, traumatic nerve damage, connective tissue disease, rheumatological disease, hereditary neuropathy with liability to pressure palsy (HNPP), chronic renal failure, and patients in the infectious process were not included. Chronic diseases of the patients were recorded.

Among the laboratory markers, white blood cell (WBC), neutrophil, lymphocyte, and platelet counts were recorded. NLR = neutrophil count / lymphocyte count and SII = neutrophil count × platelet count / lymphocyte count were calculated.

Height (cm) and weight (kg) measurements of the patients were made. BMI was calculated using the formula weight / height2 (kg/m^2). Patients with BMI < 25 kg/m^2 were considered normal-weight, patients with BMI 25-30 kg/m^2 were considered overweight, patients with BMI 30-35 kg/m^2 were considered as having class 1 obesity, and patients with BMI > 35 kg/m^2 were considered as having class 2 obesity [12].

The Boston Carpal Tunnel Syndrome Questionnaire (BCTQ), consisting of two parts, was completed independently by the patients to evaluate their functional limitation and symptom severity. The symptom severity scale consists of 11 multiple-choice questions and the functional status scale consists of eight questions. Responses are scored from 1 (mildest) to 5 points [13]. The validity and reliability studies of the Turkish adaptation of BCTQ were carried out. [14].

Statistical Analysis

Data were analyzed using the IBM SPSS V23 software package. Conformity to normal distribution was evaluated using the Kolmogorov-Smirnov test. The Chi-square test and Fisher's exact test were used to compare categorical variables between the groups. The Mann-Whitney U test was used to compare data that were not normally distributed between two groups. The Kruskal-Wallis test was used to compare data that were not normally distributed between three or more groups, and multiple comparisons were analyzed using the Dunn test. Binary logistic regression analysis was used to examine the risk factors affecting the formation of CTS. Quantitative data results are given as mean ± standard deviation and median (minimum-maximum). Categorical data results are given as frequency (percentage). The significance level was accepted as $p < 0.050$.

RESULTS

Our study included 300 patients with CTS who were diagnosed in our electrophysiology laboratory, and 100 healthy controls. One hundred eighty patients were not included in the study due to exclusion criteria. Patients diagnosed as having CTS were divided into three groups according to their electrophysiologic classification as mild, moderate, and severe CTS.

In Table 1, the demographic characteristics of the groups are shown. There was no statistically significant difference between the mild, moderate, severe CTS groups and the control group in terms of sex distribution ($p > 0.050$).

Considering the age distribution between the groups, the mean age of the control group was 42.0 ± 15.0 years, and the mean age of the CTS group was 51.0 ± 14.4 years; there was a statistically significant difference between the groups in terms of mean age ($p < 0.001$).

Considering weight distribution, it was observed that there was a statistically significant difference in terms of the median weight values between the control group and CTS groups ($p < 0.001$). In addition, a statistically significant difference was observed in terms of BMI between the control group and CTS groups ($p < 0.001$). There was no statistically significant difference between the groups in terms of chronic diseases (Table 1).

When the laboratory parameters were examined, a statistically significant difference was observed between the groups in terms of the median counts of lymphocytes and platelets ($p < 0.001$ for both). When the median SII index values were compared, the median SII index in the control group, mild CTS group, moderate CTS group, and severe CTS group was 469.5 ± 167, 747.1 ± 329.5, 824.1 ± 810.4, and 774.0 ± 275.4, respectively. A statistically significant difference was found between the groups ($p < 0.001$). No statistically significant difference was found in terms of the distributions of monocytes and neutrophils between the groups ($p > 0.050$) (Table 1, Fig. 1).

The BCTQ, consisting of two parts, was administered to patients diagnosed as having CTS. Accordingly, a statistically significant difference was found in terms of the median values of the Boston Symptom Severity Scale and Boston Functional Status Scale between the CTS groups ($p < 0.001$ for both) (Table 2).

Risk factors affecting CTS were analyzed using binary logistic regression analysis as univariate and multivariate models. Considering the multivariate logistic regression analysis, CTS risk increased 1.566 times ($p < 0.001$) as BMI increased, and CTS risk increased 1.005 times as SII increased ($p < 0.001$) (Table 3).

DISCUSSION

In our study, we evaluated whether BMI and the SII index, which was recently associated with an inflammatory process, was associated with CTS.

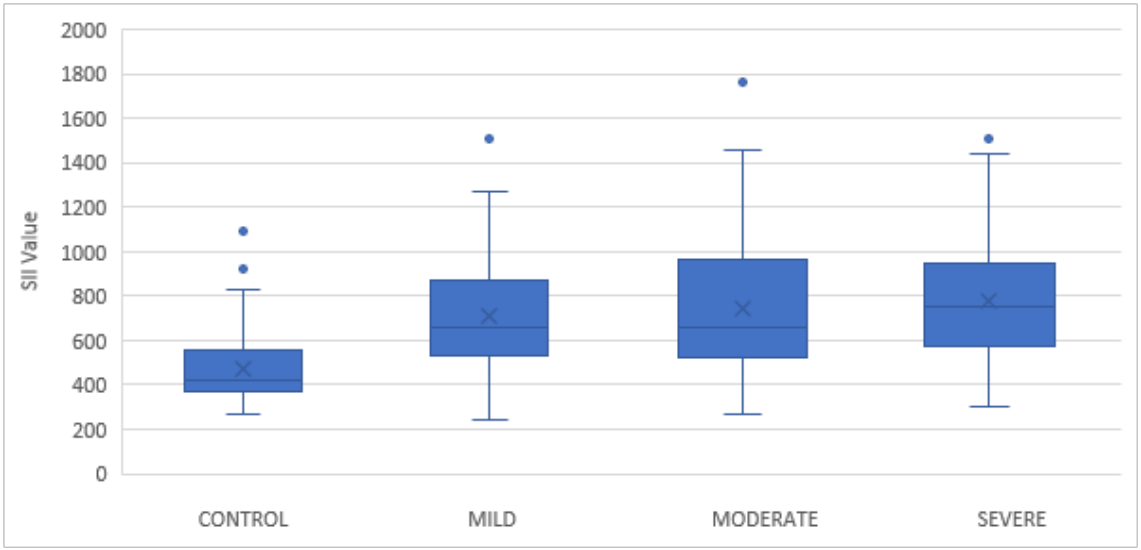

Fig. 1. Box plot of SII values.

Table 1. Comparison of demographic data and laboratory parameters between groups

	Control	Mild	Moderate	Severe	Total	Test statistics	*p value*
Gender, n (%)							
F	78 (78)	76 (76)	84 (84)	66 (66)	304 (76)	4.605	0.203*
M	22 (22)	24 (24)	16 (16)	34 (34)	96 (24)		
Age (years)	42.0 ± 15.0	52.8 ± 12.1	53.0 ± 13.5	56.0 ± 13.2	51.0 ± 14.4	22.862	**< 0.001***
	42.0 (15.0 - 68.0)[a]	52.5 (29.0 - 72.0)[b]	54.0 (23.0 - 85.0)[b]	57.0 (27.0 - 81.0)[b]	51.5 (15.0 - 85.0)		
Height (cm)	166.1 ± 7.1	163.3 ± 9.2	162.4 ± 9.0	161.1 ± 6.8	163.3 ± 8.3	11.272	0.060**
	165.0 (155.0 - 182.0)[b]	160.0 (150.0 -185.0)[ab]	160.0 (150.0 - 192.0)[ab]	160.0 (150.0 - 175.0)[a]	161.5 (150.0 - 192.0)		
Weight (kg)	68.8 ± 7.9	82.2 ± 12.7	79.1 ± 12.7	79.2 ± 12.5	77.3 ± 12.6	34.815	**< 0.001****
	69.0 (50.0 - 85.0)[a]	80.0 (60.0 - 115.0)[b]	76.5 (51.0 - 110.0)[b]	78.0 (49.0 - 105.0)[b]	75.0 (49.0 - 115.0)		
BMI (kg/m^2)	24.6 ± 4.3	30.9 ± 4.9	30.2 ± 3.7	30.6 ± 4.5	29.0 ± 5.1	66.865	**< 0.001***
	25.1 (0.0 - 29.8)[a]	29.8 (22.0 - 45.8) [b]	29.0 (24.3 - 39.1) [b]	30.1 (21.8 - 43.7)[b]	28.1 (0.0 - 45.8)		
HT							
No	88 (88)	72 (72)	76 (76)	72 (72)	308 (77)	4.856	0.183*
Yes	12 (12)	28 (28)	24 (24)	28 (28)	92(23)		
DM							
No	86 (86)	70 (70)	86 (86)	70 (70)	312 (78)	7.459	0.059*
Yes	14 (14)	30 (30)	14 (14)	30 (30)	88 (22)		
Hpl							
No	100 (100)	90 (90)	92 (92)	92 (92)	374 (93.5)	4.854	0.183*
Yes	0 (0)	10 (10)	8 (8)	8 (8)	26 (6.5)		
Neutrophil ×10^3/µL	4.2 ± 0.8	4.4 ± 1.2	4.5 ± 1.4	4.7 ± 1.2	4.4 ± 1.2	4.634	0.201*
	4.0 (2.6 - 6.2)	4.3 (2.3 - 6.7)	4.2 (2.6 - 10.3)	4.5 (2.6 - 7.4)	4.3 (2.3 - 10.3)		
Lymphocyte ×10^3/µL	2.55 ± 0.48	2.02 ± 0.54	2.03 ± 0.52	2.04 ± 0.36	2.16 ± 0.53	38.926	**< 0.001**
	2.59 (1.60 - 3.85)[a]	1.90 (1.00 - 3.66)[b]	2.00 (1.08 - 3.71)[b]	2.10 (1.30 - 2.86)[b]	2.10 (1.00 - 3.85)		
Platelet ×10^3/µL	278.6 ± 47.7	317.4 ± 56.6	372.6 ± 449.2	329.1 ± 50.4	324.4 ± 229.7	27.412	**< 0.001***
	278.0 (189.0-451.0)[a]	326.5 (189.0 - 423.0)[b]	302.0 (190.0-3455.0)[b]	341.5 (190.0-431.0)[b]	304.5 (189.0-3455.0)		
Monocyte ×10^3/µL	0.79 ± 0.76	0.64 ± 0.18	0.70 ± 0.17	0.70 ± 0.18	0.71 ± 0.41	3.844	0.279*
	0.67 (0.27 - 5.90)	0.62 (0.30 - 1.00)	0.70 (0.43 - 1.00)	0.70 (0.32 - 1.02)	0.67 (0.27 - 5.90)		
SII ×10^3/µL	469.5 ± 167.7	747.1 ± 329.5	824.1 ± 810.4	774.0 ± 275.4	703.7 ± 482.9	46.036	**< 0.001***
	422.9 (264.1-1095.3)[a]	657.2 (265.8-1762.6)[b]	674.3 (242.0-6158.9)[b]	755.1 (299.9-1508.5)[b]	613.7 (242.06158.9)		

Data are given as mean ± standard deviation or median (mininimum – maximum) or n (%). F = Female, M = Male, BMI = Body mass index, HT = Hypertension, DM = Diabetes mellitus, Hpl = Hyperlipidemia, SII = Systemic immune-inflammation index, *Chi-square test, **Kruskal-Wallis test, ab No difference between groups with the same letter.

Table 2. Comparison of Boston Symptom Severity Scale and Boston Functional Status Scale parameters between the groups

	Mild	Moderate	Severe	Total	Test statistics	*p* value
Boston Symptom Severity Scale	15.6 ± 3.4	23.9 ± 3.2	39.2 ± 7.9	26.2 ± 11.1	131.628	**< 0.001**
	16.0 (11.0 – 20.0)[a]	23.0 (20.0 – 30.0)[b]	37.5 (30.0 – 55.0)[c]	23.0 (11.0 – 55.0)		
Boston Functional Status Scale	7.6 ± 1.4	18.3 ± 2.8	30.2 ± 5.1	18.7 ± 9.9	131.569	**< 0.001**
	8.0 (5.0 – 10.0)[a]	18.0 (15.0 – 25.0)[b]	29.0 (23.0 – 40.0)[c]	18.0 (5.0 – 40.0)		

Data are given as mean ± standard deviation or median (mininimum – maximum) or n (%). Kruskal-Wallis test, [ac] No difference between groups with the same letter,

In our study, it was found that the mean age of patients with CTS was statistically significantly higher than the control group. The increased risk with age might be associated with prolonged exposure to repetitive physical movements that strained the wrist. In addition, axon loss and vascular abnormalities that develop in nerves with age may explain the relationship between age and CTS [15-16]. Becker *et al*. [17] evaluated 791 patients with CTS and emphasized that female sex, BMI > 30, age 41-60 years, and DM were independent risk factors for CTS.

It has been suggested that the hydrostatic pressure, which occurs as a result of the increase in the adipose tissue around the median nerve in individuals with obesity with increased BMI, causes a slowdown in the median nerve sensory conduction. In our study, when we compared the mild, moderate, and severe CTS groups with the control group in terms of BMI, we observed a statistically significant relationship between increased BMI and CTS. In the epidemiologic study conducted by Vessey *et al*. [18], a significant relationship was observed between BMI and CTS, similar to our study. In other study, in which the relationship between CTS and obesity was discussed from a different perspective, the relationship between abdominal obesity and CTS was investigated by measuring waist circumference-waist-hip ratio, and it was suggested that abdominal obesity might be a risk factor for CTS [19].

In another study, it was reported that there was an increase in the risk of CTS in addition to the increases in the risk of development of cardiovascular disease and type 2 DM in patients with obesity with metabolic syndrome [20]. Besides BMI being a risk factor for CTS, no relationship was found between the increase in BMI and the severity of CTS [9]. In the study of Kouyoumdjian *et al*. in which 210 patients with symptomatic CTS and 320 controls were compared, it was reported that there was a significant relationship between CTS and BMI, and CTS-wrist index. They also reported that although age and wrist index were associated with the severity of CTS, BMI was not associated with the CTS severity [21]. When the multivariate model analysis results of our study were examined, we observed that each 1 unit increase in BMI increased the risk of CTS by 1.566 times ($p < 0.001$). In other words, according to the results of our study, it can be concluded that the risk of developing CTS increases as BMI increases.

Table 3. Examination of risk factors affecting the disease

	Univariate		Multivariate (enter)[1]		Multivariate (wald)[2]	
	OR (95 % CI)	*p* value	OR (95 % CI)	*p* value	OR (95 % CI)	*p* value
Gender (male)	1.161 (0.540-2.495)	0.702	2.210 (0.733-6.664)	0.159		
Ht	0.375 (0.148-0.947)	**0.038**	1.765 (0.367-8.495)	0.478		
Dm	0.497 (0.206-1.200)	0.120	0.741 (0.188-2.930)	0.670		
Age	1.066 (1.038-1.095)	**< 0.001**	1.034 (0.995-1.074)	0.085	1.037 (1.001-1.074)	**0.046**
BMI	1.849 (1.509-2.265)	**< 0.001**	1.611 (1.307-1.985)	**< 0.001**	1.566 (1.285-1.907)	**< 0.001**
SII	1.006 (1.004-1.008)	**< 0.001**	1.005 (1.002-1.008)	**< 0.001**	1.005 (1.002-1.008)	**< 0.001**

BMI = Body mass index, HT = Hypertension, DM = Diabetes mellitus, SII = Systemic immune inflammation index. Cox&Snell R^2 = 0,3419; Nagelkerke R^2 = 0,621; Hosmer and Lemeshow Chi Square = 8,304, Accuracy= 0,850[1], Cox&Snell R^2 = 0,412; Nagelkerke R^2 = 0,610; Hosmer and Lemeshow Chi Square = 7.926, Accuracy = 0,850[2]

Investigations on the pathophysiologic mechanisms that cause compression and traction of the median nerve are still ongoing, and studies aimed at investigating the role of chronic inflammation and elucidating the pathophysiology attract attention [22]. It is known that fibrosis develops as a result of compression in the canal where the median nerve is located. The release of interleukins (IL-1, IL-2, and IL-6) increases with the increase in pressure caused by the effect of compression. Increased interleukins trigger the formation of fibrosis by increasing the release of growth factors such as vascular endothelial growth factor (VEGF) and transforming growth factor-beta (TGF-β). Subclinical systemic inflammation may also play a role in the development of fibrosis by triggering the release of cytokines and growth factors [23]. The SII index is a marker that has been shown to be associated with inflammation in many studies in different disease groups. Because we could not find a similar study evaluating its relationship with CTS in the literature, we believe ours is the first on this subject. In our study, when the patients with CTS were compared with the control group, we observed that there was a statistically significant relationship between the SII index and the development of CTS, and the risk of CTS increased 1.005 times as the SII index increased ($p < 0.001$).

In the study of Tepe *et al*. [24], C-reactive protein (CRP) and CRP/albumin ratios were evaluated in 50 controls and 50 patients with CTS, and no statistically significant difference supporting inflammation was detected, but it was suggested that re-evaluation of this hypothesis by increasing the number of patients with acute CTS might lead to decide anti-inflammatory treatment options in the treatment [24]. To investigate the contribution of the SII index to inflammation, its relationship with many different disease groups such as solid tumors, colorectal cancers, cerebrovascular diseases, and sinus vein thrombosis was evaluated, and it was emphasized that the SII index was a predictor of inflammation as a common result of the studies [25, 26]. In a study comparing inflammation markers in patients undergoing dialysis with and without CTS, it was shown that IL-1, tumor necrosis factor-alpha (TNF-α), and IL-6 levels were significant in patients with CTS and that inflammation had an effect on the process [27].

In a different study investigating the relationship of inflammation with CTS, 407 patients with CTS were compared with 206 controls. The correlation between CTS and NLR and PLR was evaluated. As a result of the study, it was reported that a 1 unit increase

in NLR level increased the risk of CTS by 1.7 times, and accordingly it was shown that the NLR level increased in severe CTS. In addition, it was suggested that subclinical systemic inflammation might cause CTS by increasing cytokines and growth factors and by causing fibrosis [28]. The contribution of inflammation in the pathophysiologic process of CTS has been demonstrated in many studies, but this evidence should be supported by studies with large case series.

When we compared the Boston symptom severity scale and functional status scale, a statistically significant difference was observed between the CTS groups ($p < 0.001$). This made us think that as the severity of compression increased in patients with CTS, the functionality of the patients might decrease and their quality of life might be adversely affected.

Studies have reported that there is a correlation between the increase in compression severity and the Boston functional score and that this relationship becomes stronger, especially in patients in whom deterioration in motor functions is evident. It has been suggested that the use of the Boston functional status scale in CTS would reflect the level of compression severity more effectively than grip strength in terms of assessing hand functions [29, 30].

Limitations

Our study had some limitations such as the limited number of patients, the inability to follow up for a long time, and the fact that the patients were not re-evaluated after the treatment.

CONCLUSION

In conclusion, we found a significant relationship between age, increased BMI, and the SII index and CTS in our study. In light of previous studies and the information obtained in our study, it was thought that advanced age and increased BMI were risk factors in CTS. According to the results of our study, in which the relationship between the SII index and CTS was evaluated for the first time, inflammation may play a role in the pathophysiology of CTS, and this suggests that systemic anti-inflammatory drugs could be beneficial in the treatment. However, to confirm the role of systemic inflammation in CTS, this hypothesis should be supported by multicenter randomized controlled prospective studies with a large number of patients.

Authors' Contribution

Study Conception: MKG, DYO; Study Design: MKG, DYO; Supervision: MKG, DYO; Funding: N/A; Materials: N/A; Data Collection and/or Processing: MKG; Statistical Analysis and/or Data Interpretation: MKG, DYO; Literature Review: MKG; Manuscript Preparation: SMKG, DYO and Critical Review: MKG, DYO.

Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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