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Original Article

Association of Monocyte/HDL-Cholesterol Ratio and Other Inflammatory Markers with Carpal Tunnel Syndrome

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Keywords: Carpal tunnel syndrome; inflammatory markers; monocyte/HDL-cholesterol ratio; neutrophil/lymphocyte ratio; platelet/lymphocyte ratio.

ABSTRACT

Objective: Carpal tunnel syndrome (CTS) is the most common compression neuropathy. Although its etiology is idiopathic, inflammatory processes are frequently implicated. Various inflammatory parameters obtained from complete blood count data are helpful in the diagnosis and prognosis of many inflammatory diseases. However, there is no consensus in the literature regarding the association between CTS and serum inflammatory markers. This study aimed to assess whether there were significant differences in various inflammatory markers between patients with severe CTS and healthy controls.

Methods: In this retrospective study, we compared 40 patients who had unilateral severe CTS which is diagnosed by clinic symptoms and positive electrodiagnostic tests (EDT) with 42 healthy patients. We investigated serum inflammatory markers such as the monocyte/HDL-cholesterol ratio (MHR), red blood cell distribution width (RDW), mean platelet volume (MPV), neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR), and monocyte/lymphocyte ratio (MLR) in patients with unilateral severe CTS compared to a control group.

Results: No significant difference was observed between the two groups in WBC, CRP, SED values analysed for the presence of active inflammation. No significant difference was observed between the two groups in the analysed parameters PLT, NEU, LYM, MON, and HDL ($p>0.05$). When inflammatory parameters were analysed, no difference was observed between the two groups in RDW, MPV, HDL, NLR, PLR, and MLR values ($p>0.05$).

Conclusion: No significant association was found between serum inflammatory markers in the two groups, suggesting that inflammation in CTS may be localized.

INTRODUCTION

Carpal tunnel syndrome (CTS) is a prevalent compression neuropathy caused by compression of the median nerve at the wrist level.[1] It is observed in approximately 3.8% of the general population and is more common in women.[2] Although most CTS are idiopathic, metabolic and inflammatory conditions including diabetes mellitus, hypothyroidism, pregnancy, menopause, obesity, renal failure, rheumatic diseases, alcoholism, vitamin deficiencies or toxicity are involved in the etiology. In addition, acute post-traumatic, mass, or occupational factors may cause carpal tunnel syndrome.[3] The initial symptoms of CTS include pain, numbness, and paresthesias along the median nerve pathway. As CTS is progressive in many patients, it can cause permanent sensory and motor function loss in the hand if not diagnosed and treated early.

Median nerve microcirculation damage, median nerve connective tissue compression, and synovial tissue hypertrophy due to increased pressure in the carpal tunnel have been advocated in the pathophysiology of CTS.[4] In addition, according to a study conducted in patients with diabetes mellitus, internal factors that damage the nerve, such as deficiency of neurotrophic factors such as growth factor, have been reported to be effective in the pathophysiology of CTS.[5]

Whether idiopathic CTS is an inflammatory condition remains controversial. The relationship between inflammatory parameters derived from complete blood count and CTS is unclear, with no consensus in the literature. This study aimed to assess whether there were significant differences in various inflammatory markers between patients with severe CTS and healthy controls.

MATERIALS AND METHODS

This study was carried out between January 2023 and May 2024. We compared 40 unilaterally severe contralateral normal CTS patients diagnosed by electrodiagnostic testing (EDT) and clinically confirmed clinical CTS symptoms with 42 healthy controls. Patients were classified using the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) guideline.[6] This study received approval from the local ethics committee with the Helsinki Declaration (Approval no: 2024-706, dated: 31.07.2024). The inclusion criteria for this study were: (i) age >18 years, (ii) pain or tingling in unilateral corresponding to the median nerve trace in the study group, (iii) electrophysiologically confirmed diagnosis of severe CTS in the study group and excluded diagnosis of CTS in the control group, (iv) laboratory values investigated less than 1 month after EDT. Exclusion criteria were history of polyneuropathy or radiculopathy, hematological diseases, chronic inflammatory and connective tissue diseases, corticosteroid or immunomodulator use, malignancy, presence of active infection, and pregnancy.

The parameters analysed included MHR, RDW, MPV, NLR, PLR, and MLR. In addition, sedimentation, C-reactive protein (CRP), and WBC (white blood cell) parameters were also analysed to evaluate the absence of active inflammation.

After the study plan was made, the patients were retrospectively screened. A total of 846 patients who underwent median nerve EDT were identified. Of the identified patients, 219 were excluded due to mild and 134 due to moderate CTS. Of the 98 patients with normal EDT, 56 were excluded for various reasons (neuropathy in 24 patients, elevated CRP in 5 patients, deficient laboratory values in 21 patients, drug use in 4 patients, rheumatological disease in 2 patients) and the remaining 42 patients were included in the control group. Of the 121 patients with severe CTS on EDT, 81 patients were excluded for various reasons (neuropathy in 33 patients, elevated CRP, WBC in 9 patients, deficient laboratory values in 29 patients, drug use in 5 patients, rheumatological disease in 5 patients) and the remaining 40 patients were included in the study group (Fig. 1).

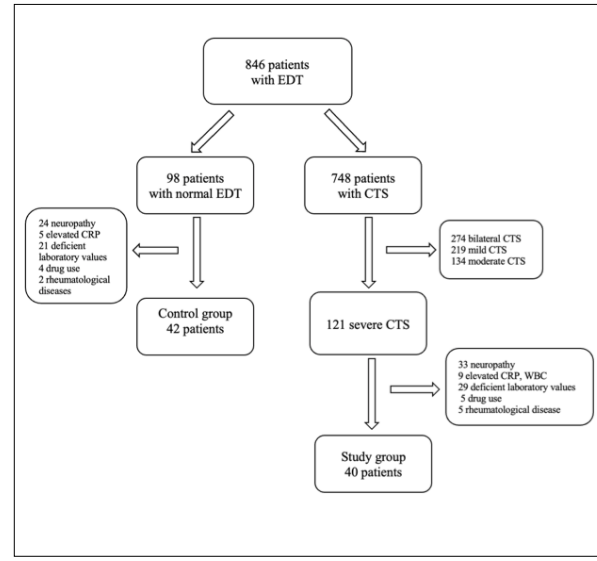

Figure 1. Flow chart.

Monocyte-to-HDL-Cholesterol Ratio (MHR) and Other Inflammatory Markers

The MHR, calculated by dividing the number of monocytes by the HDL cholesterol level in peripheral blood, is a novel marker of systemic inflammation. Monocytes play a role in the release of pro-inflammatory and pro-oxidative cytokines at sites of inflammation and are critical in chronic inflammation. Conversely, HDL has anti-inflammatory and antioxidant effects that inhibit monocyte production and mobilization.[7]

Due to its accessibility, the MHR has recently been used as a clinical indicator in conditions such as atherosclerosis, coronary artery disease, ophthalmopathies, depression, schizophrenia, Parkinson's disease, osteoporosis, and deep vein thrombosis.[7-9] However, its effectiveness in some diseases, such as seborrhoeic dermatitis, has not been demonstrated.[10] Its association with CTS has not been detected as far as investigated in the literature.

Recent studies have shown that red blood cell distribution width (RDW), mean platelet volume (MPV), neutrophil/lymphocyte ratio (NLR, neutrophil count/lymphocyte count), platelet/lymphocyte ratio (PLR, platelet count/lymphocyte count) and monocyte-to-lymphocyte ratio (MLR, monocyte count/lymphocyte count) are accessible and cost-effective systematic inflammation markers reflecting the degree of inflammation.[11] High neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) values indicate increased inflammation.[12] Calculation of NLR is a highly accessible method compared to other inflammatory cytokines including IL-6, IL-1β, and TNF-α.[13]

Statistical Analysis

Data was analyzed using IBM SPSS 25.0 (Armonk, NY: IBM Corp.) statistical software package. Descriptive statistical methods (frequency, percentage, mean, standard deviation, median, min-max) were used, and the Chi-Square (χ^2) test was applied for comparison of qualitative data. The normality of the data distribution was evaluated with the Kolmogorov-Smirnov test, skewness-kurtosis, and graphical methods (histogram, Q-Q Plot, Stem and Leaf, Boxplot). In the study, the Independent Samples t-test was used for intergroup comparisons of quantitative data showing normal distribution, while the Mann-Whitney U test was used for non-normally distributed data. The statistical significance level was set at p=0.05.

Power analysis was performed with G*Power 3.1.9.7 (Franz Faul, University of Kiel, Germany) statistical software; with n1=40 (62.6±12.6), n2=42 (49.2±12.6), SD=14.2, α=0.05, Effect Size (d) = 0.94, and power was calculated as 98%.

Table 1. Demographic characteristics and comparisons between groups

	Severe CTS (n=40)	Control (n=42)	p
Sex			
Female	29 (72.5%)	31 (73.8%)	1.000[a]
Male	11 (27.5%)	11 (26.2%)	
Age (year)	62,6 (SD 12.6)	49.2 (SD 12.6)	<0.001[b]
Site			
L	22 (55.0%)	22 (52.4%)	0.987[a]
R	18 (45.0%)	20 (47.6%)	

[a]: Chi-Square Test (n (%)), [b]: Independent Samples t Test (Mean SD).

RESULTS

The demographic data of the study are shown in Table 1 and it was observed that there was a statistically significant difference between the groups in terms of age values ($p<0.05$) and the patients in the severe CTS group were older. It was determined that 73.2% (n=60) of the patients who participated in the study were female.

The median values of the parameters analysed for the presence of active inflammation were WBC 6.8x10^9, CRP 3.0 mg/L, and SED 12.0 mm/h, and no significant difference was observed between the two groups (Table 2). In the analysed parameters, no difference was observed between the two groups in PLT, NEU, LYM, MON, and HDL values ($p>0.05$, Table 2).

When inflammatory parameters were evaluated, no difference was observed between the two groups in RDW, MPV, HDL, NLR, PLR, and MLR values ($p>0.05$, Table 2, Fig. 2).

MHR was 8.6 (2.0–18.1) in the study group and 7.7 (2.9–17.5) in the control group; NLR was 1.8 (0.9–5.3) in the study group and 1.8 (0.9–5.7) in the control group; PLR was 131.5 (SD 48.6) in the study group and 121.8 (SD 38.3) in the control group; MLR was 0.2 (0.1–0.6) in the study group and 0.2 (0.1–0.4) in the control group. No significant differences were found between these systemic inflammatory markers.

DISCUSSION

In this study, we found that serum inflammatory markers were not significantly different in patients with severe carpal tunnel syndrome compared to the control group. An increase in the frequency of CTS is observed with inflammatory diseases.[14] MHR, NLR, and PLR are used as indicators of systemic inflammation.[15,16] In this study, these markers did not show a significant difference in patients with severe CTS compared to the control group,

Table 2. Comparisons between groups

	Severe CTS (n=40)	Control (n=42)	p
WBC (10^9/L)	7.3 (SD 2.0)	7.3 (SD 1.8)	0.973[b]
CRP (mg/L)	3.0 (1.0 – 18.0)	3.0 (0.2 – 34.9)	0.167[c]
SED (mm/h)	14.5 (2.0 – 58.0)	12.0 (2.0 – 51.0)	0.140[c]
RDW (fL)	14.0 (12.4 – 25.1)	13.5 (12.7 – 40.8)	0.233[c]
MPV (fL)	10.2 (SD 1.2)	10.4 (SD 1.3)	0.348[b]
MHR	8.6 (2.0 – 18.1)	7.7 (2.9 – 17.5)	0.330[c]
NLR	1.8 (0.9 – 5.3)	1.8 (0.9 – 5.7)	0.915[c]
PLR	131.5 (SD 48.6)	121.8 (SD 38.3)	0.319[b]
MLR	0.2 (0.1 – 0.6)	0.2 (0.1 – 0.4)	0.127[c]
PLT (10^9 /L)	273.2 (SD 60.1)	272.1 (SD 65.6)	0.938[b]
NEU (10^9 /L)	4.1 (2.2 – 8.4)	4.3 (2.5 – 12.5)	0.721[c]
LYM (10^9 /L)	2.1 (0.9 – 5.2)	2.3 (1.1 – 4.5)	0.439[c]
MON (10^9/L)	0.4 (0.2 – 0.9)	0.4 (0.2 – 0.7)	0.351[c]
HDL (mg/dL)	53.1 (SD 10.6)	54.2 (SD 10.5)	0.657[b]

[a]: Chi-Square Test (n (%)), [b]: Independent Samples t Test (Mean SD), [c]: Mann-Whitney U Test (Median (Min-Max)).

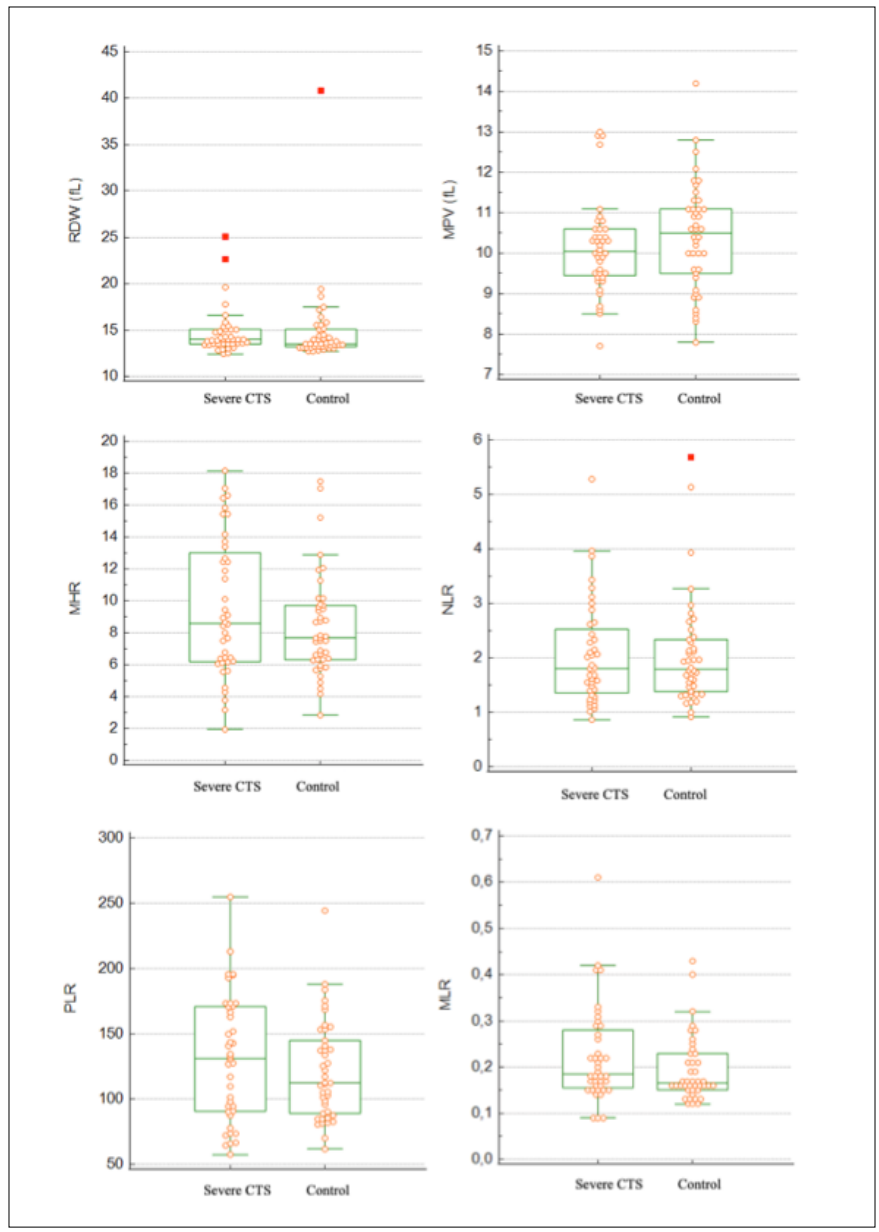

Figure 2. Comparisons between groups.

suggesting that the process might be chronic and limited to the local area.

When the literature is analysed, there is no study comparing the MHR value with CTS. However, in one study, HDL levels were investigated in CTS patients, and the authors found no significant association between them.[17] In this study, no difference was found between HDL and MHR values between the two groups.

There are conflicting results in the literature in studies investigating the relationship between inflammatory markers and CTS.[18-20] In a study evaluating the relationship between idiopathic CTS severity and inflammatory markers, it was found that there was a positive correlation between CTS severity and NLR, PLR, age and BMI, and a 1-unit increase in NLR level was associated with a 1.7-fold increase in the incidence of CTS. However, although Gunes et al.[18] found a correlation between CTS and NLR and PLR in their study, the biggest limitation of their study was the lack of a control group. Comparison with the control group could have excluded this relationship. On the other hand, there are also studies suggesting that NLR and PLR values are not associated with CTS.[19,20] In this study, no correlation was shown between NLR, PLR levels and the presence of carpal tunnel syndrome.

NLR illustrates the balance between neutrophils and lymphocytes, acting as an indicator of the adaptive immune response during various disease states. NLR can help early

detection of processes such as cancer, infection, inflammation, and stress.[21] PLR is considered a marker of systemic inflammation and platelet aggregation, reflecting the interplay between inflammatory coagulation mechanisms and platelet activation in the context of systemic immune responses.[22] Gunes et al.[18] found a correlation between NLR and PLR and CTS in their study. However, in this study, no relationship was found between these two inflammatory markers and CTS.

MPV indicates platelet size and is associated with platelet function and activation. Large platelets can synthesize more pro-thrombotic factors. The literature contains numerous studies on the role of MPV in thrombosis, inflammation and various diseases.[23,24] In a study conducted on a geriatric patient group, MPV values were found to be significantly higher in CTS patients compared to the control group.[25] This was thought to be an indicator of inflammation in CTS patients. In this study, no significant difference was found between the patient and control groups in MPV values.

MLR is another parameter used for systemic inflammation.[26] RDW is a measure of the heterogeneity in red blood cell (RBC) size and helps assess the presence and severity of inflammation and infection in conditions such as sepsis and autoimmune diseases.[19] In this study, no relationship was found between these 2 inflammatory parameters and CTS.

The pathophysiology of CTS involves various mechanisms including intraneural ischemia, reactivity of Schwann cells, demyelination, and production of neuro-inflammatory agents.[20] In a study, the authors investigated intraoperative prostaglandin E2 (PGE2), IL-1 and IL-6 levels in patients with idiopathic CTS and analysed the relationship between these levels and histopathology. According to this study, the amount of PGE2 and IL-6 in the tenosynovium of CTS patients was significantly higher than in the control group. However, there was no difference in serum PGE2 levels between these two groups. The difference between local and systemic prostaglandin production was explained by the fact that these compounds are locally effective and rapidly metabolised systemically.[27] It was shown that there were no significant changes in serum levels of inflammatory cytokines (IL-1, IL-6, IL-10, and TNFα) in patients with CTS, and that the role of these parameters in CTS is still unclear.

In another study on histopathological examination of the subsinovial connective tissue of patients with idiopathic carpal tunnel syndrome, a significant increase was found in fibroblast density and collagen fibril dimensions as well as intima thickening and vascular proliferation; in addition, the presence of collagen type III in the subsinovial connective tissue was demonstrated. However, the presence of inflammatory cells was not detected.[28] In addition, the results of other studies in which other synovial histopathological specimens were examined also reported that inflammatory cells were not found.[29] The results of these two studies are compatible with this study and we think that carpal tunnel syndrome is not a systematic inflammatory process.

Limitations

The retrospective nature of this study and the small number of patients are our most important limitations. However, the power analysis was still found to be of sufficient strength. In this study, only unilateral severe CTS patients were included and serum levels do not give an idea about the acute period because these patients usually have complaints for a long time. Also, local biomarkers were not evaluated in CTS patients. Although the reference ranges of the measured parameters do not show significant changes with age, the fact that the severe CTS group is older than the control group may have a negative effect on inflammatory parameters. Another possible limitation is that the participants included in the study were analysed with a single laboratory sample obtained at the time of their initial presentation without long-term follow-up.

Conclusion

In conclusion, no association was found between MHR and other serum inflammatory markers and severe CTS in this study. Although the small number of study groups limits the results, it is a reference for multicentre and larger study groups.

Ethics Committee Approval

The study was approved by the Ankara Etlik City Hospital Ethics Committee (Date: 31.07.2024, Decision No: AEŞH-BADEK-2024-706).

Informed Consent

Retrospective study.

Peer-review

Externally peer-reviewed.

Authorship Contributions

Concept: A.A., A.B.A.; Design: A.A., A.B.A.; Supervision: A.A.; Fundings: A.A., A.B.A.; Materials: A.B.A.; Data collection &/or processing: A.A., A.B.A.; Analysis and/or interpretation: A.A., A.B.A.; Literature search: A.A., A.B.A.; Writing: A.A., A.B.A.; Critical review: A.A., A.B.A.

Conflict of Interest

None declared.

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Monosit/HDL-Kolesterol Oranı ve Diğer İnflamatuvar Belirteçlerin Karpal Tünel Sendromu ile İlişkisi

Amaç: Karpal tünel sendromu (KTS) en sık görülen kompresyon nöropatisidir. Etiyolojisi idiyopatik olmakla birlikte, inflamatuvar süreçler sıklıkla rol oynamaktadır. Tam kan sayımı verilerinden elde edilen çeşitli enflamatuvar parametreler, birçok enflamatuvar hastalığın tanı ve prognozunda yardımcı olmaktadır. Ancak, literatürde KTS ile serum inflamatuvar belirteçleri arasındaki ilişki konusunda bir fikir birliği yoktur. Bu çalışmanın amacı, şiddetli KTS'li hastalar ile sağlıklı kontroller arasında çeşitli enflamatuvar belirteçler açısından anlamlı farklılıklar olup olmadığını değerlendirmektir.

Gereç ve Yöntem: Bu retrospektif çalışmada, elektrodiagnostik test (EDT) ile klinik olarak doğrulanmış KTS semptomları olan 40 tek taraflı şiddetli kontralateral normal KTS hastasını 42 sağlıklı kontrol ile karşılaştırıldı. Tek taraflı şiddetli KTS'li hastalarda monosit/HDL-kolesterol oranı (MHR), kırmızı kan hücresi dağılım genişliği (RDW), ortalama trombosit hacmi (MPV), nötrofil/lenfosit oranı (NLR), trombosit/lenfosit oranı (PLR) ve monosit/lenfosit oranı (MLR) gibi serum inflamatuvar belirteçleri kontrol grubuna kıyasla araştırıldı.

Bulgular: Aktif inflamasyon varlığı için analiz edilen WBC, CRP, SED değerlerinde iki grup arasında anlamlı bir fark gözlenmemiştir. Analiz edilen PLT, NEU, LYM, MON ve HDL parametrelerinde iki grup arasında anlamlı bir fark gözlenmedi ($p>0.05$). İnflamatuvar parametreler analiz edildiğinde, RDW, MPV, HDL, NLR, PLR ve MLR değerlerinde iki grup arasında fark gözlenmedi ($p>0.05$).

Sonuç: İki gruptaki serum inflamatuvar belirteçleri arasında anlamlı bir ilişki bulunmadı ve bu durum KTS'deki inflamasyonun lokalize olabileceğini düşündürdü.

Anahtar Sözcükler: Enflamatuvar belirteçler; karpal tünel sendromu; monosit/HDL-kolesterol oranı; nötrofil/lenfosit oran; trombosit/lenfosit oranı.