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THE ROLE AND ASSESSMENT OF SYSTEMIC IMMUNE-INFLAMMATION INDEX AS A NOVEL INFLAMMATORY MARKER IN PERIPHERAL AND CENTRAL FACIAL PARALYSIS

Periferik ve Santral Fasiyal Paralizide Yeni Bir İnflamatuar Belirteç Olarak Sistemik İmmün-İnflamasyon İndeksinin Rolü ve Değerlendirilmesi

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ABSTRACT

Systemic immune-inflammation index (SII) is a novel inflammatory marker and is commonly used in clinical management such as prognosis and response to therapies. In this study, the outcomes and correlations of inflammatory markers in central and peripheral facial paralysis were evaluated. The study was planned retrospectively and cross-sectionally. Totally 133 patients (group 1; 53, group 2; 80) were included in the study. The neutrophil counts were 4.7 ± 1.6 and 3.7 ± 1.6 (p=0.001), and the lymphocyte counts were 2.8 ± 0.8 and 3.3 ± 1 (p=0.007) in groups 1 and 2, respectively. While Neutrophil lymphocyte ratio (NLR) was 1.8 ± 0.9 in patients with central facial paralysis, NLR was analysed as 1.4 ± 1 in patients with peripheral facial paralysis (p=0.001). Systemic immune-inflammation index was determined as 529.5 ± 297.4 in the first group and 408.2 ± 228.1 in the second group (p=0.029). There was a positive correlation between NLR and SII (r:0.787, p<0.001). Peripheral facial paralysis was evaluated according to the H-B scale [(median:3, min-max:2-6)]. In conclusion, an elevated level of inflammatory markers was remarkable in pathologies affecting the central nervous system. NLR and SII values were increased in central facial paralysis.

Keywords: Inflammation, Facial nerve, Facial paralysis, Neutrophil Lymphocyte Ratio (NLR), Systemic Immune-Inflammation Index (SII).

ÖZ

Sistemik immün-inflamasyon indeksi (SII) yeni bir inflamatuar belirteç olup, genellikle prognoz ve tedaviye yanıt gibi klinik takiplerde kullanılır. Bu çalışmada santral ve periferik fasiyal paralizilerde inflamatuvar belirteçlerin sonuçları ve korelasyonları değerlendirildi. Çalışma retrospektif ve kesitsel olarak planlandı. Araştırmaya toplam 133 (grup 1; 53, grup 2; 80) hasta dâhil edildi. Nötrofil sayısı 1. ve 2. gruplarda sırasıyla; 4.7 ± 1.6 ve 3.7 ± 1.6 (p=0.001) iken, lenfosit sayıları 2.8 ± 0.8 ve 3.3 ± 1 (p=0.007) tespit edildi. Santral fasiyal paralizisi olanlarda Nötrofil lenfosit oranı (NLR) 1.8 ± 0.9 iken, periferik fasiyal paralizisi olanlarda NLR 1.4 ± 1 (p=0.001) olarak analiz edildi. Sistemik immün-inflamasyon indeksi; birinci grupta 529.5 ± 297.4, ikinci grupta 408.2 ± 228.1 olarak tespit edildi (p=0.029). NLR ile SII arasında pozitif korelasyon (r:0.787, p<0.001) vardı. Periferik fasiyal paralizisi olanlar H-B skalasına göre değerlendirildi [(median:3, min-max:2-6)]. Sonuç olarak, merkezi sinir sistemini etkileyen patolojilerde inflamatuar belirteçlerdeki artış dikkat çekmiştir. NLR ve SII değerleri santral fasiyal paralizide daha yükselmiştir.

Anahtar kelimeler: İnflamasyon, Fasiyal paralizi, Fasiyal sinir, Nötrofil Lenfosit Oranı (NLR), Sistemik İmmün-İnflamasyon İndeksi (SII).

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INTRODUCTION

Coronary Facial paralysis is phenotypically divided into two basic etiologies. Different clinical presentations appear due to pathologies that occur during the anatomical course of the facial nerve. Central type paralysis occurs when the lesion is located in the supranuclear location of the VIIth cranial nerve and peripheral type paralysis occurs in the infranuclear location. While ipsilateral involvement is observed in the peripheral type, contralateral involvement is observed in the central type. The most common cause of peripheral facial paralysis (PFP) is Bell's palsy. The most common known cause of Bell's palsy is herpes simplex virus (HSV) reactivation on the facial nerve (Kınar, Ulu, Bucak & Kazan, 2021). It has been suggested that HSV infection induced inflammation-mediated processes against myelin on the facial nerve (Stjernquist-Desatnik, Skoog & Aurelius, 2006). In recent studies, neutrophil-to-lymphocyte ratio (NLR) has been evaluated as an inflammatory marker in facial paralysis (Kınar et al., 2021). It has also been suggested by some authors that NLR can be used to estimate prognosis in PFP (Oya et al., 2019). Systemic immune-inflammation index (SII) is a new inflammatory index used to determine prognosis in malignancy and inflammatory diseases (Kınar et al., 2021). In a study of patients with malignancy, SII was shown to be directly associated with survival and poor prognosis ($p<00.1$) (Yang, Chang, Meng, Gao & Wang, 2018). There are limited number of publications evaluating SII in peripheral facial paralysis and investigating response to prognosis or treatment. In our study, the first examination findings and basal hemogram parameters were evaluated. Inflammatory parameters; platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), NLR and SII were calculated between patients with central facial paralysis (CFP) and PFP.

Ischemic stroke is the primary etiology of central facial paralysis, followed by multiple sclerosis-related lesions involving the brain stem, masses, and cavernomas. Central nervous system (CNS) injury is usually followed by a complex series of reactions between inflammatory processes and the immune system. Ischemic stroke causes an inflammatory response in the brain parenchyma, arterial wall and occluded vascular structures, whereas in a demyelinating plaque, immunological reactions characterized by an increase in lymphocytes ocur (Dehghanei, ArefNezhad & Motedayyen, 2020). There may also be migration of lymphocytes into the CNS due to impaired of the blood brain barrier (Pagliano, Spera, Ascione & Esposito, 2020). In a study, NLR was evaluated between patients with ischemic stroke and those with hemorrhagic cerebrovascular disease. As a result, it was found that lymphocyte and leukocyte levels were high in patients with ischemic stroke and NLR was low (Dehghanei et al., 2020).

Our primary aim in this study was to show the differences of NLR and SII as inflammatory markers between peripheral and central types in facial paralysis. We also investigated the correlation of these inflammatory indexes with the clinical severity of PFP. In particular, we investigated the relationship between SII and the clinical severity of PFP. Our hypothesis is that NLR and SII are lower in PFP because of the increase in lymphocytes compared to CFP.

MATERIAL AND METHOD

This study was analyzed by retrospectively reviewing the electronic files of patients admitted for facial paralysis (central and peripheral) between 2020 and 2022. Participants were selected from patients admitted to the departments of otorhinolaryngology and neurology. The criteria of the Declaration of Helsinki were observed throughout the study. Approval was obtained from the ethics committee of the university before the start of the study. (Ethics committee approval: 2023/4600) Patients with central type of facial paralysis were classified as group 1 and those with peripheral type as group 2. A total of 321 patients with facial paralysis (central+peripheral) were analyzed. A total of n:133 (group 1; 53, group 2; 80) patients who qualified the criteria were analyzed. Inclusion criteria were age between 18-59 years, complete examination findings and basal hemogram, CRP (C-reactive protein), and sedimentation parameters. Exclusion criteria were the use of medications that may impair hemogram values (chemotherapeutic agents, immunosuppressives, etc.), hematologic malignancy, and a history of recurrent facial paralysis. Exclusion criteria were incompatibility between the diagnosis and examination findings, more than 3 days after the disease (subacute stage). The etiology of central facial paralysis was categorized as 1. ischemic stroke, 2. hemorrhagic stroke, 3. demyelinating lesion, 4. other (newly diagnosed malignancy, cavernoma, etc.). The etiology of peripheral facial paralysis was categorized as 1. Bell's palsy, 2. Acute otitis media, 3. External otitis, 4. Trauma, 5. Malignancy, 6. Other. The House-Brackmann (HB) scale (grades 1-6) was used to describe the clinical severity of peripheral facial paralysis (House & Brackmann, 1985). Furthermore, the severity of clinical involvement was divided into two categories according to the HB scale (mild-moderate severity, HB score $\leq$ 3, and severe severity, HB score >3). Systemic inflammatory index was calculated according to the formula: platelet count x neutrophil count / lymphocyte count (Dehghanei et al., 2020). Blood samples taken peripherally from the patients were analyzed for CRP, sedimentation and hemogram parameters on Abbott Laboratuvar®, Arkray® devices.

Statistical Analysis

SPSS® 26.0 program was used for all statistical data and analyses in the study. Kolmogorov-Smirnov test (n<50) was used for normality distributions. Arithmetic mean, standard deviation, minimum and maximum values were used to summarize numerical data, frequency distributions and percentages were used to summarize categorical data (Case summuries). Independent Samples t-test was used for the comparison of group 1 and group 2, while Mann-Whitney U test was used for non-parametric tests. Independent categorical variables were analyzed by Chi-Square test [min. expected count: Pearson Chi-Square ≥ 25, Continuity Correction if between 5-25, Fischer's Exact Test for ≤ 5]. Spearman's test was used for correlation analysis. p<0.05 was considered significant in all statistical interpretations.

RESULTS

The study included 53 patients (M/F:27/26) in group 1 and 80 patients (M/F:32/48) in group 2 (p=0.287). The median age of the participants was 43 (min-max:19-59) and 35 (min-max:20-58) years in groups 1 and 2, respectively (p=0.088). The most common etiology in group 1 was ischemic stroke, while Bell's palsy was the most common etiology in group 2 (Table 1).

Table 1. Individual and Etiological Characteristics of the Groups

Age	**Group 1**			**Group 2**		
	n	**Mean ± SD**	**Median (min-max)**	**n**	**Mean ± SD**	**Median (min-max)**
Male	27	39.07 ± 12.27	35 (21-59)	32	35.78 ± 11.23	32 (21-589
Female	26	42.00 ± 12.78	43 (19-58)	48	36.94 ± 12.49	38 (20-58)
Total	53	40.51 ± 12.49	43 (19-59)	80	36.48 ± 11.94	35 (20-58)
Etiology	33 (62%)	Ischemic stroke		31 (38.7%)	Bell's palsy	
	7 (13.2%)	Intracerebral hemorrhage		14 (17.5%)	AOM	
	2 (3.7%)	Demyelinating diseases		8 (10%)	EO	
	11 (20.7%)	Other		6 (7.5%)	Trauma	
				5 (6.2)	Malignancy	
				4 (5%)	Other	
				12 (15%)	Unknown	

Examination the hemogram parameters between the two groups, it was remarkable that the neutrophil and lymphocyte values were different. The mean neutrophil count: 4.7 ± 1.6 and 3.7 ± 1.6 in the 1st and 2nd groups, respectively (p=0.001). Mean lymphocyte counts were 2.8 ± 0.8 and 3.3 ± 1 (p=0.007) in the 1st and 2nd groups, respectively. Among the inflammatory markers, NLR and SII increased in favour of group 1. While the NLR: 1.8 ± 0.9 in patients with central facial paralysis, the NLR: 1.4 ± 1 (p=0.001) in PFP. SII of the first group (529.5 ± 297.4)

was higher than the second group (408.2 ± 228.1) (p=0.029). In addition, when the median SII values of both groups [1st group: 492.6 (min-max: 104.1-1334.3), 2nd group: 355.7 (min-max: 79.9-843.2)] were analysed, it was observed that the 1st group was significantly higher (Table 2).

Table 2. Comparison of Facial Palsy Types and Inflammatory Markers

	Group 1		Group 2		Test sta.	p
	Mean ± SD	Median (min-max)	Mean ± SD	Median (min-max)		
Hgb	13.7 ± 1.3	13.6 (11.4-16.8)	14.1 ± 1.7	14 (11.3-16.7)	1808.5	0.152
Hct	44.9 ± 4.4	44.7 (37.1-51.7)	44.7 ± 4	44.5 (37.1-51.7)	2060.5	0.784
Neu	4.7 ± 1.6	5.2 (1.8-7.3)	3.7 ± 1.6	3.2 (1.7-7.1)	1385.5	0.001
Lym	2.8 ± 0.8	2.8 (1.2-4.2)	3.3 ± 1	3.5 (1.1-4.8)	1537	0.007
Mo	0.7 ± 0.3	0.7 (0.2-1.1)	0.7 ± 0.2	0.7 (0.2-1.1)	1839	0.193
Plt	287.5 ± 75.2	271.9 (164.2-390.6)	276.9 ± 74	278 (153.4-394.3)	1891	0.293
WBC	8.7 ± 3.2	8.6 (4-18.2)	8.3 ± 2.8	8.3 (3.9-18.9)	2011.5	0.618
NLR	1.8 ± 0.9	1.7 (0.4-3.8)	1.4 ± 1	1 (0.4-5.4)	1412.5	0.001
MLR	0.3 ± 0.2	0.3 (0.1-0.9)	0.2 ± 0.1	0.2 (0.1-0.8)	1829	0.181
PLR	111.5 ± 47.1	105.3 (39.1-229.7)	101.6 ± 42.3	101.1 (36.1-216.8)	1831	0.184
SII	529.5 ± 297.4	492.6 (104.1-1334.3)	408.2 ± 228.1	355.7 (79.9-843.2)	1645	0.029
ESR	16.5 ± 10.9	18 (1-47)	16.3 ± 8.4	16 (1-41)	2085	0.872

Mann Whitney U test

SD: Standart deviation, Test sta: Test statistics, Hgb: hemoglobin g/dL, Hct: Hematocrit %, Neu: Neutrophil x 10^3/uL, Lym: lymphocyte x 10^3/uL, Mo: Monocyte x 10^3/uL, Plt: Platelet x 10^3/uL, WBC: White blood cell x 10^3/uL, NLR: Neutrophil-to-lymphocyte rate, MLR: Monocyte-to-lymphocyte rate, PLR: Platelet-to-lymphocyte rate, SII: Systemic immune-inflammation index, ESR: Erythrocyte sedimentation rate/ mm/h

The correlation of inflammatory markers was analysed, a strong positive correlation was observed between NLR and SII (r:0.787, p<0.001). Another strong positive correlation was detected between SII and PLR (r:0.748, p<0.001) (Table 3). However, the NLR was not found to be different in the comparison between the groups, this result is suggestive and it may be necessary to increase the sample size or to compare the PLR value of healthy individuals with these groups. Thus, a clearer assessment of PLR as an inflammatory marker can be made.

Patients with peripheral facial paralysis were graded by H-B scale according to clinical phenotype. There were 39 patients with House-Brackmann score ≤ 3 and 41 patients with H-B score>3. H-B score was analysed as median:3 (min-max:2-6). There was no significant difference between the inflammatory parameters of patients with mild to moderate paralysis (H-B score ≤ 3) and those with severe paralysis (H-B score >3) (Table 4).

Table 3. Correlation Effect of Inflammatory Markers

	NLR	MLR	PLR	SII
NLR	1			
MLR	0.446**	1		
PLR	0.467**	0.477**	1	
SII	0.787**	0.441**	0.748**	1

Spearman's Correlation Test
** Correlation is significant at the 0.01 level (2-tailed).

Table 4. Assessment of the Baseline Examination of the 2nd Group According to the House-Brackmann Facial Paralysis Scale and comparison of the Paralysis Grade with Inflammatory Markers

	H-B scale ≤ 3 (n:39)		H-B scale >3 (n:41)		Test sta.	P
	Mean ± SD	Median (min-max)	Mean ± SD	Median(min-max)		
Neu	3.7 ± 1.5	3.2 (1.7-7.1)	3.8 ± 1.7	3.2 (1.7-7.1)	788.5	0.916
Lym	3.2 ± 1	3.3 (1.1-4.8)	3.3 ± 1	3.6 (1.1-4.8)	766.5	0.75
Mo	0.7 ± 0.2	0.7 (0.3-1.1)	0.7 ± 0.2	0.7 (0.2-1.1)	757	0.678
Plt	269.4 ± 71.9	262.5 (157.2-391.3)	284.1 ± 76.1	293 (153.4-394.3)	708.5	0.381
WBC	9.1 ± 2.9	8.9 (4-18.9)	7.5 ± 2.4	7.5 (3.9-12.8)	555	0.019
NLR	1.4 ± 1.1	1 (0.4-5.4)	1.4 ± 1	1.1 (0.4-5.4)	797.5	0.985
MLR	0.3 ± 0.1	0.2 (0.1-0.8)	0.2 ± 0.1	0.2 (0.1-0.8)	732.5	0.519
PLR	100.8 ± 42	96.5 (43.2-216.5)	102.4 ± 43.1	101.7 (36.1-216.8)	782	0.866
SII	408.9 ± 226.5	340.6 (79.9-843.2)	407.5 ± 232.4	359.3 (86.6-843.2)	794.5	0.962

Mann Whitney U test
SD: Standart deviation, Test sta: Test statistics, H-B scale: House-Brackmann scale, Neu: Neutrophil x 10^3/uL, Lym: lymphocyte x 10^3/uL, Mo: Monocyte x 10^3/uL, Plt: Platelet x 10^3/uL, WBC: White blood cell x 10^3/uL, NLR: Neutrophil-to-lymphocyte rate, MLR: Monocyte-to-lymphocyte rate, PLR: Platelet-to-lymphocyte rate, SII: Systemic immune-inflammation index

DISCUSSION

The systemic immune-inflammation index is a new marker of inflammation that has been used for several years. Nowadays, it is generally used in clinical follow-up such as prognosis and response to treatment in systemic diseases. When we examine the recent studies on this subject, we see that SII has an increasing importance in gastro intestinal malignancies and autoimmune diseases. The first article examining the relationship between facial paralysis and SII was published in 2020. In that study, the role of SII in PFP was discussed (Kınar et al., 2021).

In PFP, involvement on one side of the face is expected, symptoms peak in the first 1 week and improvement is observed after 2-3 weeks. The most common cause of PFP is Bell palsy (House & Brackmann, 1985). In some studies, various pathologies and theories have been proposed for the etiology of Bell's palsy, including viral infections, exposure to severe cold weather, immune-inflammatory-related theory, and ischemia-related theory (Zhang et al., 2020). According to the viral infection theory, latent HSV-1, HSV-2 and VZV (varicella-zoster virus) reactivation is observed on the facial nerve (Looker et al., 2015). In the inflammation

theory, inflammatory cell destruction in 'facial nerve neuritis' after damage to the myelin sheath of the facial nerve (compression in the fallopian canal) is proposed (Zhang et al., 2020). One study reported elevated serum cytokine levels including interleukin-1 (IL-1), IL-6 and tumor necrosis factor-alpha in Bell's palsy patients compared to the control group (Yılmaz et al., 2002). Based on these data, we examined the status of inflammatory markers and indices in supranuclear lesion of the facial nerve, SFP. In our study, the neutrophil count in group 1 (4.7 ± 1.6) was higher than in group 2 (3.7 ± 1.6) (p=0.001). Another noteworthy hemogram parameter was lymphocytes; lymphocyte count was 2.8 ± 0.8 in group 1 and 3.3 ± 1 in group 2 (p=0.007). The increase in lymphocyte count in the second group suggests that viral and autoimmune pathogenesis play a role in PFP. Considering that autoimmune patients were excluded in the study, we may think that viral pathogenesis came to the forefront. When we evaluate this situation in terms of group 1, we can say that neutrophil-macrophage mediated reactions are present in cases affecting the CNS (most commonly ischemic stroke). From another perspective, we can suggest that there is no early lymphocyte infiltration in the blood brain barrier. In a publication supporting this idea, the authors blamed neutrophils as a component of the inflammatory system in reperfusion and cerebral ischemia (Emsley, Smith, Tyrrell & Hopkins, 2008; Urra, Cervera, Villamor, Planas & Chamorro, 2009).

In a 2014 case-control study of 25 patients with Bell's palsy and healthy individuals, the NLR value was 2.16 ± 0.8 in patients and 1.36 ± 0.48 in healthy individuals (p=0.001). In the same study, the researchers also compared the NLR value with the degree of facial paralysis. They found a moderate positive correlation (r=0.661, p=0.001) between the severity of facial paralysis and NLR. As a result, the authors suggested that NLR value increases in Bell's palsy and can be evaluated as a predictive marker in prognosis (Özler & Günak, 2014). In a similar study published in 2017, NLR and PLR values in patients with Bell's palsy were compared with healthy individuals. The study also evaluated the baseline and one week later parameters of both groups. The baseline NLR values were analyzed as 1.7 ± 1.2 and 0.9 ± 0.2 (0=0.002) and PLR values as 129.4 ± 15.4 and 139.4 ± 19.4 (p=0.65) in the patient and control groups, respectively. When the same individuals were examined one patient later, the NLR values were 1.5 ± 0.9 and 11.1 ± 0.1 (p=0.03), and the PLR values were 135.2 ± 19 and 134.2 ± 12 (p=0.80) in the patient and control groups, respectively. In this study, the researchers found no significant correlation (P>0.05) between NLR and PLR values according to the HB scale (between HB score 2 and 3) (Sahin & Varım, 2017). In a study conducted with a larger patient group (n:656), an increase in NLR value was found in PFP (Kum et al., 2015). Although there are many

publications in the literature examining the relationship between PFP and NLR, there is limited data on SFP. In our study, the NLR values in SFP and PFP were 1.8 ± 0.9 and 1.4 ± 1 (p=0.001), respectively. According to these results, it was observed that NLR was higher in SFP. This may seem to be a different result compared to previous studies. However, in this study, we did not compare the healthy control group with the patients with PFP. Therefore, we may think that the inflammatory response on the facial nerve was found to be higher due to supranuclear injury. We may suggest that this result is due to the increase in neutrophils due to central nervous system involvement. We evaluated the relationship between the clinical grade of peripheral facial paralysis and NLR, PLR, MLR and SII. To assess clinical severity, the group was divided into two categories (HB Score ≤ 3 and >3). We did not detect a significant relationship between these four inflammatory markers and the clinical severity of PFP (P>0.05).

There is no clear consensus in the literature on the relationship between the degree of PFP and NLR, SII. However, the general opinion on this subject is that there is an increase in NLR in PFP. In 2015, two similar studies conducted on adult and pediatric patient groups did not find a significant difference or correlation between NLR and HB grade (Kum et al., 2015; Özler & Günak, 2014). There are few publications examining the value of PLR in facial paralysis. It has been reported that PLR is generally an important marker in more systemic diseases (Gunaldi et al., 2015). In this study, SII was 529.5 ± 297.4 in group 1 and 408.2 ± 228.1 in group 2 (p=0.029). We observe that central nervous system involvement leads to an increase in SII. We found that the most common etiology in patients with SFP was ischemic stroke (n:33, 62.2%). The increase in SII may possibly be related to this ischemic pathogenesis. In the early period of ischemia, neuronal swelling, followed by hyperchromasia and pyknosis occur. After these chain reactions that develop within minutes, chromatoliosis, swelling and disintegration of astrocytes and endothelial cells occur. Thus, a transient inflammatory reaction occurs in the first days of ischemia (Mărgăritescu et al., 2009). The increase in SII we obtained in our study can be explained by this pathogenesis. Publications examining the relationship between CNS pathologies and SII are increasing day by day. A study published in 2021 examined SII in acute ischemic stroke. This retrospective study included 277 patients with a mean age of 73.2±13.4 years. In the study, baseline SII (599.2 ± 297) and 1-year clinical improvement of the patients were followed. The researchers found no correlation between SII and prognosis after this study (Li et al., 2021). In this study, the reliability of the data is low due to the high mean age and increased comorbid conditions. In a case-control study (patient n:88, control n:50) in patients with Bell's palsy, the results of SII on the degree of paralysis, response to treatment and

prognosis were discussed. SII was higher in the patient group [median: 657.11 (min-max: 187.91-14,589.47)] than in the control group [median: 416.75 (177.95-3187.71)] (P=0.001). After this study, the authors stated that inflammation theory plays a role in the pathogenesis of Bell's palsy. In the same study, it was also suggested that SII could be used as a marker for prognosis and response to treatment [(AUC: 0.731- (sens %: 65.9 76.0, spec %: 76.0) p=0.001] (Kınar et al., 2021).

Limitations

In this study, two patient groups were compared. Due to the lack of a healthy control group, the correlation evaluation of inflammatory markers between the groups may be insufficient. Baseline parameters of the patients included in the study were evaluated. The lack of post-treatment parameters of the same patients stands out as another limitation. Finally, we do not know the effect of SII and NLR values on questions such as prognosis and response to treatment due to the lack of information about the examination findings and recovery time of the patients after a certain period.

CONCLUSION

In this study on the role of inflammatory reactions in facial paralysis, we analyzed that the results of SII, NLR were different between SFP and PFP. In SFP caused by CNS damage, the number of neutrophils increased, whereas in Bell's palsy, the number of lymphocytes increased. These results show that different inflammatory responses occur in PFP and SFP. Studies on this subject in the literature are not yet sufficient and more long-term studies are needed.

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