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Clinical characteristics and hematological inflammatory markers in pediatric peripheral facial paralysis: A retrospective study

Pediatrik periferik fasiyal paralizide klinik özellikler ve hematolojik inflamatuvar belirteçler: Retrospektif bir çalışma

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ABSTRACT

Aim: This study aimed to determine whether inflammatory indices derived from complete blood count indicate systemic inflammation at presentation in pediatric idiopathic Bell's palsy.

Material and Methods: We retrospectively analyzed 23 children with Bell's palsy and 69 age- and sex-matched healthy controls (March 2022–June 2025). Peripheral blood was drawn at presentation and before corticosteroid therapy. Indices included neutrophil/lymphocyte ratio, platelet/lymphocyte ratio, systemic immune-inflammation index, systemic inflammation response index, lymphocyte/monocyte ratio, eosinophil/lymphocyte ratio, derived neutrophil/lymphocyte ratio, and red cell distribution width/platelet ratio, mean platelet volume, platelet distribution width, plateletcrit were analyzed comparatively.

Results: Of the patients, 12 (52.2%) were female and 11 (47.8%) were male, with a mean age of 12.1 ± 3.8 years (range: 2–17). Based on the House–Brackmann classification, 17 (73.9%) patients were grade 2, 5 (21.7%) were grade 3, and 1 (4.4%) was grade 4. No statistically significant differences were found between the patient and control groups in terms of neutrophil/lymphocyte ratio, platelet/lymphocyte ratio, systemic immune-inflammation index, systemic inflammation response index, lymphocyte/monocyte ratio, eosinophil/lymphocyte ratio, derived neutrophil/lymphocyte ratio, plateletcrit, and red cell distribution width/platelet ratio values (for all variables $p > 0.05$). Mean platelet volume and platelet distribution width were lower in patients (Cliff's $\delta = -0.34$ for both; 95% CI –0.58 to –0.10, $p = 0.015$ for each). House–Brackmann grade correlated with plateletcrit (Spearman's $\rho = 0.58$; 95% CI 0.24 to 0.79; $p = 0.004$).

Conclusion: Modest reductions in mean platelet volume and platelet distribution width, along with a positive correlation between plateletcrit and clinical severity, may indicate platelet-axis involvement. Given the limited sample size and statistical power, these findings require confirmation in larger, standardized cohorts.

Keywords: Hematologic markers, inflammatory markers, pediatric bell's palsy

ÖZ

Amaç: Çocukluk çağı idiyopatik Bell paralizisinde başvuru anındaki tam kan sayımı kaynaklı inflamatuvar indekslerin sistemik inflamasyonu gösterip göstermediğini belirlemek amaçlandı.

Gereç ve Yöntemler: Mart 2022–Haziran 2025 tarihleri arasında üçüncü basamak pediatrik nöroloji kliniğinde izlenen Bell paralizili 23 çocuk ile yaş ve cinsiyete göre eşleştirilmiş 69 sağlıklı kontrol geriye dönük olarak analiz edildi. Periferik kan örnekleri başvuruda ve kortikosteroid tedavisi öncesinde alındı. İncelenen indeksler: nötrofil/lenfosit oranı, trombosit/lenfosit oranı, sistemik immün-inflamasyon indeksi, sistemik inflamasyon yanıt indeksi, lenfosit/monosit oranı, eozinofil/lenfosit oranı, türetilmiş nötrofil/lenfosit oranı, eritrosit dağılım genişliği/trombosit oranı, ortalama trombosit hacmi, trombosit dağılım genişliği ve plateletkrit; karşılaştırmalı analiz yapıldı. İstatistiksel anlamlılık için $p<0,05$ kabul edildi.

Bulgular: Hastaların 12'si (%52,2) kız, 11'i (%47,8) erkek; ortalama yaş 12,1±3,8 yıl (2–17). House–Brackmann sınıflamasına göre 17'si (%73,9) derece 2, 5'i (%21,7) derece 3, 1'i (%4,4) derece 4'tü. Nötrofil/lenfosit oranı, trombosit/lenfosit oranı, sistemik immün-inflamasyon indeksi, sistemik inflamasyon yanıt indeksi, lenfosit/monosit oranı, eozinofil/lenfosit oranı, türetilmiş nötrofil/lenfosit oranı, plateletkrit ve eritrosit dağılım genişliği/trombosit oranı açısından hasta ve kontrol grupları arasında anlamlı fark saptanmadı (tüm değişkenler için $p > 0,05$). Ortalama trombosit hacmi ve trombosit dağılım genişliği hasta grubunda daha düşüktü (Cliff's $\delta = -0.34$; %95 güven aralığı –0.58 ile –0.10; her iki değişken için $p = 0,015$). House–Brackmann derecesi ile plateletkrit arasında pozitif korelasyon saptandı (Spearman's $\rho = 0,58$; 95% güven aralığı 0.24–0.79; $p = 0,004$).

Sonuç: Ortalama trombosit hacmi ve trombosit dağılım genişliğindeki ılımlı düşüşler ile plateletkrit ve House–Brackmann derecesi arasındaki pozitif korelasyon, trombosit ekseninin olası etkilenimini düşündürmektedir. Ancak sınırlı örneklem nedeniyle sonuçlar, daha geniş ve metodolojik olarak standart kohortlarda teyit edilmelidir.

Anahtar Kelimeler: Hematolojik belirteçler, inflamatuvar belirteçler, pediatrik bell paralizisi

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Highlights

- This retrospective study evaluated a broad panel of complete blood count–derived inflammatory indices in children with idiopathic peripheral facial paralysis compared with healthy controls.
- Modest reductions in MPV (Mean Platelet Volume) and PDW (Platelet Distribution Width), together with a positive correlation between PCT (Plateletcrit) and disease severity, suggest platelet-axis involvement in pediatric BP (Bell's palsy).
- As routinely available parameters, these indices may help support early clinical assessment in pediatric BP across diverse clinical settings.

INTRODUCTION

Peripheral facial paralysis is one of the most common motor cranial neuropathies. A lesion occurring at any point along the course of the facial nerve—the seventh cranial nerve—extending from its motor nucleus in the brainstem to the muscles of facial expression, may result in peripheral facial paralysis (1). Idiopathic peripheral facial paralysis, also known as Bell's palsy (BP), is characterized by sudden-onset unilateral facial muscle weakness in the absence of identifiable causes such as other cranial neuropathies, neoplasms, or infections. Clinical manifestations typically include the inability to close the eyelid on the affected side, drooping of the corner of the mouth, alterations in taste sensation, and hypersensitivity to sound (2).

Although not fully elucidated, BP is most attributed to edema that confines the facial nerve in the fixed bony canal, resulting in compression. Theories involving infection/inflammation and vascular ischemia support this mechanism (3). While the condition is most often idiopathic, viral infections—particularly herpes simplex virus—along with immune and inflammatory mechanisms have been implicated in its pathogenesis (4). The annual incidence in children has been reported as 2–4 per 100,000 (5). A favorable prognosis is expected, with approximately 70–90% of patients with BP recovering completely without treatment (6).

Hematological inflammatory parameters are biomarkers derived from complete blood counts (CBC) and serve as indirect indicators of systemic inflammation. In particular, the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and lymphocyte-to- monocyte ratio (LMR) is widely used to assess inflammatory responses in various chronic diseases, infections, and malignancies (7). More recently, newly derived indices such as the Systemic Immune-Inflammation Index (SII) and the Systemic Inflammation Response Index (SIRI) have gained importance as prognostic markers (8). These parameters are commonly preferred in clinical practice due to their accessibility, low cost, and reliability (9).

The favorable response of BP to corticosteroids, along with contrast enhancement on MRI (magnetic resonance imaging), supports an inflammatory component in its pathogenesis. As a reliable inflammatory marker, NLR has been reported to increase during inflammatory processes in adult patients with BP. Mean platelet volume (MPV) and PLR are additional parameters associated with vascular diseases and inflammatory conditions. However, evidence in children is sparse, with only a few studies examining these hematologic markers in BP (3, 5, 10-12).

This study aimed to describe the clinical characteristics of pediatric BP and to examine potential associations between pathophysiology and selected CBC-derived inflammatory indices. By comparing these markers between patients and healthy controls, the study sought to assess whether such indices—derived from routine blood tests—might offer supportive evidence of systemic inflammation during the acute phase of the condition.

MATERIAL and METHODS

This retrospective cross-sectional study included 23 pediatric patients aged ≤ 18 years with a first episode of idiopathic BP, diagnosed at a tertiary pediatric neurology clinic between March 2022 and June 2025. Eligible patients were required to have complete clinical records and CBC parameters obtained at the time of presentation. Exclusion criteria included recurrent facial palsy, secondary causes of facial paralysis, acute infection, chronic systemic disease, use of immunomodulatory or anti-inflammatory medication prior to blood sampling, and incomplete clinical or laboratory data. Of all children presenting with facial paralysis, three were diagnosed with congenital facial palsy, two had facial nerve–involving tumors, and one had traumatic facial nerve injury; these cases were excluded from the analysis (Figure 1). The control group consisted of 69 healthy children, selected consecutively from routine outpatient visits at a 3:1 control-to-case ratio to improve statistical power due to the relative rarity of pediatric BP. Controls were matched to cases at the group level for age and sex ($p > 0.05$ for both) and were required to have no history of neurologic, infectious, inflammatory, or chronic disease, no medication use or infection within the preceding two weeks, and available CBC results. Demographic and clinical data, including age, sex, side of facial involvement, comorbidities, time to presentation, and

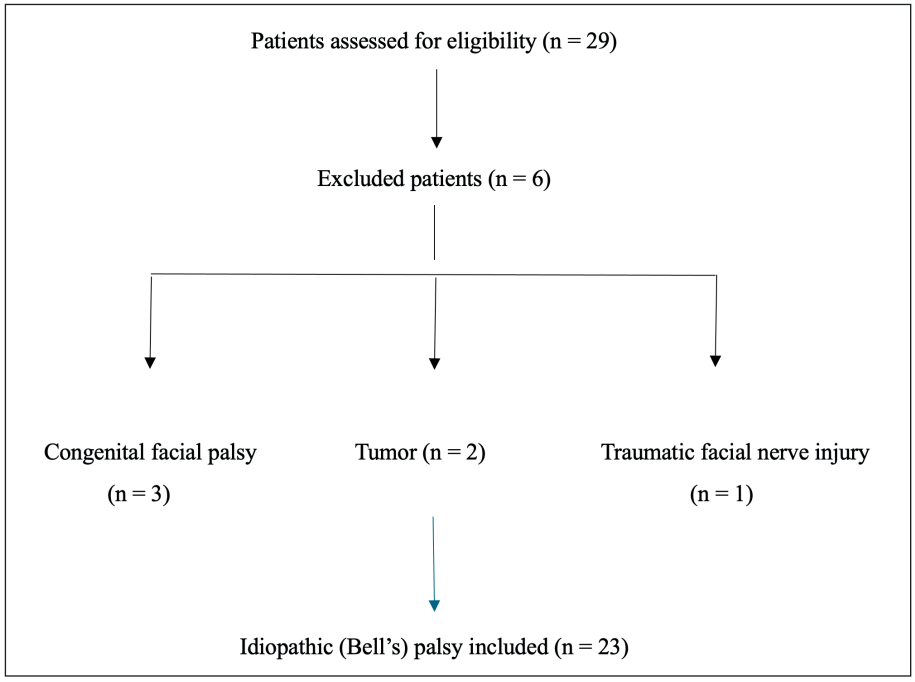

Figure 1: Flow diagram of patient selection

treatments administered, were retrieved retrospectively from medical records. Patients were classified according to the House–Brackmann (HB) grading system. The HB grading system classifies facial function into six grades, ranging from normal function (Grade I) to complete paralysis (Grade VI), based on standardized evaluation of resting symmetry, voluntary movement, and synkinesis. Grade I reflects normal facial function; Grade II indicates mild dysfunction with slight asymmetry; Grade III represents moderate dysfunction with obvious but non-disfiguring weakness; Grade IV corresponds to moderately severe dysfunction with incomplete eye closure; Grade V reflects severe dysfunction with barely perceptible movement; and Grade VI denotes total facial paralysis (13).

All blood samples were obtained at the initial presentation for facial palsy and before initiation of corticosteroid therapy. All CBC analyses were performed using the same automated hematology analyzer, Sysmex XN-1000, in the hospital's central laboratory to ensure consistency. From the CBC results, the following laboratory parameters were obtained: total leukocyte (×10³/μL), neutrophil (×10³/μL), lymphocyte (×10³/μL), monocyte (×10³/μL), eosinophil (×10³/μL), platelet (×10³/μL), MPV (fL), platelet distribution width (PDW) (%), red cell distribution width (RDW) (%), and plateletcrit (PCT) (%). Several derived inflammatory indices were calculated as follows: SII was calculated by multiplying the neutrophil and platelet counts and dividing the result by the lymphocyte count (neutrophil × platelet) / lymphocyte; SIRI was obtained by multiplying the neutrophil and monocyte counts and dividing by the lymphocyte count (neutrophil × monocyte)/ lymphocyte; LMR was calculated as lymphocyte count divided by monocyte count; eosinophil-to-lymphocyte ratio (ELR) as eosinophil count divided by lymphocyte count; derived NLR (d-NLR) as neutrophil count divided by the difference between total white blood cell count and neutrophil count; and RDW-to-platelet ratio (RPR) as RDW (%) divided by platelet count (10³/μL).

Data were evaluated using IBM SPSS Statistics, Version 25. Normality checks used the Kolmogorov–Smirnov and Shapiro–Wilk procedures. Skewed variables appear as median (min–max); normally distributed variables as mean ± SD. To interpret non-significant findings more accurately, we conducted a sensitivity analysis to estimate the minimal detectable effect for our sample size (n=23 vs. 69; α=0.05; 80% power). For two-group analyses, independent-samples t-tests were applied when assumptions held; otherwise, Mann–Whitney U tests were used. Categorical outcomes were analyzed with Pearson's chi-square or Fisher's exact tests. Significance threshold: $p<0.05$. Between-group contrasts were summarized with standardized effect sizes and 95% confidence intervals, reported as Cliff's delta (with a probability-of-superiority interpretation). All effect sizes are reported as Cliff's delta. For outcomes analyzed with independent-samples *t* tests, δ was derived from Hedges' *g* using $\delta = 2 \cdot \Phi(g/\sqrt{2}) - 1$, with confidence intervals transformed accordingly. Associations between HB grade (ordinal) and hematologic indices were summarized with Spearman's ρ and 95% CIs (Fisher's z method).

The study protocol was reviewed and approved by the Malatya Turgut Özal University Health Sciences Scientific Research Ethics Committee (Approval No: 2025/250, Date: August 5, 2025), and all procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

A total of 23 children were included in the final analysis. Among these, 12 were female (52.2%) and 11 were male (47.8%). Facial involvement was observed on the left side in 12 patients (52.2%) and on the right side in 11 patients (47.8%). The age range of the patients was between 2 and 17 years, with a mean age of 12.1 ± 3.8 years. Two patients (8.7%) had comorbid conditions, epilepsy (n=1) and scaphocephaly (n=1). The control group consisted of 69 healthy children (33 males, 36 females) with similar age and sex characteristics (Table 1).

When diagnoses were evaluated by year, five patients were identified in 2022, three in 2023, ten in 2024, and five in 2025. The highest number of cases by month was observed in February (4 patients) and September (3 patients). There was no statistically significant difference in the monthly (p=0.947) or seasonal (p=0.924) distribution of cases.

According to the HB classification, 17 patients (73.9%) were classified as grade 2, 5 patients (21.7%) as grade 3, and 1 patient (4.4%) as grade 4. The mean duration from symptom onset to clinical presentation was 3.6 days, ranging from 1 to 10 days. Steroid therapy was administered to 16 patients (69.5%). Central nervous system imaging was performed in 13 patients (56.5%); results were normal in 11, and incidental arachnoid cysts were detected in 2.

No statistically significant differences were found between the patient and control groups in terms of NLR, PLR, SII, SIRI, LMR, eosinophil count, ELR, RDW, derived NLR, PCT, and RDW/PLT values ($p > 0.05$). MPV and PDW were lower in patients (Cliff's $\delta = -0.34$ for both; 95% CI −0.58 to −0.10; $p = 0.015$) (Table 2). Values of hematologic indices across HB grades and their correlations with HB grade are shown in detail (Table 3). Among all markers, PCT showed a moderate, statistically significant correlation with clinical severity (Spearman's $\rho = 0.58$; 95% CI 0.24–0.79; p =

Table 1: Demographic characteristics of the patient and control groups

	Patient Group (n=23)	Control Group (n=69)	p-value
Sex (Female/Male)	12/11	36/33	1.000[a]
Mean age ±SD (years)	12.1 ± 3.8	12.1 ± 4.01	0.935[b]
Median age (min-max) (years)	12.5 (2.0-17.0)	12.5 (1.8-17.5)	

[a]Pearson Chi-square, [b]Mann-Whitney U
(n: number, max: maximum, min: minimum, SD: standard deviation)

Table 2: Comparison of Hematological Inflammatory Markers Between the Patient and Control Groups

	Patient Group	Control Group	p-value	Effect size (Cliff's δ; 95% Confidence Interval)
NLR	1.39 (0.37-5.91)	1.38 (0.26-6.03)	0.531[a]	δ = 0.09 (95% CI −0.19 to 0.36)
PLR	0.113 ± 0.042	0.110 (0.048-0.410)	0.763[b]	δ = −0.04 (95% CI −0.31 to 0.23)
ELR	0.034 (0.00-0.20)	0.053 (0.01-0.36)	0.104[a]	δ = −0.23 (95% CI −0.48 to 0.03)
LMR	5.59 ± 2.79	4.76 (1.14-12.01)	0.675[b]	δ = 0.06 (95% CI −0.22 to 0.34)
SII	465.9 (61.8-2224)	405 (84.1-2044.1)	0.384[a]	δ = 0.12 (95% CI −0.16 to 0.40)
SIRI	850 (214.8-5275.5)	780 (162.4-7568.8)	0.567[a]	δ = 0.08 (95% CI −0.20 to 0.36)
MPV	9.53 ± 1.00	10 (8.2-12.4)	0.015[a]*	δ = −0.34 (95% CI −0.58 to −0.10)
PCT	0.291 ± 0.085	0.315 ± 0.062	0.146[b]	δ = −0.20 (95% CI −0.44 to 0.07)
PDW	9.8 (8.3-14.4)	11.4 (8.0-16.9)	0.015[a]*	δ = −0.34 (95% CI −0.58 to −0.10)
d-NLR	1.034 (0.33-4.06)	1.101 (0.23-3.42)	0.404[a]	δ = 0.12 (95% CI −0.16 to 0.39)
RDW/PLT	0.043 (0.02-0.10)	0.041 (0.03-0.09)	0.925[a]	δ = −0.01 (95% CI −0.29 to 0.26)

[a]Mann Whitney U, [b]Independent Samples t-test

d-NLR: derived Neutrophil-to-Lymphocyte Ratio, **ELR:** Eosinophil-to-Lymphocyte Ratio, **LMR:** Lymphocyte-to-Monocyte Ratio, **MPV:** Mean Platelet Volume, **NLR:** Neutrophil-to-Lymphocyte Ratio, **PCT:** Plateletcrit, **PDW:** Platelet Distribution Width, **PLR:** Platelet-to-Lymphocyte Ratio, **PLT:** Platelet, **RDW:** Red Cell Distribution Width, **SII:** Systemic Immune-Inflammation Index, **SIRI:** Systemic Inflammatory Response Index (Data are presented as mean ± standard deviation or median (min–max) according to the distribution)

(Study sensitivity: with n=23 vs 69, minimal detectable effect at 80% power ≈ d=0.67 (Hedges' g) ≈ δ=0.37 (Cliff's delta), i.e., AUC≈0.685.)

Table 3: Correlations between House–Brackmann Grade and Hematologic Indices

	HB Grade 2 (n=17)	HB Grade 3 (n=5)	HB Grade 4 (n=1)	p-value	Effect size vs HB grade (Spearman ρ; 95% CI)
NLR	1.52 (0.37-3.95)	1.31 (0.81-5.91)	5.17	0.665[a]	ρ = 0.10 (95% CI –0.42 to 0.56)
PLR	0.105 ± 0.035	0.124 ± 0.051	0.200	0.247[a]	ρ = 0.25 (95% CI –0.20 to 0.62)
ELR	0.034 (0.010-0.201)	0.070 ± 0.074	0.005	0.477[a]	ρ = –0.16 (95% CI –0.53 to 0.43)
LMR	5.80 ± 2.89	5.56 ± 2.48	2.11	0.574[a]	ρ = –0.12 (95% CI –0.53 to 0.31)
SII	488 ± 264	400 (241-2124)	2224.0	0.364[a]	ρ = 0.20 (95% CI –0.34 to 0.64)
SIRI	825 (214-3368)	1043 (252-4557)	5275.5	0.364[a]	ρ = 0.20 (95% CI –0.34 to 0.64)
MPV	9.30 ± 0.92	10.14 ± 1.15	10.4	0.089[a]	ρ = 0.36 (95% CI –0.14 to 0.69)
PCT	0.26 ± 0.05	0.36 ± 0.09	0.45	0.004[a]*	ρ = 0.58 (95% CI 0.24 to 0.79)
PDW	9.7 (8.3-14.4)	10.98 ± 2.29	12.0	0.341[a]	ρ = 0.21 (95% CI –0.54 to 0.63)
d-NLR	1.29 ± 0.68	0.97 (0.68-4.06)	3.46	0.712[a]	ρ = 0.08 (95% CI –0.45 to 0.57)
RDW/PLT	0.044 (0.03-0.10)	0.037 ± 0.010	0.03	0.109[a]	ρ = –0.34 (95% CI –0.64 to 0.03)

[a]Spearman correlation

d-NLR: derived Neutrophil-to-Lymphocyte Ratio, **ELR:** Eosinophil-to-Lymphocyte Ratio, **LMR:** Lymphocyte-to-Monocyte Ratio, **MPV:** Mean Platelet Volume, **NLR:** Neutrophil-to-Lymphocyte Ratio, **PCT:** Plateletcrit, **PDW:** Platelet Distribution Width, **PLR:** Platelet-to-Lymphocyte Ratio, **PLT:** Platelet, **RDW:** Red Cell Distribution Width, **SII:** Systemic Immune-Inflammation Index, **SIRI:** Systemic Inflammatory Response Index (Data are presented as mean ± standard deviation or median (min–max) according to the distribution)

0.004), whereas correlations for other indices were weak and non-significant.

In the 1:1 age- and sex-matched subset (n = 23 vs. 23), both MPV and PDW values remained lower in patients compared to controls, although the differences were no longer statistically significant (p > 0.05). The direction of change was consistent with the main 3:1 analysis, suggesting that the primary findings were not driven by unequal group sizes but rather by the limited power of the smaller matched sample. The positive correlation between PCT and HB grade (ρ = 0.58, p = 0.004) persisted, supporting its robustness across analytic approaches.

DISCUSSION

In this study, we evaluated the hematological inflammatory markers in pediatric patients diagnosed with BP at presentation and compared the findings with those of a healthy control group. The primary objective was to investigate whether routinely available blood-based biomarkers could reflect an underlying systemic inflammatory process in these patients. The important role of inflammation in the pathogenesis of BP has been well documented in the literature. Öztürk et al. reported that SII and SIRI demonstrated high predictive value in the diagnosis of pediatric BP cases (AUC = 0.77 and 0.68, respectively) (10). Similarly, Liu et al. reported that NLR, PLR, and SIRI values were significantly increased in children with BP compared to the control group (4). Kim et al. suggested that NLR and PLR could serve as prognostic indicators in pediatric BP (14). In a recent pediatric cohort, NLR, PLR, and monocyte-to-lymphocyte ratio did not significantly differ between patients and controls but showed a positive correlation with recovery time, suggesting that leukocyte-derived ratios may serve as prognostic indicators in children with BP (11). Despite several prior reports in pediatric and adult BP showing significant shifts in inflammatory markers (15-18), we observed no statistically significant patient–control differences for most indices, including NLR, PLR, SII, SIRI, LMR, eosinophil count, ELR, RDW, derived NLR, PCT, and RDW/PLT. Several factors may account for this discrepancy. First, our study population consisted predominantly of patients with mild clinical presentations, as evidenced by the high proportion of HB grade 2 cases. It is possible that systemic inflammatory responses are more pronounced in moderate to severe cases, and thus, our cohort may not have exhibited sufficient variation in inflammatory marker levels to yield statistically significant results. Çelik et al. reported that commonly used hematologic marker did not demonstrate meaningful prognostic value in pediatric BP. In their cohort, early clinical severity was the only parameter associated with recovery, while CBC-derived inflammatory indices failed to predict short-term outcomes (19). Our findings are also in line with a recent single-center pediatric cohort by Di Sarno et al., who systematically evaluated prognostic factors, laboratory markers, and treatment strategies in 88 children with BP. In that study, no significant associations were observed between NLR, PLR, and HB scores at 2-month follow-up, despite an overall complete recovery rate of 81.8%. Taken together with our negative results for a broad panel of CBC-derived indices, these data suggest that readily available systemic inflammatory markers may have limited and inconsistent prognostic utility in

pediatric BP. Moreover, Di Sarno et al. reported that corticosteroid use—and particularly higher prednisone doses—was associated with a greater proportion of incomplete recovery, while vitamin supplementation had no measurable impact on outcomes. This pattern supports the notion that pediatric BP may be characterized by relatively subtle or heterogeneous systemic inflammatory changes that are not reliably captured by routine blood-based biomarkers and may not be readily modifiable by standard anti-inflammatory or adjunctive therapies (12). However, given the relatively small samples and predominance of mild cases in both cohorts, modest prognostic effects of these markers cannot be definitively excluded.

The patient group showed modestly lower MPV and PDW values compared with controls (Cliff's δ = –0.34 for both; 95% CI –0.58 to –0.10), suggesting that in approximately two-thirds of randomly paired observations, the corresponding control value was higher. Although statistically significant, the standardized effects are small-to-moderate, suggesting that any platelet-related change is subtle at the systemic level. Platelet size heterogeneity and activation are reflected by MPV and PDW. In classical systemic inflammation, they often increase, reflecting the release of larger, metabolically active platelets from the marrow. Conversely, several pediatric acute inflammatory states have reported lower MPV in the early phase, plausibly due to preferential consumption/margination of larger platelets at inflamed microvascular beds or a transient shift toward producing smaller platelets under cytokine pressure. In some settings, MPV has even been described as a negative acute-phase marker. Our samples were obtained at presentation and before corticosteroid therapy, consistent with an early-phase profile. Together with the positive correlation between PCT and clinical severity (ρ = 0.58), these findings may suggest a platelet-axis involvement in pediatric BP—i.e., altered platelet mass/turnover with relatively smaller and more uniform circulating platelets rather than a broad leukocyte-driven response. The relatively decreased MPV and PDW in affected patients could reflect a blunted or insufficient bone marrow response, or a distinct inflammatory pattern differing from classical systemic inflammation (20, 21). Sen et al. reported significantly elevated WBC, neutrophil, and platelet counts in pediatric patients with BP; however, the findings regarding MPV and PDW were inconsistent (5). In one study, a selective reduction in MPV was reported in pediatric BP, occurring exclusively in affected children (6/17 vs 0/17 controls, p = 0.007) (11). Further research is required to better understand how MPV and PDW are associated with pediatric BP.

Apart from PCT, the correlations of HB grade with CBC-based markers (NLR, PLR, SII, SIRI, LMR, ELR, d-NLR, RDW/PLT, MPV, PDW) were weak and did not reach statistical significance. A moderate positive and statistically significant correlation was observed between HB grade and PCT (Spearman's ρ=0.58, p = 0.004), indicating that greater clinical severity may be associated with increased total platelet mass. As it incorporates both platelet count and volume, PCT may provide a composite measure of platelet activation and systemic inflammatory burden. A previous study reported that PCT levels were significantly higher in patients with BP compared to healthy controls (p = 0.008); however, no significant association was observed between PCT values and recovery time (22). Its potential role as a marker of disease severity in pediatric BP deserves further investigation in larger, prospective studies.

The etiology of BP remains largely idiopathic; however, immune-mediated, viral, and ischemic mechanisms are widely believed to contribute to its pathogenesis. Neurotropic viral exposure is thought to activate inflammatory pathways in the facial nerve, leading to focal edema, myelin loss, and ischemia. In addition to infectious triggers, various systemic and environmental factors—such as hyperglycemia, uncontrolled hypertension, severe pre-eclampsia, migraine, and prior exposure to radiation—have been reported to predispose individuals to facial nerve palsy by promoting or exacerbating underlying inflammatory and ischemic processes (23, 24). This multifactorial etiology may explain the heterogeneity in clinical presentations and the inconsistent findings regarding laboratory-based inflammatory markers observed across different studies.

This study has several noteworthy strengths. The use of an age- and sex-matched healthy control group enhances the reliability of the comparisons and reduces potential confounding. This study provides valuable insight to the existing literature by focusing exclusively on a pediatric population with idiopathic BP, whereas most prior studies have been conducted in adult or mixed-age cohorts using heterogeneous definitions and analytic approaches. By employing standardized formulas for both traditional and newly derived hematologic indices, reporting effect sizes alongside significance levels, and examining correlations with clinical severity, this study provides a more nuanced understanding of the inflammatory profile in pediatric BP. To our knowledge, while pediatric cohorts assessing SII and SIRI in BP have recently been reported, most prior pediatric studies have focused on traditional indices such as NLR, PLR, MPV, PDW and LMR. This study is the first pediatric analysis to evaluate ELR and RPR in relation to BP severity. Furthermore, the simultaneous evaluation of multiple indices within a single, well-defined cohort enhances interpretability and supports future meta-analytic integration. Together, these features strengthen the methodological quality and contextual contribution of the study to the pediatric neuroinflammation literature.

These findings should be interpreted in light of several limitations. The study is single-center and retrospective with limited power (n=23 vs 69; ~80% power for effects ≥ d≈0.67). Disease severity was mostly mild. Despite age/sex matching, hospital-based controls may differ on unmeasured factors. Although sampling occurred at presentation before steroids, onset-to-sampling times were not uniformly documented. With only a single patient in the HB grade 4 subgroup, precision for severity-stratified analyses was limited.

Conclusion

Modest reductions in MPV and PDW, together with a positive correlation between PCT and disease severity, suggest platelet-axis involvement in pediatric BP. Although their clinical utility remains limited, these readily available indices may provide supportive information when assessing initial disease severity or anticipating short-term recovery patterns. Incorporating such markers into routine evaluation may help refine early prognostic monitoring. From a clinical perspective, these results may cautiously support using MPV, PDW, and PCT as easily accessible, supplementary information when evaluating children with BP, while primary assessment and management should continue to rely on established clinical criteria. Larger, prospective studies are warranted to validate their role and clarify the underlying pathophysiological mechanisms.

Author Contributions

Study conception and design: **Meral Karadağ, Melek Yaman Ortaköylü;** data collection: **Meral Karadağ, Melek Yaman Ortaköylü;** analysis and interpretation of results: **Meral Karadağ, Melek Yaman Ortaköylü;** draft manuscript preparation: **Meral Karadağ, Melek Yaman Ortaköylü.** The authors reviewed the results and approved the final version of the article.

Conflicts of Interest

The authors declare that there is no conflict of interest to disclose.

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Ethical Approval

This study was approved by the Malatya Turgut Özal University Health Sciences Scientific Research Ethics Committee (date: 05.08.2025, number: 2025-250)

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