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Original Article

Hematological Findings in Patients with Acute Peripheral Facial Palsy

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OBJECTIVES: This study aimed at evaluating the clinical significance of hematological findings in patients with acute peripheral facial palsy.

MATERIALS and METHODS: For this retrospective case series review, 84 patients who visited our university hospital and were diagnosed with Bell's palsy (BP) or Ramsay Hunt syndrome (RHS) between March 2017 and March 2019 were enrolled. We documented their epidemiological details, final diagnoses, House-Brackmann (HB) palsy grades, and pretreatment and day 7 post-hospitalization complete blood counts. The outcome was considered favorable if the HB grade at weeks 10–16 was I or II. We analyzed the hematological findings in terms of diagnosis and the final treatment outcomes.

RESULTS: A higher pretreatment neutrophil-to-lymphocyte ratio (NLR) and neutrophil count and a lower day-7 lymphocyte count were observed in patients with RHS with unfavorable outcomes. In such patients, moderate positive correlations were observed between the pretreatment white blood cell, neutrophil, and basophil counts; the NLR and basophil-to-lymphocyte ratio; and the initial HB grade. Only the latter was a significant risk factor for a poor treatment outcome. In patients with BP, both the initial HB grade and the pretreatment eosinophil count were included in a regression model predicting prognosis.

CONCLUSION: Inflammation plays an important role in RHS pathogenesis. Initial RHS severity and the response to corticosteroids may determine the final treatment outcome. However, inflammatory markers do not predict all BP outcomes; BP may be etiologically heterogeneous.

KEYWORDS: Facial paralysis, prognosis, inflammation

INTRODUCTION

Reactivation of herpes simplex virus type 1 (HSV-1), human herpes virus 6 (HHV-6), or varicella-zoster virus (VZV) causes most episodes of acute peripheral facial palsy (APFP) [1]. Early confirmation of zoster sine herpete (VZV DNA in saliva and/or pain) may aid in planning the Bell's palsy (BP) treatment [1, 2]. Combined steroids and antivirals are the mainstay treatments for severe BP [3]. Adjuvant intratympanic steroid injections are administered, but their effects remain unclear [4]. Apart from the viruses mentioned above, inflammation, ischemia, and acute cold exposure may play roles in APFP pathogenesis [5]. Inflammatory markers, including the eosinophil-to-lymphocyte ratio (ELR), neutrophil-to-lymphocyte ratio (NLR), and basophil-to-lymphocyte ratio (BLR) are useful for the evaluation of various diseases [6]. NLR has been reported to be elevated in patients with BP, correlating positively with the House-Brackmann (HB) grade; higher NLR predicted poorer BP prognosis [7]. Another study found that patients with BP had higher NLRs and neutrophil counts than controls, which was associated with unfavorable outcomes [8]. Most APFP studies derived the NLR using the pretreatment complete blood count (CBC). A follow-up CBC (during treatment) is desirable; comparison of the 2 CBCs would reveal the changes in various inflammatory markers over time, whether the markers accurately predicted prognosis, and whether additional hematological data are required. Here we evaluated the clinical significance of hematological findings in patients with APFP.

MATERIALS AND METHODS

Patients

We examined the records of all patients (18 years or older) who visited the Department of Otolaryngology or the emergency room of our university hospital from March 2017 to March 2019 for the treatment of APFP and who were admitted to the hospital for 1 week. The

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Table 1. Eosinophil-to-lymphocyte, neutrophil-to-lymphocyte, and basophil-to-lymphocyte ratios at admission and discharge (day 7)

	Total			Bell's palsy			Ramsay-Hunt syndrome		
	Pretreatment	Day 7	p	Pretreatment	Day 7	p	Pretreatment	Day 7	p
ELR	0.107±0.075	0.029±0.021	<0.001	0.105±0.074	0.028±0.020	<0.001	0.113±0.079	0.032±0.022	<0.001
NLR	2.986±3.231	2.378±1.389	0.097	2.579±3.094	2.505±1.507	0.864	3.896±3.405	2.095±1.053	0.008
BLR	0.019±0.020	0.012±0.011	<0.001	0.016±0.010	0.011±0.008	<0.001	0.026±0.032	0.013±0.015	0.002

ELR: eosinophil-to-lymphocyte ratio; NLR: neutrophil-to-lymphocyte ratio; BLR: basophil-to-lymphocyte ratio.

exclusion criteria were treatment for more than 7 days, bilateral facial palsy, and/or missing medical data. We recorded patients' age, sex, affected side, recurrence status, extent of palsy, any comorbidity such as diabetes and/or hypertension, and accompanying dizziness [9]. Ramsay Hunt syndrome (RHS) was diagnosed if vesicles in the ear or oropharynx were evident either before or during treatment, or if an anti-VZV IgM antibody test was positive. All other patients with APFP were diagnosed with BP. All patients underwent electroneurography (ENoG); the ENoG value was the mean compound action potential of the frontalis, orbicularis oculi, nasalis, orbicularis oris, and mentalis muscles. Magnetic resonance imaging (MRI) was used to rule out the central lesions. The HB scale was used to grade palsy. The institutional review board of our hospital approved the study and waived the need for written informed patient consent because of the retrospective nature of the work.

ELR, NLR, and BLR Calculations

In total, 2 differential CBCs were performed, 1 on the day of admission (before treatment) and 1 on hospital day 7 (the discharge date). The eosinophil, lymphocyte, monocyte, and neutrophil levels/mL were determined, and the ELR, NLR, and BLR were calculated.

Treatments

All patients received oral methylprednisolone (MPD) (48 mg/day for 4 days followed by a tapering dose of 8 mg every 2 days). In patients weighing less than 60 kg, 0.8 mg/kg of MPD was prescribed for 4 days and then tapered over time. In addition, 3,000 mg/day of oral valacyclovir was prescribed for 7 days.

Facial Paralysis Severity

The extent of facial palsy was assessed using HB grading at the initial visit and at weeks 2, 6, and 10–16 after treatment. Final recovery was defined as an HB grade of I or II at weeks 10–16 [1, 9].

Statistical Analysis

We used the Shapiro–Wilk test of normality and the two-tailed Fisher's exact and/or chi-squared test to compare the nominal variables. We employed the student's t-test and the Mann–Whitney U test to compare the continuous variables. We used the paired t-test to compare the paired variables. We calculated Pearson's correlations between the inflammatory marker levels and the HB grade. We performed a forward conditional logistic regression analysis of the covariates that differed significantly by the treatment outcome. All analyses were performed using the IBM Statistical Package for the Social Sciences Statistics for Macintosh ver. 26.0 (IBM Corp., Armonk, NY, USA); $p<0.05$ were considered statistically significant.

MAIN POINTS

- Timing and extent of inflammation may vary by diagnosis in patients with acute peripheral facial palsy.
- In patients with Ramsay-Hunt syndrome, inflammation seems to play an important role from the time of palsy commencement.
- Inflammatory markers do not predict the prognosis of Bell's palsy, suggesting that Bell's palsy may be etiologically heterogeneous.

RESULTS

Patient Characteristics

Of the 84 patients enrolled, 44 were men and 40 were women. The mean (±standard deviation) age of the patients was 47±14.35 (range, 18–76) years; 44 (52.4%) had right-sided and 40 (47.6%) left-sided facial palsy. The mean time from palsy onset to treatment was 1.71±1.56 (range, 0–7) days. Furthermore, 2 patients (2.4%) had previous histories of BP. In terms of comorbidities, 7 (8.3%) patients had diabetes, and 18 (21.4%) patients had hypertension. The mean hearing threshold on the affected side was 20.64±20.55 dB, which was significantly poorer than that of the non-affected side (13.70±10.63 dB) ($p<0.001$). Overall, 58 patients (69.0%) were diagnosed with idiopathic BP and the remaining 26 with RHS (31.0%), and 17 (20.2%) patients had accompanying dizziness. The initial HB grade was 3.00±0.776 (range, 2–5). The mean ENoG value, indicating the extent of facial nerve degeneration, was 60.67%±19.64% (range, 16.80%–100%); 5 patients (6.0%) were anti-HSV IgM antibody positive, and 77 patients (91.7%) were anti-HSV IgG antibody positive. In terms of VZV status, 6 patients (7.1%) were anti-VZV IgM antibody positive, and 80 patients (95.2%) were anti-VZV IgG antibody positive.

Hematological Findings

The white blood cell (WBC), neutrophil, lymphocyte, and monocyte counts increased over time ($p<0.001$) in all patients. The eosinophil count decreased significantly ($p<0.001$), but the basophil count did not change ($p>0.05$). Similar trends were observed irrespective of the final diagnosis. None of the inflammatory markers, including the ELR, NLR, and BLR, varied with the diagnosis ($p>0.05$). The ELR and BLR decreased over time in all patients (Table 1) ($p<0.001$). However, the NLR decrease was not significant in patients with BP or other patients ($p>0.05$). In patients with RHS, all markers, including the ELR, BLR, and NLR, decreased significantly over time ($p<0.01$). However, some hematological findings differed by the final treatment outcome and disease entity. In all patients, lower lymphocyte counts on both the pretreatment day ($p=0.017$) and day 7 ($p=0.009$) were associated with unfavorable outcomes (Figure 1A). In patients with

BP, lower WBC (p=0.015) (Figure 1B) and eosinophil (p=0.026) (Figure 1C) counts on day 7 were associated with unfavorable outcomes. In patients with RHS, a higher pretreatment neutrophil count (p=0.041) (Figure 1D) and a lower day-7 lymphocyte count (p=0.005) (Figure 1E) were associated with unfavorable outcomes.

Although the outcomes of neither the patients with BP nor the other patients differed by their ELR, NLR, or BLR, the data of patients with RHS were of interest. First, the initial NLR in patients with RHS with unfavorable outcomes was 6.79±3.27, which was significantly higher than that of those who recovered (3.21±3.13) (p=0.031) (Figure 1F). Second, the pretreatment WBC (r=0.564, p=0.003), neutrophil (r=0.586, p=0.002), and basophil (r=0.518, p=0.007) counts and the NLR (r=0.419, p=0.033) and BLR (r=0.430, p=0.029) correlated with the initial HB grade; no other immunological marker correlated with the HB grade at any time (p>0.05). However, no correlation was observed between any hematological marker and the HB grade in either the patients with BP or the other patients at any time (p>0.05).

Other Factors Prognostic of Final Outcomes

In all patients, age, sex, treatment onset time, recurrence status, any accompanying disease, or the initial hearing level of either side did not differ by final treatment outcome (all p>0.05). However, the ENoG (p=0.003) and HB grade (p<0.001) differed at all times. The ENoG of the patients who recovered was 57.89±2.94%, which was lower than that of the patients who exhibited unfavorable recovery (76.61±18.28%). During the entire follow-up period, the HB grade was better in patients who recovered well. In patients with BP, the ENoG (p=0.014), the initial HB grade (p=0.001), and the HB grades at other times (p<0.001) varied by treatment outcome. In patients with RHS, the HB grade (except that at week 2) differed significantly by the final treatment outcome (p=0.002 at the initial visit and p<0.001 at week 6 and weeks 10–16). The HB grades of the patients who recovered well were better than those who recovered poorly, both at the initial visit and in weeks 6 and 10–16.

Finally, we performed a forward conditional logistic regression analysis to identify the factors prognostic of the final treatment outcome. For patients with RHS, the covariates included the HB grade at the initial visit, the pretreatment NLR and neutrophil count, and the day-7 lymphocyte count. The initial HB grade was independently prognostic of recovery [B=−2.763, EXP (B)=0.063, 95% confidence interval (CI)=0.006–0.665, p=0.021]. However, no other factor was significant. For patients with BP, the initial HB grade [B=−2.119, EXP (B)=0.120, 95% CI=0.020–0.722, p=0.021] and the pretreatment eosinophil count [B=0.071, EXP=1.074, 95% CI=1.005–1.147, p=0.035] were included in a regression model. For all patients, the HB grade at week 2 [B=−2.707, EXP (B)=0.067, 95% CI=0.010–0.465, p=0.006] and the lymphocyte count on day 7 [B=0.002, EXP (B)=1.002, 95% CI=1.000–1.003, p=0.036] were significantly prognostic of treatment outcomes.

DISCUSSION

Some hematological findings differed in patients with APFP with favorable and unfavorable outcomes. Patients with BP with poorer outcomes exhibited lower WBC and eosinophil counts on day 7 than those with favorable outcomes. Patients with RHS with unfavorable outcomes exhibited a higher pretreatment neutrophil count and NLR and a lower day-7 lymphocyte count than those with favorable outcomes. For all patients, lower pretreatment and day-7 lymphocyte counts were observed in those with unfavorable outcomes, suggesting that the timing and extent of inflammation may vary by diagnosis.

We prescribed MPD for all patients. Corticosteroids may affect blood parameters to varying extents, increasing the circulating neutrophil levels by enhancing neutrophil production in the bone marrow, reducing cellular movement into the inflamed tissue, and inhibiting apoptosis, thereby increasing the WBC count [10–12]. Neutrophils are the first-line immune cells recruited to the sites of infection with bacteria, fungi, and viruses; these cells are both beneficial and harmful. Neutrophils prevent aggravation of infectious diseases and mortality from infection but can also damage healthy tissues by producing excessive levels of cytokines, defensins, peroxidases, and reactive oxygen species [13]. Steroids reduce the circulating eosinophil, monocyte, and lymphocyte numbers. The numbers of circulating T lymphocytes are reduced by emigration, inhibition of interleukin-2 synthesis and impaired release thereof, and induction of apoptosis [12]. However, an

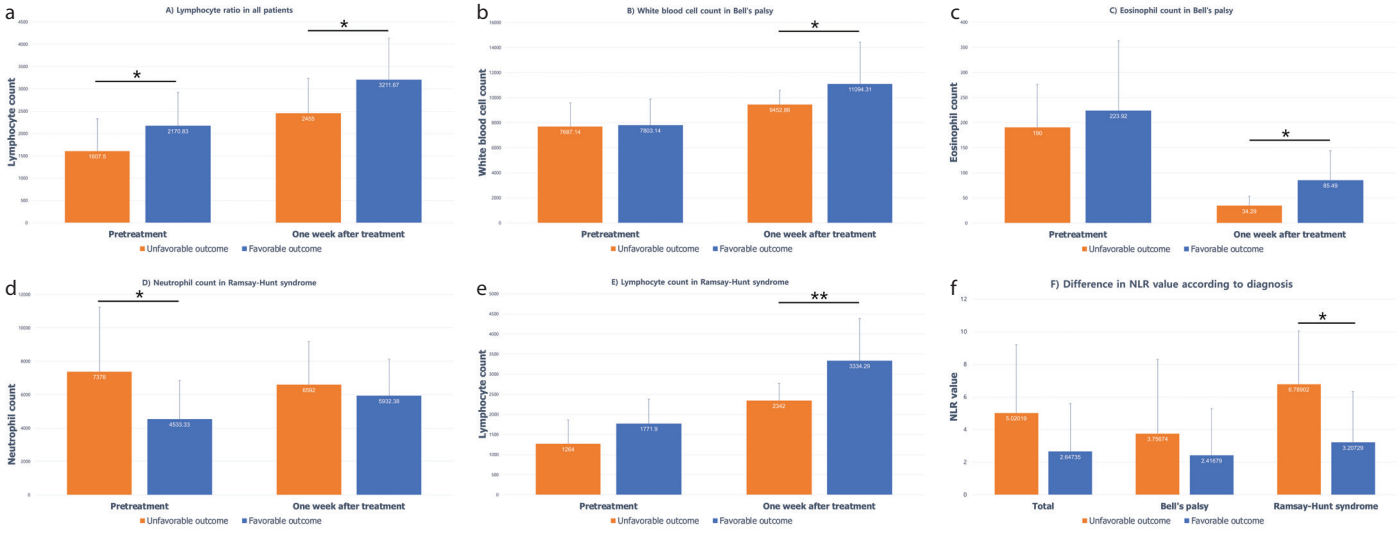

Figure 1. a-f. Hematological findings in patients with acute peripheral facial palsy. a) Lymphocyte ratio in all patients. b) White blood cell count in Bell's palsy. c) Eosinophil count in Bell's palsy. d) Neutrophil count in Ramsay Hunt syndrome. e) Lymphocyte count in Ramsay Hunt syndrome. f) Differences in NLR values according to diagnosis.

initial decrease followed by a later increase in the lymphocyte numbers was reported after MPD treatment [14]. Glucocorticoids reduce the blood eosinophil numbers by compromising their generation, survival, and function [15].

Consistent with common beliefs, different hematologic findings in APFP suggest that the pathological mechanism of APFP may vary with diagnosis. In patients with RHS, inflammation seems to play an important role from the time of palsy commencement. Initially, high neutrophil counts in patients experiencing unfavorable outcomes indicated that inflammation was more severe at the initial presentation. Furthermore, WBC, neutrophil, and basophil counts correlated with the initial HB grade, suggesting that RHS is basically an inflammatory disease. Patients experiencing favorable outcomes had higher lymphocyte counts on day 7. Steroids initially decrease but later increase the lymphocyte counts, and such patients may exhibit better responses to steroids.

In patients with BP, later inflammatory responses seem to be of more prognostic significance. A lower WBC count on day 7 predicted an unfavorable outcome; steroids may inadequately suppress late inflammation. In addition, the eosinophil counts on day 7 were higher in patients experiencing favorable outcomes. Eosinophilia can be triggered by an allergy, infection, autoimmune or idiopathic condition, and/or malignancy. In general, dose-dependent (negative) relationships between steroid levels and eosinophil counts have been reported in both patients with asthma and healthy controls [16, 17]. We assumed that an initial good anti-inflammatory response to steroids and a higher eosinophil count at the initial manifestation may enhance the BP prognosis. However, inflammation may not underlie all BP cases; the etiology of BP may be heterogeneous.

Next, a higher NLR, reflecting more severe inflammation, was observed in patients with RHS experiencing unfavorable outcomes, but the initial HB grade was the only independent factor prognostic of recovery; the NLR was not significant. The NLR may play only a limited prognostic role in patients with RHS in contrast to a previous study reporting that patients with RHS with higher NLRs experienced poor outcomes [18]. For BP, the NLR was not associated with BP severity or prognosis. Consistent with our finding, the NLR was not prognostic of recovery in children with BP [19]. A Turkish group reported that not only the NLR but also the platelet-to-lymphocyte (PLR) ratio may have some relevance in BP prognosis [20]. In contrast, a recent meta-analysis found no significant difference between the PLRs of patients with BP and controls; NLR was the only prognostic factor indicative of BP recovery [21].

Our findings differ from those of previous studies reporting higher NLRs in patients with BP experiencing poor outcomes [8], a positive correlation between the NLR and disease severity [22], longer recovery time for patients with higher NLRs [23], and higher NLRs in patients in whom the facial nerves were enhanced on MRI [24]. A Japanese group reported that the NLR was higher in an RHS than in a BP group, suggesting that the severity of inflammation caused by a viral infection might correlate with the prognosis [25]. They developed a prognostic tool employing pretreatment age, sex, and the NLR for patients with BP; and age, monocyte level, mean corpuscular volume, and platelet count for patients with RHS [25, 26]. Taken together, any clinical significance of the NLR, which reflects inflammation severity, in patients with BP may be limited; BP etiologies are heterogeneous. Apart from inflammation, microvascular compromise could play a role [24].

Our study had several limitations. Various factors can affect the CBC and thus the NLR (e.g., room temperature storage of blood samples before automated analyses and the type of analyzer employed) [27]. Moreover, our work was retrospective in nature, and the sample size was small.

CONCLUSION

Hematological findings of patients with APFP varied with diagnosis because the pathogenesis of APFP differs. Inflammation might play an important role in RHS onset. The initial severity of RHS and the response to corticosteroids may determine the final treatment outcome. BP is not triggered exclusively by inflammation, and several BP etiologies could be in play.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Eulji University (EMC IRB 2019-12-012).

Informed Consent: Informed consent is not necessary due to the retrospective nature of this study.

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Conflict of Interest: The authors have no conflict of interest to declare.

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