The Journal of International Advanced Otology

Int Adv Otol 2014; 10(3): 281-3 • DOI: 10.5152/iao.2014.250

Original Article

Increased Mean Platelet Volume in Patients with Bell's Palsy

Gül Soylu Özler, Şemsettin Okuyucu

Department of Otolaryngology, Mustafa Kemal University Faculty of Medicine, Hatay, Turkey

OBJECTIVE: The aim of this study is to investigate the mean platelet volume (MPV) levels in patients with Bell palsy (BP). Moreover, we aimed to find out any correlation between MPV levels and the severity and prognosis of BP.

MATERIALS and METHODS: The study group consisted of 30 subjects who presented with BP and 30 control subjects with no evidence of facial nerve pathology. The evaluation of subjects included a detailed history, general physical examination, and assessment of laboratory blood parameters.

RESULTS: The mean MPV and platelet distribution width (PDW) values in patients with BP were significantly higher than the control group (p=0.02, p=0.0001 respectively). The mean platelet count (PC) values in the BP group and control group were similar (p=0.169). There was positive correlation between MPV values and grade of facial paralysis (r=0.716, p=0.0001). Also, there was positive correlation between PDW values and grade of facial paralysis (r=0.376, p=0.041). In contrast, there was no correlation between MPV and PDW values and prognosis of facial paralysis (r= 0.275, p=0.142; r= 0.073, p=0.703 respectively).

CONCLUSION: There is no previous study that has investigated the association between MPV values and BP in the literature. Higher MPV values in BP patients may be a predictor of worse severity.

KEY WORDS: Bell's palsy, microcirculatory failure of the vasa nervosum, mean platelet volume

INTRODUCTION

Bell's palsy (BP) is an idiopathic, acute peripheral-nerve palsy involving the facial nerve. The annual incidence of BP is 15 to 30 per 100.000 persons[1]. Bell's palsy is named after Sir Charles Bell (1774-1842), who first described the syndrome. Although 2 centuries has passed after its first description, the etiology and prognostic factors of BP still remain unclear. Microcirculatory failure of the vasa nervosum, ischemic neuropathy, and infectious, genetic, and immunologic causes have been hypothesized as etiological factors [1-3].

Mean platelet volume (MPV) is an indicator of platelet functions, which reflects the platelet production rate and stimulation [4]. MPV levels increase in vascular events, like atherosclerosis, acute syndromes, venous and arterial thrombosis, or thromboembolism [5-7]. According to the theory of microcirculatory failure of the vasa nervosum, it may be a predictor for severity and prognosis in BP[8].

The aim of this study was to investigate any relationship between MPV levels and the severity and prognosis of BP.

MATERIALS and METHODS

Study Population

Thirty subjects who were referred to the otorhinolaryngology department of Mustafa Kemal University between December 2012 and April 2014 with BP were included in the study. The exclusion criteria for BP subjects were as follows: otitis media in the previous 4 weeks; trauma or barotrauma in the previous 4 weeks; a history of otologic surgery; neurologic disorders predisposing them to facial paralysis; neoplasm within the previous 2 years; or other major diseases (such as heart failure, hypertension, coronary artery disease, cor pulmonale, liver or renal dysfunction, diabetes mellitus, chronic obstructive pulmonary disease (COPD), obstructive sleep apnea, connective tissue diseases, inflammatory bowel diseases, Lyme disease) and smoking history. The control group was 30 subjects with no evidence of ear or facial nerve pathology or cardiovascular diseases. The evaluation of the subjects included a detailed history, general physical examination, and assessment of laboratory blood parameters. All patients received the same therapeutic protocol, which included intravenous administration of prednisolone (100 mg on the first day; 75 mg on second, third, and fourth days; 50 mg on the fifth and sixth days; and 25 mg from the seventh to tenth day) and acyclovir 500 mg intravenous thrice daily. Ethics committee approval was obtained, and the study was conducted, adhering to the Declaration of Helsinki. Informed consent was obtained from all participants.

Corresponding Address:
Gül Soylu Özler, Department of Otolaryngology, Mustafa Kemal University Faculty of Medicine, Hatay, Turkey
Phone: +90 505 398 07 78; E-mail: soylugul@yahoo.com

Submitted: 05.05.2014 Revision received: 27.06.2014 **Accepted:** 10.07.2014 **Available Online Date:** 15.10.2014

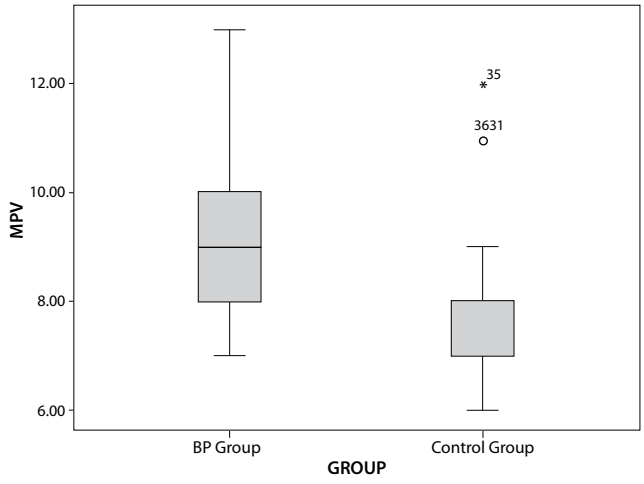

Figure 1. The mean MPV values of the BP group and control group (MPV-mean platelet volume, BP-Bell's palsy)
MPV: mean platelet volume; BP: Bell palsy

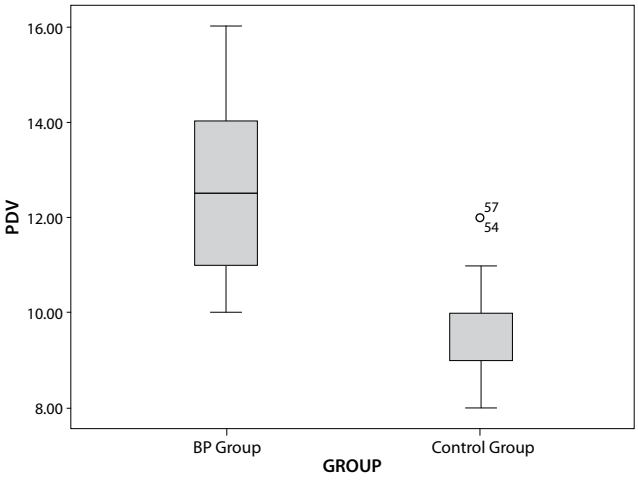

Figure 2. The mean PDW values of the BP group and control group (PDW-platelet distribution width, BP-Bell's palsy)
PDW: platelet distribution width; BP: Bell palsy

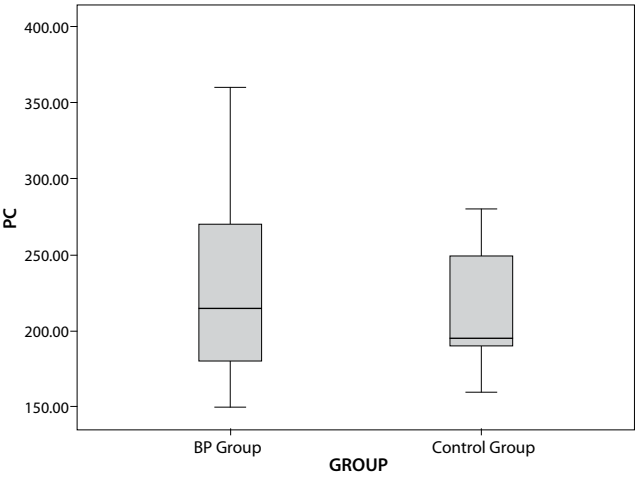

Figure 3. The mean PC values of the BP group and control group (PC-platelet count, BP-Bell's palsy)
PC: platelet count; BP: Bell palsy

Laboratory Evaluation

Haemogram were evaluated using peripheral venous blood samples obtained at admission. Blood samples were collected into tubes containing calcium ethylen ediamine tetra acetic acid (EDTA) tube at 8 a.m. following an overnight fast. Mean platelet volume (MPV), platelet distribution width (PDW) and platelet count (PC) were measured with an automated blood cell counter (Mindray BC 6800- Shenzhen Mindray Bio-Medical Electronics Co Ltd, Shenzhen, China). To avoid platelet swelling, MPV and PDW were measured in the blood samples between 15 and 30 min after sampling. All samples were run in duplicate.

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) 19.0 Evaluation for Windows. Descriptive statistics were stated as mean±SD (standard deviation). Normal distribution of continues variables were tested with Kolmogorov-Smirnov test. Chi-square test was used for comparisons between categorical variables and Mann-Whitney U tests were used for continuous variables when comparing the groups. The correlations between continuous variables were assessed by Pearson correlation coefficient.The statistically significant level was accepted as a p value<0.05.

RESULTS

Demographic Properties

Mean age of the patients with BP and the control group was 39.8± 10.68 and 37.1±6.91 years, respectively; 56.6% of the BP group and 50% of the control group were females. The groups were similar in terms of age and gender (p=0.262, p=0.799).

Evaluation of Facial Paralysis

According to the House-Brackmann grading system, the subjects presented with the following distribution 3 to 4 days after the initiation of the palsy[9]: 7 subjects were diagnosed with grade II, 9 subjects were diagnosed with grade III, 6 with grade IV, 5 with grade V, and 3 with grade VI. After a 3-month follow-up, complete recovery was seen in 24 (80%) subjects; 3 (10%) subjects still presented with grade III and 3 (10%) subjects with grade II paresis.

Laboratory Evaluation

The mean MPV values were 9.36±1.79 in the BP group and 7.96 ± 1.44 in the control group. The mean MPV values in the BP group were significantly higher than the control group (p=0.02) (Figure 1). There was a positive correlation between MPV values and grade of facial paralysis (r=0.716, p=.0001). The mean MPV values in the grade VI BP group were significantly higher than the other groups (p=0.0001). In contrast, there was no correlation between MPV values and prognosis of facial paralysis (r=0.275, p=0.142).

The mean PDW values were 12.43±1.63 in the BP group and 9.80±0.99 in the control group. The mean PDW values in the BP group were significantly higher than the control group (p=0.0001) (Figure 2). There was a positive correlation between PDW values and grade of facial paralysis (r=0.376, p=0.041). The mean PDW values in the grade VI BP group were significantly higher than the other groups (p=0.0001). There was no correlation between PDW values and prognosis of facial paralysis (r=0.073, p=0.703).

The mean PC values were 231.666±60.234 in the BP group and 213.333±39.639 in the control group. The mean PC values in the BP group and control group were similar (p=0.169) (Figure 3).

DISCUSSION

The most important finding of our study was that MPV and PDW levels were significantly higher in patients with BP than in the control group.

Mean platelet volume is one of the markers of platelet function. Increased MPV contributes to the prethrombotic state in acute syndromes. Therefore, larger platelets are hemostatically more active and may play a specific role in the development of ischemic stroke, coronary thrombosis, and myocardial infarction[10-13].

The etiology of BP is still uncertain and has been implicated in some theoretical pathways, such as viral infections[14-16], immune–mediated disease[17-19], and microcirculatory failure of the vasa nervosum [20-24].

According to our knowledge, MPV and PDW values in patients with BP have not been assessed previously. MPV and PDW values in our patients with BP were higher than in the control group, and positive correlations were observed between MPV and PDW values and the severity of BP at admission. In contrast, there was no correlation between MPV and PDW values and the prognosis of BP. This is the first study investigating the relationship between MPV and PDW levels and BP.

The limitation of our study is the number of subjects we investigated. Further studies with larger groups will be beneficial.

In conclusion, increased MPV and PDW values were observed in patients with BP. Moreover, higher MPV values in BP patients may be a predictor of worse severity.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Mustafa Kemal University/03.04.2014-14

Informed Consent: Informed consent was obtained from the patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - G.S.O., S.O.; Design - G.S.O.; Supervision - S.O.; Funding - G.S.O., S.O.; Materials - G.S.O., S.O.; Data Collection and/or Processing - G.S.O., S.O.; Analysis and/or Interpretation - G.S.O., S.O.; Literature Review - G.S.O., S.O.; Writing - G.S.O., S.O.; Critical Review - G.S.O., S.O.; Other - G.S.O., S.O.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Gilden DH. Clinical practice. Bell's palsy. N Engl J Med 2004; 351: 1323-31. [CrossRef]
2. Bibas T, Jiang D, Gleeson J. Disorders of the facial nerve. In: Gleeson M (ed). Scott-Brown's Otorhinolaryngology Head and Neck Surgery. 7th ed. London: Edward Arnold Ltd; 2008: 3883-6. [CrossRef]
3. Engstrom M, Thuomas K-Å, Naeser P, Stelberg E, Jonsson L. Facial nerve enhancement in Bell's palsy demonstrated by different gadolinium-enhanced magnetic resonance imaging techniques. Arch Otolaryngol Head Neck Surg 1993; 119: 221-5. [CrossRef]
4. Briggs C. Quality counts: new parameters in blood cell counting. Int Jnl Lab Hem 2009; 1: 277-97. [CrossRef]
5. Martin JF, Shaw T, Heggie J, Penington DG. Measurement of the density of platelets and its relationship to volume. Br J Haematol 1983; 54: 337-52. [CrossRef]
6. Machin SJ, Briggs C. Mean platelet volume: a quick easy determinant of thrombotic risk? J Thromb and Haemost 2009; 8: 146-7. [CrossRef]
7. Braekken SK, Mathiesen EB, Njolstad I. Mean platelet volume is a risk factor for venous thromboembolism: the Tromso study. J Thromb Haemost 2009; 8: 157-62. [CrossRef]
8. Smith IM, Heath JP, Murray JA, Cull RE. Idiopathic facial (Bell's) palsy: a clinical survey of prognostic factors. Clin Otolaryngol Allied Sci 1988; 13: 17-23. [CrossRef]
9. House JW, Backmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg 1985; 93: 146-7.
10. Bath PM, Butterworth RJ. Platelet size: measurement, physiology and vascular disease. Blood Coagul Fibrinolysis 1996; 7: 157-61. [CrossRef]
11. Butterworth R, Bath P.The relationship between mean platelet volume, stroke subtype and clinical outcome. Platelets 1998; 9: 359-64. [CrossRef]
12. Del Zoppo GJ. The role of platelets in ischemic stroke. Neurology 1998; 51: 9-14. [CrossRef]
13. Khandekar MM, Khurana AS, Deshmukh SD, Kakrani AL, Katdare AD, Inamdar AK. Platelet volume indices in patients with coronary artery disease and acute myocardial infarction: an Indian scenario. J Clin Pathol 200; 59: 146-9.
14. Mc Cormick DP. Herpes simplex virus as a cause of Bell's palsy. Lancet 1972; 1: 937. [CrossRef]
15. Leibowitz U. Epidemic incidence of Bell's palsy. Brain 1969; 92: 109-14. [CrossRef]
16. Kynvett AF. Viral infection of the nervous system. Med J Aust 1959; 2: 118-21.
17. Abramsky O, Webb C, Teitelbaum D, Arnon R. Cellular immune response to peripheral nerve basic protein in idiopathic facial paralysis (Bell's palsy). J Neurol Sci 1975; 26: 13-20.[CrossRef]
18. Mc Govern FH, Estevez J, Jackson R. Immunological concept for Bell's palsy: further experimental study. Ann Otol Rhinol Laryngol 1977; 86: 300-5. [CrossRef]
19. Mulkens PS, Bleeker JD, Schroeder FP. Acute facial paralysis: a virological study. Clin Otolaryngol 1980; 5: 303-9. [CrossRef]
20. Tomita M, Fukuuchi Y. Leukocytes, macrophages and secondary brain damage following cerebral ischemia. Acta Neurochirurgica, Supplement. 1996; 1996: 32-9.
21. Ross AM, Hurn P, Perrin N, Wood L, Carlini W, Potempa K. Evidence of the peripheral inflammatory response in patients with transient ischemic attack. J Stroke Cerebrovasc Dis 2007; 16: 203-7. [CrossRef]
22. Emsley HCA, Smith CJ, Tyrrell PJ, Hopkins SJ. Inflammation in acute ischemic stroke and its relevance to stroke critical care. Neurocritical Care 2008; 9: 125-38. [CrossRef]
23. Moxon-Emre I, Schlichter LC. Evolution of inflammation and white matter injury in a model of transient focal ischemia. J Neuropathol Exp Neurol 2010; 69: 1-15. [CrossRef]