# Mean Platelet Volume in Nasal Polyps

G Aktaş<sup>1</sup>, M Sit<sup>2</sup>, H Tekce<sup>1</sup>, A Alçelik<sup>1</sup>, H Savli<sup>1</sup>, T Simsek<sup>3</sup>, E Ozmen<sup>1</sup>, AZ Isci<sup>1</sup>, T Apuhan<sup>3</sup>

## ABSTRACT

**Background:** Many studies in literature point out that inflammation related to nasal polyp is mostly dependent on eosinophils and their inflammatory products. Beside eosinophils, platelets may have a role in nasal polyp development. Platelets are involved in haemostasis, tissue repairing and inflammation. However, to our knowledge, there are no reports in the literature that study the association between platelet parameters and nasal polyps.

**Subjects and Methods:** Forty-three patients with nasal polyps and forty-nine healthy controls were enrolled in the study, retrospectively. Laboratory data of patients with nasal polyp were obtained at the time of diagnosis.

**Results:** There were no statistically significant differences between the two groups in terms of white blood count, haemoglobin, haematocrit and platelet count. The mean platelet volume (MPV) value of the nasal polyp group was significantly lower than the control group (p = 0.025). Mean eosinophil count was significantly elevated in the nasal polyp group compared to the control group (p < 0.001). **Conclusions:** Reduction in MPV may be an indicator for nasal polyp formation. Further studies with a larger study population are needed to detect the possible correlation between eosinophil count and MPV values in patients with nasal polyps.

Keywords: Eosinophil, inflammation, mean platelet volume, nasal polyps, platelet

## Volumen Medio de las Plaquetas en los Pólipos Nasales

G Aktaş<sup>1</sup>, M Sit<sup>2</sup>, H Tekce<sup>1</sup>, A Alçelik<sup>1</sup>, H Savli<sup>1</sup>, T Simsek<sup>3</sup>, E Ozmen<sup>1</sup>, AZ Isci<sup>1</sup>, T Apuhan<sup>3</sup>

#### RESUMEN

Antecedentes: Muchos estudios en literatura señalan que las inflamaciones relacionadas con los pólipos nasales dependen en su mayoría de los eosinófilos y sus productos inflamatorios. Además de los eosinófilos, las plaquetas pueden jugar un papel en el desarrollo de los pólipos nasales. Las plaquetas participan en la hemostasia, la reparación de tejidos, y la inflamación. Sin embargo, que sepamos, la literatura existente no reporta estudios acerca de la asociación entre los parámetros de las plaquetas y los pólipos nasales.

**Sujetos y métodos:** Cuarenta y tres pacientes con pólipos nasales y cuarenta y nueve controles sanos se inscribieron en el estudio, de forma retrospectiva. Se obtuvieron datos de laboratorio de los pacientes con pólipos nasales en el momento del diagnóstico.

**Resultados:** No se encontraron diferencias estadísticamente significativas entre los dos grupos en términos de glóbulos blancos, hemoglobina, hematocritos y conteo de plaquetas. El valor medio de las plaquetas (MPV) del grupo con pólipos nasales fue significativamente menor que el del grupo control (p = 0.025). El conteo medio de eosinófilos fue significativamente elevado en el grupo con pólipos nasales en comparación con el grupo control (p < 0.001).

**Conclusiones:** La reducción de MPV puede ser un indicador para la formación de pólipos nasales. Se necesitan estudios adicionales con una mayor población de estudio para detectar la posible correlación entre los valores de MPV y el conteo de eosinófilos en pacientes con pólipos nasales.

Correspondence: Dr G Aktaş, Abant Izzet Baysal University, Medical Faculty, Department of Internal Medicine, Bolu, Turkey. E-mail: draliaktas@yahoo.com

From: <sup>1</sup>Department of Internal Medicine, <sup>2</sup>Department of General Surgery and <sup>3</sup>Department of Otolaryngology, Abant Izzet Baysal University Hospital, Bolu, Turkey.

Palabras claves: Eosinófilos, inflamación, volumen medio de plaquetas, pólipos nasales, plaquetas

West Indian Med J 2013; 62 (6): 516

#### **INTRODUCTION**

Nasal polyps are oedematous inflammatory lesions located in the nasal cavity and paranasal sinuses. They originate from paranasal sinus mucosa and protrude into the nasal cavity. Nasal polyps contain oedematous stroma and inflammatory cells (1). Nasal obstruction, purulent or non-purulent secretion, headache and loss of smell are complications of nasal polyps (2).

A number of inflammatory molecules are expressed in nasal polyp tissue. Many studies in the literature point out that inflammation related to nasal polyp is mostly dependent on eosinophils and their inflammatory products (3). Activated eosinophils are involved in nasal polyp formation (4). Furthermore, eosinophil survival time is longer in nasal polyp tissue compared to normal tissue (5, 6).

Platelets play a key role in haemostasis and tissue repairing (7). In addition to certain actions in haemostasis and thrombosis, it is now understood that platelets have a decisive function in the process of inflammation. Recent observations found out that platelets are involved in the accumulation of eosinophils and the development of inflammation in the lungs of allergen-sensitized mice (8). They have cytoplasmic granules that contain several substantial inflammatory products, such as platelet activation factor (PAF), platelet-derived growth factor (PDGF), platelet factor 4 (PF4), beta-thromboglobulin (b-TG), interleukin-1 (IL-1), leukotriens and prostaglandins (9, 10). CD40 ligand (CD40L) is also expressed on activated platelets (11). It is stored in platelet cytoplasm and shed when platelets are activated. Secreted CD40L directly stimulates inflammation on the endothelium. Several studies have revealed an association between platelets and certain diseases (12–14).

Mean platelet volume (MPV) is a widely used marker of platelet function. It reflects activation and production rate of the platelets (15). It has been shown that it reflects the inflammatory process in some conditions, such as preeclampsia, unstable angina, myocardial infarction, ulcerative colitis and Crohn's disease (16–24). However, to our knowledge, there are no reports on the association between platelet parameters and nasal polyps.

In this retrospective study, we aimed to investigate the correlation between nasal polyps and MPV.

#### SUBJECTS AND METHODS

Forty-three patients with nasal polyps and forty-nine healthy controls were enrolled in the study. None of the subjects in the study and control groups had a history of use of medications. Laboratory data of patients with nasal polyps were obtained at the time of diagnosis before nasal polyp surgery. White blood cell count (WBC), eosinophil count (EOS), haemoglobin (Hb), haematocrit (Htc), platelet count (PLT) and MPV of the participants were obtained from computerized medical database of the hospital.

Venous blood samples were collected in sterile standard tubes containing constant amounts of anticoagulant. Laboratory tests were conducted within several minutes after blood samples were obtained. The complete blood count analyses were performed using an automatic analyser of LH 780 model of Beckman Coulter device (Beckman Coulter, Inc., Brea, CA, USA). Original kits of the producer were used in the analyses.

Data were assessed by using SPSS programme (SPSS 15.0; SPSS Inc., Chicago, IL, USA). Results were expressed as mean  $\pm$  SD. Variables were conducted with independent samples *t*-test and Mann-Whitney U test. Pearson correlation was used to examine the association between MPV and eosinophil count. A *p*-value of < 0.05 was considered as statistically significant. The study was approved by the local ethics committee of Abant Izzet Baysal University School of Medicine.

#### RESULTS

There were 34 males and nine females in the nasal polyp group and 32 males and 17 females in the control group. Although there were more female subjects in the control group, the difference did not reach statistical significance (p =

Table: General features and laboratory data of the study group

	Nasal polyp group	Control group	<i>p</i> -value
Mean age (years)	39.1 ± 12.9	38.3 ± 10.6	0.74
Gender	34 males, 9 females	32 males, 17 females	0.14
WBC (cells/mm <sup>3</sup> )	$7000 \pm 1900$	$7300 \pm 2100$	0.54
Hb (g/dL)	$15.1 \pm 1.4$	$14.6 \pm 1.1$	0.14
Htc (%)	$44.4 \pm 3.8$	$43.1 \pm 3.1$	0.08
PLT (cells/mm <sup>3</sup> )	$271\ 000\pm 71\ 000$	$250\ 000\pm 62\ 000$	0.14
MPV (fL)	$7.54 \pm 0.7$	$7.9 \pm 0.9$	0.025
Eosinophil count (cells/mm <sup>3</sup> )	$440 \pm 320$	$150 \pm 84$	< 0.0001

 $WBC-white \ blood \ cell \ count, \ Hb-haemoglobin, \ Htc-haematocrit, \ PLT-platelet \ count, \ MPV-mean \ platelet \ volume$ 

0.146). Mean age of the subjects was  $39.1 \pm 12.9$  years in the nasal polyp group and  $38.31 \pm 10.6$  years in the control group. The difference was not statistically significant (p = 0.746). Furthermore, there were no statistically significant differences between the two groups in terms of WBC, Hb, Htc, and PLT (Table).

Mean MPV value in the nasal polyp group was lower than that of the control group (p = 0.025). Mean eosinophil count was significantly elevated in the nasal polyp group compared to the control group (p < 0.0001). The Table shows the general features and the laboratory data of the study population. Although both eosinophil count and MPV were significantly different in the nasal polyp group compared to the control group, no correlation was found between MPV and eosinophil count by Pearson test.

#### DISCUSSION

In this study, we found that MPV values and eosinophil counts of patients with nasal polyps were significantly different from the control group.

Eosinophils have a significant effect in the development of nasal polyps (4). Patients with nasal polyps have elevated eosinophil counts. Several studies in the literature have demonstrated that interleukin-5 levels and survival of eosinophils is significantly increased in nasal polyp tissue compared to non-polyp tissue (5, 6). Di Lorenzo *et al* reported that patients with nasal polyp have significantly elevated blood eosinophil levels compared to subjects without nasal polyp (25). These data were also confirmed by Arbag *et al* (26). In our study, eosinophil levels of patients with nasal polyp were significantly elevated compared to the control group, and this finding was compatible with the data in the literature.

Platelets are the smallest cells in blood. As well as haemostasis, activated platelets also have a key role in inflammation and tissue repair (7). Mean platelet volume is a marker of platelet activation (16, 18, 23, 27). Furthermore, it has been shown that MPV was associated with inflammatory burden of several diseases (16–24). Polinska *et al* enrolled 16 patients with ulcerative colitis and 32 healthy subjects as controls in their study. They found that patients with ulcerative colitis had significantly decreased MPV values compared to healthy subjects (7). Similarly, Yuksel *et al* noticed significantly reduced MPV in patients with ulcerative colitis compared to controls (28). Kapsoritakis *et al* confirmed these results in their study (23). Utilization and validation of MPV has not been established yet, but authors discuss the usage of MPV in inflammatory disorders (23).

Kisacik *et al* observed MPV values of 32 patients with rheumatoid arthritis, 30 patients with ankylosing spondylitis and 29 healthy subjects. They reported that MPV was significantly decreased in patients with rheumatoid arthritis and with ankylosing spondilitis compared to the control group (24). Similarly, we showed that MPV values of patients with nasal polyps were significantly lower than that of healthy subjects. This reduction in MPV in inflammatory diseases needs to be explained. Activated platelets tend to have a greater MPV and they might be utilized in such active inflammatory processes, leaving smaller platelets causing a reduction in MPV (7). Other authors speculate that overproduction of proinflammatory cytokines and acute phase reactants can suppress the dimensions of platelets by interfering with megakaryopoiesis in the bone marrow (16).

The results in the present study must be taken with caution because of the small sample population and retrospective design of our work. These results should be confirmed by randomized controlled trials. On the other hand, MPV and eosinophil counts were not correlated in our report. This may also be due to the small sample size.

In conclusion, MPV value may be associated with pathophysiological mechanisms and disease activity in nasal polyps. Further studies with larger study population are needed to detect the possible correlation between eosinophil count and MPV values in patients with nasal polyps.

### REFERENCES

- Jankowski R. Eosinophils in the pathophysiology of nasal polyposis. Acta Otolaryngol 1996; 116: 160–3. Epub 1996 Mar 01.
- Bachert C, Gevaert P, Holtappels G, Johansson S, Van Cauwenberge P. Total and specific IgE in nasal polyps is related to local eosinophilic inflammation. J Allergy Clin Immunol 2001; **107:** 607.
- Staikuniene J, Vaitkus S, Japertiene LM, Ryskiene S. Association of chronic rhinosinusitis with nasal polyps and asthma: clinical and radiological features, allergy and inflammation markers. Medicina (Kaunas) 2008; 44: 257–65. Epub 2008 May 13.
- Bachert C, Geveart P. Effect of intranasal corticosteroids on release of cytokines and inflammatory mediators. Allergy 1999; 54 (Suppl 57): 116–23. Epub 1999 Nov 24.
- Bachert C, Wagenmann M, Hauser U, Rudack C. IL-5 synthesis is upregulated in human nasal polyp tissue. J Allergy Clin Immunol 1997; 99: 837–42.
- Simon H-U, Yousefi S, Schranz C, Schapowal A, Bachert C, Blaser K. Direct demonstration of delayed eosinophil apoptosis as a mechanism causing tissue eosinophilia. J Immunol 1997; 158: 3902–8.
- Polinska B, Matowicka-Karna J, Kemona H. Assessment of the influence of the inflammatory process on the activation of blood platelets and morphological parameters in patients with ulcerative colitis (colitis ulcerosa). Folia Histochem Cytobiol 2011; 49: 119–24. Epub 2011 Apr 29.
- Pitchford SC, Momi S, Giannini S, Casali L, Spina D, Page CP et al. Platelet P-selectin is required for pulmonary eosinophil and lymphocyte recruitment in a murine model of allergic inflammation. Blood 2005; 105: 2074–81.
- Grove EL, Hvas AM, Kristensen SD. Immature platelets in patients with acute coronary syndromes. Thromb Haemost 2009; 101: 151–6. Epub 2009 Jan 10.
- Ranjith MP, Divya R, Mehta VK, Krishnan MG, KamalRaj R, Kavishwar A. Significance of platelet volume indices and platelet count in ischaemic heart disease. J Clin Pathol 2009; 62: 830–3. Epub 2009 Sep 08.
- Henn V, Slupsky JR, Grafe M, Anagnostopoulos I, Forster R, Muller-Berghaus G et al. CD40 ligand on activated platelets triggers an inflammatory reaction of endothelial cells. Nature 1998; **391:** 591–4. Epub 1998 Feb 19.

- Jayashree K, Manasa G, Pallavi P, Manjunath G. Evaluation of platelets as predictive parameters in dengue fever. Indian J Hematol Blood Transfus 2011; 27: 127–30.
- Fajardo LF, Rao S. Platelet enlargement in malaria. Mil Med 1971; 136: 463–4. Epub 1971 May 01.
- Varma N, Naseem S. Hematologic changes in visceral leishmaniasis/ kala azar. Indian J Hematol Blood Transfus 2010; 26: 78–82. Epub 2011 Sep 03.
- Briggs C. Quality counts: new parameters in blood cell counting. Int J Lab Hematol 2009; 31: 277–97. Epub 2009 May 20.
- Bath PM, Butterworth RJ. Platelet size: measurement, physiology and vascular disease. Blood Coagul Fibrinolysis 1996; 7: 157–61. Epub 1996 Mar 01.
- Choi CU, Seo HS, Kim YK, Na JO, Lim HE, Kim JW et al. Can mean platelet volume predict coronary vasospasm? Platelets 2011; 22: 173–8.
- Endler G, Klimesch A, Sunder-Plassmann H, Schillinger M, Exner M, Mannhalter C et al. Mean platelet volume is an independent risk factor for myocardial infarction but not for coronary artery disease. Br J Haematol 2002; 117: 399–404.
- Gasparyan AY, Stavropoulos-Kalinoglou A, Toms TE, Douglas KM, Kitas GD. Association of mean platelet volume with hypertension in rheumatoid arthritis. Inflamm Allergy Drug Targets 2010; 9: 45–50. Epub 2009 Nov 13.
- Ha S-I, Choi D-H, Ki Y-J, Yang J-S, Park G, Chung J-W et al. Stroke prediction using mean platelet volume in patients with atrial fibrillation. Platelets 2011; 22: 408–14.

- Järemo P, Lindahl T, Lennmarken C, Forsgren H. The use of platelet density and volume measurements to estimate the severity of preeclampsia. Eur J Clin Invest 2000; 30: 1113–8.
- Jaremo P, Sandberg-Gertzen H. Platelet density and size in inflammatory bowel disease. Thromb Haemost 1996; 75: 560–1. Epub 1996 Apr 01.
- Kapsoritakis AN, Koukourakis MI, Sfiridaki A, Potamianos SP, Kosmadaki MG, Koutroubakis IE et al. Mean platelet volume: a useful marker of inflammatory bowel disease activity. Am J Gastroenterol 2001; 96: 776–81. Epub 2001 Mar 31.
- Kisacik B, Tufan A, Kalyoncu U, Karadag O, Akdogan A, Ozturk MA et al. Mean platelet volume (MPV) as an inflammatory marker in ankylosing spondylitis and rheumatoid arthritis. Joint Bone Spine 2008; 75: 291–4.
- 25. Di Lorenzo G, Drago A, Esposito Pellitteri M, Candore G, Colombo A, Gervasi F et al. Measurement of inflammatory mediators of mast cells and eosinophils in native nasal lavage fluid in nasal polyposis. Int Arch Allergy Immunol 2001; **125**: 164–75. Epub 2001 Jul 04.
- Arbağ H, Kurnaz G, Eryılmaz MA. The role of allergy in etiology of nasal polyposis. Selçuk Üniv Tıp Derg 2011; 27: 59–61.
- Bath P, Algert C, Chapman N, Neal B. Association of mean platelet volume with risk of stroke among 3134 individuals with history of cerebrovascular disease. Stroke 2004; 35: 622–6.
- Yuksel O, Helvaci K, Basar O, Koklu S, Caner S, Helvaci N et al. An overlooked indicator of disease activity in ulcerative colitis: mean platelet volume. Platelets 2009; 20: 277–81. Epub 2009 May 22.