Klinik Çalışma

Mean Platelet Volume as a Potential Marker for Predicting Hypoxia in Children with Adenotonsillar Hypertrophy

Şahin Ulu*, Abdulkadir Bucak*, Reşit Köken**, Selçuk Kuzu*, Abdullah Ayçiçek*

Abstract

Objective: Adenotonsillar hypertrophy is a common cause of upper airway obstruction in children in the age group of 2-12 years. It is strongly associated with obstructive manifestations. Mean platelet volume (MPV) is one of the platelet activation markers. It reflects the platelet production rate which increases in hyopxia.We aimed to evaluate the relationship between MPV levels and adenotonsillar hypertrophy.

Materials and Methods: Sixty-seven patients which were operated after the diagnosis of adenotonsillar hypertrophy and 41 age and sex matched healthy subjects were enrolled to the study. Comparative multivariate analyses between indicator factors and adenotonsillar hypertrophy were conducted.

Results: Patients and control group were similar in terms of age and gender. MPV and platelet distribution width (PDW) levels of the patients with adenotonsillar hypertrophy were significantly higher than the control group *(p<0,001), (p<0,001) respectively*.

Conclusion: As a conclusion, MPV may be helpful for the clinicians to evaluate adenotonsillar hypertrophy which may cause many cardiovascular complications due to obstruction and hypoxia. We should not overlook MPV values while evaluating these patients because it may speed up operation decision to prevent its complications.

Key words: Mean platelet volume, hypoxia, airway obstruction

Introduction

Adenotonsillar hypertrophy is the most common cause of the upper airway obstruction. Adenotonsillar hypertrophy commonly occurs between the ages of 2 and 12 years and frequently leads to pharyngeal obstruction (1). Many studies in the literature show that adenotonsillar hypertrophy causes hypoxia, pulmonary hypertension, ventricular hypertrophy, systemic hypertension and poorer quality of life (2,3). The decision of operation should be given more quickly in children with adenotonsillar hypertrophy to prevent complications.

*Department of Otolaryngology, Afyon Kocatepe University, Faculty of Medicine, Afyonkarahisar, Turkey
**Department of Pediatrics, Afyon Kocatepe University Faculty of Medicine, Afyonkarahisar, Turkey
Corresponding Author: Sahin ULU, MD
Department of Otorhinolaryngology, School of Medicine, University of Afyon Kocatepe, Afyonkarahisar, TURKEY, 03200
Phone: 90 272 2463321
Fax: 90 272 2133066
e-mail: drsahinulu@yahoo.com
Makalenin Geliş Tarihi: 14.01.2014
Makalenin Kabul Tarihi: 07.07.2014

Severe adenotonsillar hypertrophy is one of the main causes of nocturnal respiratory pathologies and hypoxia as confirmed by the improvement of symptoms seen after adenotonsillectomy (4).

MPV is a parameter as part of routine complete blood count tests which is usually overlooked by clinicians and it is one of the most widely used surrogate markers of platelet function (5,6). It is known that, large platelets are more adhesive and likely to aggregate than small ones (7).

Platelet activation and increased MPV levels in hypoxic conditions were shown before (8).

Recently, in our previous study, we showed the relationship between nasal obstruction and MPV by using acoustic rhinometry in patients with septum deviation (9).

But according to our knowledge, MPV values in adenotonsillar hypertrophy has not been investigated to date. Based on this background we aimed to evaluate this relationship.

Materials and Methods

Study Population

Sixty-seven patients which were operated for adenotonsillar hypertrophy and age and sex matched 41 healthy control subjects were enrolled to the study. Our study was approved by the local ethics committee and conducted in accordance with the ethical principles described by the Declaration of Helsinki. Written informed consent form was obtained from all participants before the study. The diagnosis of adenotonsillar hypertrophy was based on physical and endoscopic examination. Patients who had severe adenotonsillar hypertrophy (grade 3+ and 4+ tonsillar hypertrophy and adenoid hypertrophy larger than >75%) were enrolled to the study. Patients who were operated for chronic or recurrent tonsillitis and /or adenoiditis, with turbinate hypertrophy, septal deviation, diseases causing nasal passage obstruction such as nasal polyps, systemic disease such as any known cardiac or lung disease, any arhythmias, diabetes mellitus, chronic renal or hepatic disease and any infection were excluded.

Physical Examination

All subjects with adenotonsillar hypertrophy underwent physical examination of nasal passages, nasopharynx, oropharynx, larynx by flexible fiberoptic endoscope (Karl Storz 11101 VP, Germany). The degree of tonsillar hypertrophy was determined. *(0)* Tonsils are entirely within the tonsillar fossa. *(1+)* Tonsils occupy less than 25 percent of the lateral dimension of the oropharynx as measured between the anterior tonsillar pillars. *(2+)* Tonsils occupy less than 50 percent of the lateral dimension of the oropharynx. *(3+)* Tonsils occupy less than 75 percent of the lateral dimension of the oropharynx. *(4+)* Tonsils occupy 75 percent or more of the lateral dimension of the oropharynx (10). Then patients with grade 3-4 were enrolled to the study.

It was considered as small adenoid, when adenoid occupied less than half of choana; medium adenoid, around 50% and 70% of the choana; and it was considered as large adenoid, when it occupied over 75% of full choanal area (11).

Biochemical and Haematological Analyses

MPV, Platelet distribution width (PDW) and Platelet count (PC) measurements were evaluated in blood samples before the treatment. Initially, patients with abnormal findings in fasting glycemia, creatinine, total cholesterol and fractions, triglycerides and TSH were excluded. Venous blood samples for hemogram were collected into tubes containing ethylenediaminetetraacetic acid (EDTA) at 8 a.m. following an overnight fast. To avoid platelet swelling, MPV and PDW were measured in the blood samples between 15-30 min after sampling. An automated blood cell counter was used for these measurements (Sysmex XT 2000i, Kobe, Japan). All samples were run in duplicate, and the mean values were used for statistical analysis.

Statistical Analysis

SPSS 18 programme was used for statistical analysis. The data were summarized using descriptive statistics. Chi-square test and Student's t tests were used to compare the groups. Spearman correlation analysis was used to explore the relationship between the parameters. $p<0.05$ were accepted as significant.

Results

Male/Female ratio of patients with adenotonsillar hypertrophy was 42/25 and the control group’s was 24/17. Mean age of patients with adenotonsillar hypertrophy was 5.9±1.8 years and the control group’s was 5.8±2.4 years. Patients and control groups were similar in terms of age and gender. The characteristics, MPV and PDW values and platelet counts of the adenotonsillar hypertrophy patients and the control group were outlined in Table 1.

Mean MPV values of adenotonsillar hypertrophy patients was 9.3±0.7 fl and the control group’s was 8.6±0.4fl. Mean MPV values in patients with adenotonsillar hypertrophy were significantly higher than the control group ($p<0.001$) (Figure 1).

Mean PDW values of patients were 10.5±1.2 fl and the control group’s were 9.4±0.7 fl. Mean PDW values in patients with adenotonsillar hypertrophy were significantly higher than the control group ($p<0.001$).

Mean platelet count values of patients were 359000 ± 71000/u and the control group’s were 384000 ± 122000/u. MPV values in patients with adenotonsillar hypertrophy were lower than the control group but it could not reach to statistical level ($p=0.185$).

Table 1. The characteristics, MPV, PDW values and platelet counts of the ATH patients and the control group

Variables	Control Group mean±st. Deviation	Patient Group mean±st. deviation	p value
Age (year)	5.8 ± 2.4	5.9± 1.8	0.800
MPV (fl)	8.6 ± 0.4	9.3 ± 0.7	**<0.001**
PDW (fl)	9.4 ± 0.7	10.5 ± 1.2	**<0.001**
Platelet(\u)	384000± 122000	359000± 71000	0.090

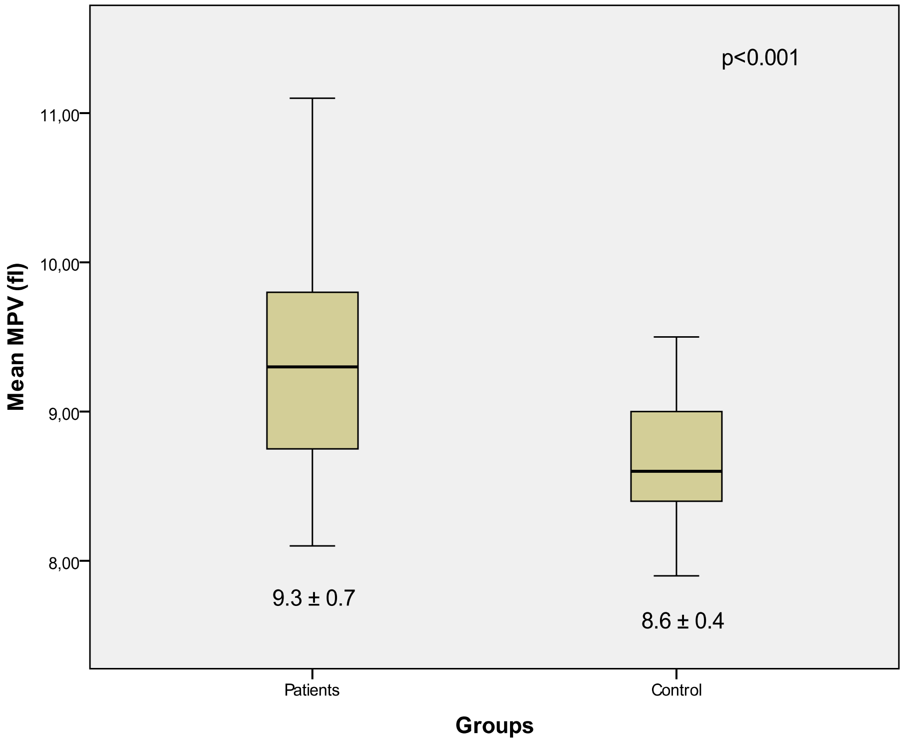

Fig. 1. Mean MPV values of the groups (patients with adenotonsillar hypertrophy and the control group).

MPV and PDW values were correlated significantly in adenotonsillar hypertrophy patients (p<0.001, r=0.458; p<0.001, r=0.457 respectively). MPV levels were not correlated with the degree of adenotonsillar hypertrophy.

Discussion

According to our knowledge, this is the first study investigating MPV levels in patients with adenotonsillar hypertrophy. In our study, MPV levels in patients with severe adenotonsillar hypertrophy were found significantly higher than the control group. This may mean that, severe adenotonsillar hypertrophy may cause platelet activation via hypoxia.

Adenotonsillar hypertrophy is a common cause of upper airway obstruction in children in the age group of 2-12 years. While investigating the etiology of hypoxia, adenotonsillar hypertrophy is frequently being overlooked as a cause of hypoxia. Whereas adenotonsillar hypertrophy may cause many pulmonary and cardiovascular complications beside poorer quality of life (12,13,14). Adenotonsillar hypertrophy causes pulmonary hypertension, ventricular hypertrophy and systemic hypertension, poorer life quality and more severe adenotonsillar hypertrophy symptoms in the pediatric population via causing OSA (2).

MPV is one of the platelet activation markers which reflects the platelet production rate, stimulation, activation and aggregation. It is one of the most widely used surrogate markers of platelet function today (5-7).

MPV levels increase in hypoxic conditions via elevated IL-6 levels which can lead to platelet activation (8,15). The relationship between nasal obstruction caused hypoxia and increased MPV levels was shown before (9).

MPV values may increase due to hypoxia in adenotonsillar hypertrophy. However, there has not been any study about this subject to date. Based on our study, It may be suggested that; MPV may be a novel-potential indicator of hypoxia in patients with adenotonsillar hypertrophy and may be a simple and helpful marker for the clinicians to give the operation decision to prevent many cardiovascular complications due to adenotonsillar hypertrophy.

This study had some limitations. Firstly, this is a retrospective study and patients had not polysomnography test and post-operative MPV values. In addition, MPV levels were not correlated with the degree of adenotonsillar hypertrophy. This result may be due to our small sample size composed from patients with severe adenotonsillar hypertrophy. We believe that, new studies with broad participation which had polysomnography test and post-operative MPV values would shed light on this issue.

Ortalama Trombosit Hacmi Adenotonsiller Hipertrofisi Olan Çocuklarda Hipoksi Belirteci Olarak Potansiyel Marker

Özet

***Amaç:** Adenotonsiller hipertrofi, 2-12 yaş grubundaki çocuklarda üst solunum yolu obstrüksiyonunun sık görülen bir nedenidir. Ortalama trombosit hacmi (MPV) trombosit aktivasyonu endekslerinden biridir ve trombosit üretim hızını yansıtır. Hipoksi durumlarında MPV ve trombosit üretim hızı artar. Çalışmamızda, MPV düzeyleri ile adenotonsiller hipertrofi arasındaki ilişkiyi incelemeyi amaçladık.*

***Gereç ve Yöntem:** Adenotonsiller hipertrofi tanısı sonrasında opere edilen 67 hasta ve 41 yaş ve cinsiyet uyumlu sağlıklı birey çalışmaya dahil edildi. Araştırılan belirteçler ve adenotonsiller hipertrofi arasında karşılaştırmalı çok değişkenli analizler yapıldı.*

***Bulgular:** Hastalar ve kontrol grubu yaş ve cinsiyet açısından benzerdi. Adenotonsiller hipertrofi si olan hastaların MPV ve trombosit dağılım genişliği (PDW) düzeyleri kontrol grubuna göre anlamlı olarak yüksek bulundu. (p<0,001, p <0,001) sırasıyla.*

***Sonuç:** Sonuçta MPV, obstrüksiyon ve hipoksi nedeniyle birçok kardiyovasküler komplikasyona neden olabilen adenotonsiller hipertrofisini değerlendirmede klinisyenler için faydalı olabilir. Bu hastaların değerlendirilmesinde olası komplikasyonları önlemek amacıyla operasyon kararını hızlandırmak için MPV değerleri göz ardı edilmemelidir.*

***Anahtar kelimeler:** Ortalama Trombosit Hacmi, hipoksi, havayolu tıkanması*

References

1. Chang SJ, Chae KY. Obstructive sleep apnea syndrome in children: Epidemiology, pathophysiology, diagnosis and sequelae. Korean J Pediatr 2010; 53(10):863-871.
2. Tatlıpınar A, Biteker M, Meriç K, Bayraktar Gİ, Tekkeşin Aİ, Gökçeer T. Adenotonsillar hypertrophy: correlation between obstruction types and cardiopulmonary complications. Laryngoscope 2012; 122(3):676-680.
3. Tatlipinar A, Duman D, Uslu C, Egeli E. The effects of obstructive sleep apnea syndrome due to adenotonsillar hypertrophy on the cardiovascular system in children. Turk J Pediatr 2011; 53(4):359-363.
4. Passàli D, De Benedetto M, Lauriello M, Passàli FM. Influence of Waldeyer's ring hypertrophy on snoring and sleep apnea. Adv Otorhinolaryngol 2011; 72:132-135.
5. 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(3): 776-781.
6. 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(3):291-294.
7. Schoene NW. Design criteria: tests used to assess platelet function. Am J Clin Nutr 1997; 65(5 Suppl): 1665-1668.
8. Sagit M, Korkmaz F, Kavugudurmaz M, Somdas MA. Impact of septoplasty on mean platelet volume levels in patients with marked nasal septal deviation. J Craniofac Surg 2012; 23(4):974-976.
9. Ulu S, Ulu MS, Bucak A, Kahveci OK, Yucedag F, Aycicek A. Evaluating the relationship between nasal obstruction and mean platelet volume by using acoustic rhinometry in patients with septum deviation. Rhinology 2013; 51(3):249-252.
10. Chan J, Edman JC, Koltai PJ. Obstructive sleep apnea in children. Am Fam Physician 2004; 69(5):1147-1154.
11. Lourenço EA, Lopes Kde C, Pontes A Jr, Oliveira MH, Umemura A, Vargas AL. Comparison between radiological and nasopharyngolaryngoscopic assessment of adenoid tissue volume in mouth breathing children. Braz J Otorhinolaryngol 2005; 71(1):23-27.
12. Balbani AP, Weber SA, Montovani JC. Update in obstructive sleep apnea syndrome in children. Braz J Otorhinolaryngol 2005; 71(1):74-80.
13. de Serres LM, Derkay C, Astley S, Deyo RA, Rosenfeld RM, Gates GA. Measuring quality of life in children with obstructive sleep disorders. Arch Otolaryngol Head Neck Surg 2000; 126(12):1423-1429.
14. Goodman RS, Goodman M, Gootman N, Cohen H. Cardiac and pulmonary failure secondary to adenotonsillar hypertrophy. Laryngoscope 1976; 86(9):1367-1374.
15. Nena E, Papanas N, Steiropoulos P, Zikidou P, Zarogoulidis P, Pita E, et al. Mean Platelet Volume and Platelet Distribution Width in non-diabetic subjects with obstructive sleep apnoea syndrome: new indices of severity? Platelets 2012; 23(6):447-454.