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MPV Values in Children with Adenoid Hypertrophy and Correlation with Adenoid Size

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Abstract

Background: Adenoid hypertrophy (AH) leads to chronic upper airway obstruction. Upper airway obstruction may cause pulmonary hypertension and right ventricle dysfunction. Mean platelet volume (MPV) may be used as a marker of platelet activation and it is shown that MPV is related to cardiovascular disorders. We investigated in our study if MPV is correlated with adenoid size and can be used as an indicator of obstruction due to adenoid hypertrophy. Methods: 95 children with AH and 99 healthy controls were enrolled in the study. White blood cell, hemoglobin, platelet, Mean platelet volume (MPV) levels were measured before adenoidectomy. Lateral cephalometric graphs were obtained from all patients to measure adenoid/nasopharynx ratio (A/N). The distance between the outermost point of convexity of adenoid shadow and basiocciput was divided to the distance between sphenobasiocciput and posterior end of the hard palate. A/N ratio and MPV levels have been correlated by using Pearson correlation test. Results: There were 45 male and 50 female patients between the ages of 2 and 14 years (mean age: 6.47 ± 2.44). There were no significant differences between children with AH and controls regarding white blood cell, hemoglobin, platelet, MPV and A/N ratio. We didn't detect any correlation between MPV levels and A/N ratio. Conclusion: MPV level was in normal limits in AH children and there was no significant relation between MPV and adenoid severity.

Keywords

Adenoid Size, Adenoid Vegetation, Children, Mean Platelet Volume

Subject Areas: **Otorhinolaryngology, Pediatrics**

1. Introduction

Adenoid hypertrophy (AH) commonly leads to upper airway obstruction, obstructive sleep apnea and hypoxia in

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children [1]. Obstructive sleep apnea causes hypoxia, pulmonary hypertension and right ventricle (RV) dysfunction [2]. Obstructive sleep apnea exists in 1% - 3% of children and can occur at any age [3]. The adenoids, like all lymphoid tissue, enlarge when infected. Although lymphoid tissue does act to fight infection, sometimes bacteria and viruses can lodge within it and survive. Chronic infection, either viral or bacterial, can keep the pad of adenoids enlarged for years, even into adulthood. Some viruses, such as the Epstein-Barr Virus, can cause dramatic enlargement of lymphoid tissue. Primary or reactivation infections with Epstein Barr Virus, and certain other bacteria and viruses, can even cause enlargement of the adenoidal pad in an adult whose adenoids had previously become atrophied. Enlarged adenoids can become nearly the size of a ping pong ball and completely block airflow through the nasal passages. Even if enlarged adenoids are not substantial enough to physically block the back of the nose, they can obstruct airflow enough so that breathing through the nose requires an uncomfortable amount of work, and inhalation occurs instead through an open mouth. Adenoids can also obstruct the nasal airway enough to affect the voice without actually stopping nasal airflow altogether [4].

Platelet size is correlated with platelet activation and large platelets have more hemostatic activation [5]. So mean platelet volume (MPV) may be used as a marker of platelet activation and may also be associated with atherosclerosis as a simple assessment tool [6]. MPV levels increase in some conditions such as hypertension, hypercholesterolemia, diabetes mellitus, acute myocardial infarction and acute ischemic stroke [7]. Varol *et al*. [8] suggested that MPV levels were higher in adult patients with severe obstructive sleep apnea than healthy patients. Sagit *et al*. [9] showed that patients with septal deviation had increased MPV levels. Onder *et al*. [10] revealed no significant relation between MPV levels and obstructive adenoid hypertrophy.

There are conflicting studies on MPV levels in patients with obstructive conditions. So we aimed to investigate the MPV levels in AH patients and the relationship between MPV levels and adenoid size in children.

2. Methods

The current study consisted of 95 patients presented to the pediatrics outpatient clinic of Bozok University Medical faculty with AH (45 male, 50 female; mean age: 6.47 years).

Children with nasal septal deviation, sinonasal infection, hematological disease, chronic diseases, and history of previous adenoidectomy were excluded from the study

Routine preoperative blood samples were drawn from the antecubital vein by careful vein puncture. Blood samples were collected in EDTA, and laboratory data were screened via a computerized database in the hospital. The reference values for MPV ranged between 5.0 and 15.0 f L.

Lateral cephalometric graphs were obtained from all patients to measure adenoid/nasopharynx (A/N) ratio. A/N ratio has been shown to have a good correlation with endoscopic examination findings [11]. A/N ratio was calculated as defined by Fujioka *et al.* [12] before. The distance between the outermost point of convexity of adenoid shadow and basiocciput was divided to the distance between sphenobasiocciput and posterior end of the hard palate.

Statistical Analyses

The statistical analyses were carried out by Statistical Package for Social Sciences (SPSS) 18. Before performing the Student t test, the data was checked for normality of distribution by Kolmogorov-Smirnov test. Variables were expressed as mean ± SD. Comparisons of variables were performed using unpaired Student *t* test and Chi-square test. Bivariate associations of the variables were assessed using Pearson's correlation coefficients and p value < 0.05 was considered indicate statistical significance.

3. Results

The AH group consisted of 95 patients (50 females and 45 males) with a mean age of 6.47 years, and the control group had 99 patients (57 females and 42 males) with a mean age of 5.77 years. There were no significant differences between the study and control groups in terms of age and sex. All characteristics of the AH and control groups, including MPV, hemoglobin, WBC, MCV and MPV are listed in **Table 1**.

The mean MPV levels were 9.39 f L in patients with AH and 9.64 f L in the control group and there was no statistical differences (p = 0.156). The mean ANS level was 0.78 ± 0.065 in patients with AH.

There were no correlation between ANS and other parameters by Pearson correlation analysis.

Table 1. Comparison (mean ± SD) of clinical characteristics.

Variable	Children with adenoid hypertrophy	Healthy controls	*p* value
	n = 95	n = 99	
Age (years)	6.47 ± 2.44	5.77 ± 3.10	0.081
Male/female	45/50	42/57	0.314[&]
WBC	8.78 ± 2.56	9.23 ± 3.10	0.280
Hb	12.54 ± 0.96	12.75 ± 1.34	0.198
Platelet	337.74 ± 81.31	338.82 ± 76.44	0.924
MCV	85.98 ± 7.85	79.82 ± 4.53	0.409
MPV	9.39 ± 1.13	9.61 ± 0.94	0.156

Student's t test, [&]Chi-square test, *p* level is significant at the <0.05 level. WBC: white blood cell, Hb: hemoglobin, MCV: mean corpuscular volume, MPV: mean platelet volume.

4. Discussion

AH causes upper airway obstruction and leads to increased pulmonary hypertension and corpulmonale [13] and also upper airway resistance syndrome by increased upper airway resistance as a result of labored breathing [14]. It is shown that adenotonsillectomy decreases pulmonary arterial hypertension to normal values in children [15]. When sufficient airflow is not meet nasal way, mouth breathing begins and this causes turbulent airflow. The intraluminal air pressure cannot reach the appropriate level to keep the nasopharyngeal and orophayngeal structures open in the supine position; moreover, the bulk of the tongue is displaced backwards by the influence of gravity, obstructing the airway. Therefore, this chain of events results in alveolar hypoventilation, hypoxia, and hypercarbia during sleep [14] [16].

If the children who had AH or tonsillar hypertrophy left untreated, they can experience abnormal orofacial anatomy, poor appetite, growth retardation, aggressive behavior, anxiety, impaired attention, depression, and somatization disorders over the long term [17] [18].

Chronic alveolar hypoxia causes hypoxemic pulmonary vasoconstriction that leads to hypertension and RV heart failure [19] [20]. MPV level is showed to be higher in patients with pulmonary hypertension [21]. MPV shows platelet activity and large platelets are more active and have more prothrombotic potential [9].

In current study we didn't found any differences between AH and healthy controls in terms of MPV level and we didn't detect any relationship between MPV values and adenoid size. One possible reason for this is that the study group included pediatric patients; chronic hypoxia exposure time is shorter in the pediatric population than in adult patients. MPV values in children may not be related to upper airway obstruction. Önder *et al*. [10] revealed that there was no significant relation between MPV levels and obstructive adenoid hypertrophy consisted with our results. Cevik *et al*. [22] found that MPV values were significantly lower in patients with nasal polyps than controls. Yılmaz *et al*. [23] been showed that adenotonsillary hypertrophy causes higher pulmonary artery pressure values and adenotonsillectomy is an effective therapeutic measure in such patients. Varol *et al*. [24] revealed that six months of CPAP therapy caused significant reductions in median MPV values in adult patients with severe OSA. Sagit *et al*. [9] found that septoplasty has a curative effect for hypercoagulopathy with reducing MPV values. Kucur *et al*. [25] found that MPV values were found to be higher in children with AH than controls and there was a significant reduction in MPV levels after adenoidectomy. They demonstrated that MPV levels may indicate the risk of cardiopulmonary diseases. Steiropoulos *et al*. [26] also reported that patients with chronic obstructive pulmonary disease had high MPV values. In contrast, Çevik *et al*. [27] suggested that MPV values in children with AH were significantly lower than those in control subjects. It has been suggested that chronic hypoxia causes catecholamine-dependent platelet activation and that this leads to changes in platelet shape with an increase in platelet swelling, leading to an increase in MPV and PDW [28] [29]. Tuncel *et al*. [30] found that there is no significant difference in MPV values between asthmatic children and control groups.

5. Conclusion

MPV values are in normal limits in children with AH and are not correlated with adenoid size.

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