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Original Article

Platelet Indices in Pediatric Patients with Congenital Heart Disease with and without Pulmonary Arterial Hypertension

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Abstract

Background: Pulmonary arterial hypertension (PAH) is an uncommon syndrome characterized by dyspnea and fatigue resulting from a progressive elevation in pulmonary vascular resistance. Recent research suggests that the relationship between pulmonary arterioles and platelets could be connected to the ongoing changes in pulmonary blood vessels seen in PAH. Therefore, this study aimed to assess platelet indices in pediatric patients with PAH.

Methods: This study in pediatric cardiology was conducted in 2020. Laboratory and clinical data were obtained retrospectively from hospital records. All patients had undergone physical examination and echocardiography. The study included 44 pediatric cases with congenital heart disease (CHD).

Results: Mean and standard deviation (SD) of age in cases with and without PAH were 7.75 ± 3.64 and 5.97 ± 3.71, respectively; the male/female ratio in the two groups was 10:12. The mean and SD of pulmonary artery pressure (PAP) in cases with and without PAH were 36.81 ± 6.08 and 19.80 ± 2.78 mm Hg ; also, right ventricular systolic diameter (RVSD) and right ventricular diastolic diameter (RVDD) did not show a statistically significant difference in echocardiography. Hemoglobin (Hb) (P = 0.146), hematocrit (HCT) (P = 0.712), and platelet count did not show statistically significant differences. However, mean platelet volume (MPV), Platelet distribution width (PDW), and plateletcrit (PCT) in PAH cases were significantly higher than cases with normal pulmonary tension (P = 0.0001). Also, our study showed a correlation between MPV, PCT, PDW, and PAH.

Conclusion: Our study demonstrated a notable elevation in MPV and PDW among pediatric patients with PAH and identified a relationship between PDW and MPV in this patient population.

Keywords: Platelet count, Platelet function, Congenital heart disease, Pulmonary hypertension, Pediatrics

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Introduction

PAH is an uncommon syndrome characterized by dyspnea and fatigue resulting from a progressive elevation in pulmonary vascular resistance, which ultimately leads to right ventricular (RV) failure (1). PAH is defined as an increased resting pulmonary artery pressure (PAP) of more than 25 mm Hg , measured by the catheterization of the right heart. As PAH is a hemodynamic disorder rather than a primary disease, almost all disorders that influence the pulmonary vasculature could lead to pulmonary hypertension (PH) (2). As group 1 of the Venice classification, PAH includes a group of conditions that are defined based on thrombotic and hypertrophic obstructive changes in the arterial trees (3,4). Also, as a common side effect of left-to-right shunts, it increases the resistance of pulmonary vasculature, which can lead to considerable mortality and morbidity. The clinical categorization delineates five distinct groups of PH, with group 1 characterized by PAH (2,5).

The pathobiology of PAH is complex and involves multiple factors, with endothelial dysfunction as its primary component. Pulmonary vessel wall remodeling, thrombosis, and vasoconstriction contribute to enhanced resistance in pulmonary vessels (6). Each type of cell (smooth muscle, fibroblast, and endothelial cells), in addition to platelets and inflammatory cells, can significantly affect PAH (7).

Recent studies have indicated that the association between pulmonary arterioles and platelets may be linked to the progressive alterations in pulmonary vascularity observed in PAH. Platelet distribution width (PDW) measures the size of platelet variability and is considered a platelet activation marker (8,9). Medical professionals have utilized PDW to distinguish platelet disorders, such as reactive thrombocytosis, from essential thrombocythemia. Another indicator of platelet activation is mean platelet

volume (MPV) (10,11).

Based on the defect type, prediction of post-surgery reversibility is difficult. The risk factors of irreversible PAH in some pediatric cases with congenital heart disease (CHD) are poorly detected (4). CHD represents the most prevalent and significant congenital anomaly, occurring in approximately nine cases per 1000 live births (12). However, abnormal platelet activation and platelet abnormalities are important factors for the progression of PAH, as these factors can induce pulmonary artery obstruction and remodeling of vessels (13,14).

Our research is a case-control study involving a comprehensive examination of platelet factors in pediatric patients with CHD. The exact function of platelets in the onset of PAH in children is not thoroughly comprehended. Therefore, this study aimed to examine the relationship between this marker and the severity of PAH and assess whether these blood parameters could help predict the prognosis of PAH.

Methods

This was a matched case-control study conducted in 2020 at Amirkabir hospital in Arak city, Iran. The research population was divided into two groups:

- Group 1 included 22 children diagnosed with CHD with secondary PAH (case).
- Group 2 comprised 22 children with CHD but without PAH (control).

None of the patients had a family history of PAH.

The sample size in this study was determined to be 22 patients in each group, using the results of a similar study (11) and the STATA software, with a confidence level of 95%.

Estimated sample size for two-sample comparison of means

Test Ho: m1 = m2, where m1 is the mean in population 1 and m2 is the mean in population 2.

Assumptions:
alpha = 0.0500 (two-sided)
power = 0.9000
m1 = 14.3
m2 = 11.9
sd1 = 2.9
sd2 = 1.9
n2/n1 = 1.00
Estimated required sample sizes:
n1 = 22
n2 = 22

Inclusion criteria

Children aged between 2 and 15 years with PAH for the case group and without PAH for the control group were included. Control group children were selected among children referred to Amirkabir hospital who had CHD but were healthy in terms of PAH; the control group children were matched with the case group in terms of age and gender.

Exclusion criteria

The study excluded children with long-term respiratory conditions, sudden heart failure, ongoing liver or kidney issues, platelet disorders, and those taking antiplatelet or anticoagulant medications.

All patients in our study (case and control) were non-cyanotic.

The inventory of variables examined in this research encompasses measurements of weight, height, oxygen saturation (O_2 saturation), respiratory rate (RR), heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), PDW, MPV, platelet count, hematocrit (HCT), hemoglobin (Hb), and plateletcrit (PCT).

Complete blood count

Using the direct current detection method, blood count analysis was conducted using an automated hematology analyzer to measure various blood count parameters, such as platelet count and MPV. Individual blood film scanning was also conducted to eliminate any potential analytical errors. PCT was determined by multiplying the MPV and platelet count to evaluate the functional platelet mass.

Conventional echocardiographic measures of right ventricular dimensions

All patients enrolled in the study underwent transthoracic echocardiography using an ultrasound system (MyLab™ Seven). The simplified Bernoulli equation was used to calculate the pressure gradient across the tricuspid valve. This was then combined with the right atrial pressure (RAP) to determine the pulmonary artery systolic pressure (PASP), which is equal to the right ventricular systolic pressure (RVSP). pulmonary hypertension (PH) was diagnosed if the RVSP was greater than 25 mm Hg . Additionally, echocardiographic assessments included the evaluation of right ventricular systolic diameter (RVSD), right ventricular diastolic diameter (RVDD), and PAP. PH was diagnosed when the mean PAP at rest exceeded 25 mm Hg .

Statistical analysis

The data were analyzed using SPSS for Windows, version 26.0. Descriptive statistics, such as numbers and percentages, were used to describe qualitative data. The qualitative data comparison between the two groups was conducted using the χ^2 test. The Kolmogorov–Smirnov test was utilized to evaluate the normality of the data distribution. Mean and standard deviation (SD) were used to present quantitative data, and comparisons were made using one-way analysis of variance (ANOVA). Using the Pearson correlation test, the study examined the correlation between platelet markers and clinical data.

Furthermore, the predictive value of platelet (PLT) and PCT in forecasting pediatric conditions at various cut-off levels was assessed using receiver operating characteristic (ROC) curve analysis. The significance level was set at 0.05.

Results

Forty-four cases were evaluated: 22 cases with PAH as the case group and 22 without PAH as the control group.

Age, gender, and anthropometric information of participants

The mean and SD of age in cases with and without PAH were 7.75 ± 3.64 and 5.97 ± 3.71, respectively; the male/female ratio in the two groups was equal to 10:12. Also, anthropometric data, including height (P=0.199) and weight (P=0.911), did not show a statistically significant difference between the two groups. In addition, DBP (P=0.301), SBP (P=0.888), RR (P=0.301), and HR (P=0.065) did not show a statistically significant difference. However, the O_2 saturation in PAH cases was significantly lower than in cases with normal tension (P=0.0001) (Table 1).

The following information presents the number of patients categorized by their respective heart defects:

In the case group:
Ventricular septal defect: 10
Large patent ductus arteriosus: 5
Large atrial septal defect: 2
Pulmonary stenosis: 2
Aortic stenosis: 2
Coarctation of the aorta: 1

In the control group:
Ventricular septal defect: 8
Large patent ductus arteriosus: 5
Large atrial septal defect: 4
Pulmonary stenosis: 2
Aortic stenosis: 2
Coarctation of aorta: 1

Table 1. Age, gender, anthropometric, and clinical data of participants

Variables	Groups		*P* value
	PAH	Non-PAH	
Age (y)	7.75 ± 3.64	5.97 ± 3.71	0.055 *
Gender (male/female)	10/12	10/12	1.0 **
Height (cm)	110.18 ± 18.77	101.18 ± 26.37	0.199 *
Weight (kg)	26.97 ± 11.90	27.04 ± 23.94	0.911 *
SBP	88.63 ± 15.90	87.95 ± 16.10	0.888 *
DBP	57.95 ± 14.11	53.40 ± 14.69	0.301 *
RR	17.50 ± 1.79	17.54 ± 1.71	0.932*
HR	94.36 ± 15.18	102.63 ± 13.68	0.065*
O_2 saturation	92.81 ± 0.90	93.86 ± 1.42	0.006 *

* Independent samples *t*-test.
** Chi-square.

Mean and SD of PAP, RVSD and RVDD

The mean and SD of PAP in cases with and without PAH were 36.81 ± 6.08 and 19.80 ± 2.78, respectively. Also, the RVSD and RVDD did not show a statistically significant difference (Table 2).

Hemoglobin, HCT, platelet count, and platelet function tests in two groups

HB (P=0.146), HCT (P=0.712), and platelet count did not show statistically significant differences. However, MPV, PDW, and PCT in PAH cases were significantly higher than in cases with normal pulmonary tension (P=0.0001) (Table 3).

ROC curve and related analysis of data with a significant difference

Oxygen saturation below 92.5 mm Hg had a sensitivity of 54% and specificity of 77% in predicting PH in children with CHD. Meanwhile, PDW above 8.9 fL had a sensitivity of 90% and specificity of 96%. Procalcitonin levels above 43% had a sensitivity of 72% and specificity of 87%, and MPV above 9.25 had a sensitivity of 95% and specificity of 94% (Figure 1).

Discussion

The results of the present study revealed a correlation between MPV, PCT, PDW, and PAH. Pediatric patients with PAH demonstrated notably increased levels of MPV, PCT, and PDW compared to those without PAH. The research unequivocally suggested that elevated MPV, PCT, and PDW levels could serve as valuable diagnostic markers for identifying PAH associated with CHD in pediatric patients with left-to-right shunt.

Furthermore, factors associated with platelet count

Table 2. PAP, RVSD, and RVDD in the two groups

Variables	Groups		*P* value *
	PAH	Non-PAH	
PAP	36.81 ± 6.08	19.80 ± 2.78	-
RVSD	1.52 ± 0.30	1.45 ± 0.27	0.416
RVDD	2.03 ± 0.28	1.92 ± 0.33	0.248

* Independent samples *t*-test.

Table 3. Hemoglobin, hematocrit, platelet count, and platelet function tests

Variables	Groups		*P* value *
	PAH	Non-PAH	
Hb	11.90 ± 1.00	12.38 ± 1.10	0.146
HCT	35.72 ± 3.36	34.93 ± 9.42	0.712
MPV	9.65 ± 1.08	8.29 ± 0.73	0.0001
PDW	11.27 ± 1.93	8.00 ± 0.82	0.0001
PCT	52.77 ± 12.65	35.00 ± 11.68	0.0001
PLT	317.27 ± 87.73	287.72 ± 93.05	0.285

* Independent samples *t*-test.

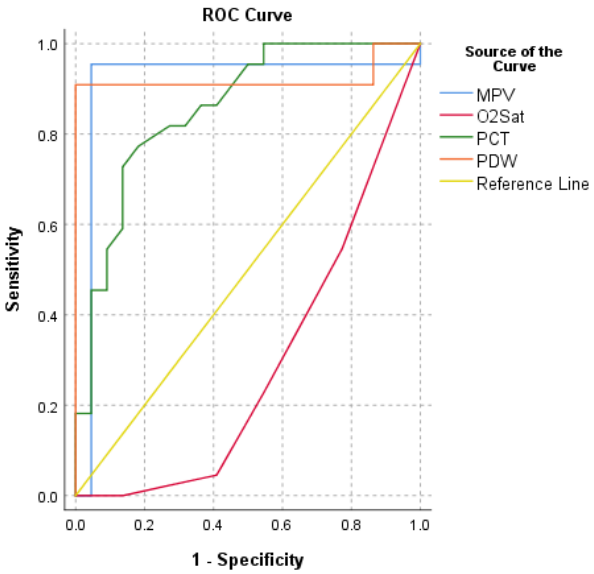

Figure 1. Roc curve of MPV, PDW, PCT, and O_2 saturation in CHD diagnosis

and function play a predominant role in the processes of hemostasis and thrombosis. Additionally, a decrease in O_2 saturation could be considered a factor in identifying PAH. In this regard, the accuracy of these tests is as follows: An O_2 saturation level below 92.5 mm Hg demonstrated a sensitivity of 54% and specificity of 77% in predicting PH in children with CHD. Meanwhile, a PDW exceeding 8.9 FL showed a sensitivity of 90% and specificity of 96%. Additionally, a PCT level surpassing 43% exhibited a sensitivity of 72% and specificity of 87% in the prediction of PH in this population. MPV at a cut-off greater than 9.25 exhibited a sensitivity of 95% and specificity of 94%. MPV is a simple factor for evaluating platelet function and reflects the platelet production and stimulation rate.

In patients with this condition, in situ thrombosis in pulmonary vessels is a notable observation. This occurrence may stem from endothelial injury, abnormal fibrinolysis, heightened procoagulant activity, and platelet irregularities (15).

MPV has been demonstrated to have an inverse relationship with the overall platelet count, potentially indicating smaller platelets and the subsequent generation of larger reticulated platelets as a compensatory mechanism (16). In our investigation, a trend of decreasing platelet counts was observed from the control group to the patients with PAH. Nevertheless, this difference did not achieve statistical significance.

Varol et al (14) demonstrated a notable increase in MPV among patients with PAH compared to control subjects. These findings suggest heightened MPV, a marker of platelet activation, in individuals with PAH.

MPV and PDW are important indicators that reflect platelet production and may be indirectly related to platelet function and activity (17). Elevated levels of MPV, PCT, and platelet PDW have been associated with metabolic syndromes such as acute ischemic stroke, myocardial infarction, diabetes, metabolic syndrome, obesity, hypertension, and other related conditions (17,18).

In a recent study conducted by Varol et al (14), MPV was found to be significantly elevated in adult patients with PAH compared to the control group. Furthermore, Can et al (19) demonstrated a statistically significant elevation in MPV among individuals with idiopathic PAH compared to control subjects. The findings of the two studies were consistent with ours, as we too noted elevated levels of MPV, PCT, and PDW in individuals with PAH.

In some research investigations, platelet function tests were assessed in the context of rheumatologic diseases, revealing a decrease in MPV in cases of active rheumatologic conditions such as ankylosing spondylitis and rheumatoid arthritis (20). In research conducted by Liu et al (21), a notable decrease in PDW and MPV was observed in pediatric patients with Kawasaki disease. The underlying causes for this reduction remain unidentified. However, in the present study, an increase in the levels of MPV and PDW was noted in cases with PAH.

In our study, which was comparable to Varol and colleagues' and Can and colleagues' studies (14,19), we observed enhancement in MPV, PCT, and PDW. Based on our findings and the findings of other studies in this field, MPV, PCT, and PDW increase in pediatric cases with PAH. We also noted a reduction in O_2 saturation in PAH cases.

The findings of the present study indicated that children diagnosed with PAH exhibit decreased levels of PDW and MPV. Furthermore, a relationship between PDW and MPV was noted in children with PAH. The study suggests that the increased platelet-derived growth factor gene expression in patients with CHD and PAH may contribute to the reduced PDW and MPV values. These results establish an association between PDW, MPV, and PAH, with notably lower PDW and MPV values observed in PAH patients than those without PAH. The study emphasizes the potential diagnostic significance of lower PDW and MPV values in assessing PAH secondary to CHD in patients with left-to-right shunt. Platelets play a critical role in thrombosis and hemostasis (13).

We have demonstrated a significant elevation in MPV levels in patients diagnosed with PAH compared to those in the control group. These findings suggest the presence of heightened MPV, a marker of platelet activity, in individuals with PAH. There is a dearth of research on the impact of elevated MPV on the prognosis of PAH patients. The increased MPV values may also indicate a potential heightened risk of systemic thromboembolism in PAH patients with elevated MPV due to enhanced platelet activation. Further randomized studies with larger sample sizes are imperative to determine whether MPV can effectively identify patients with PAH who are at an increased clinical risk and whether therapeutic

interventions targeting this marker may lead to amelioration in the clinical trajectory of the disease.

In conclusion, our research, which was the first study conducted in this field, showed a significant increase in MPV, PCT, and PDW in pediatric cases with PAH. The detection of PAH in pediatric cases with CHD is challenging. However, it may be supported by increased MPV, PCT, and PDW. Hematology markers are simple indices that do not need expensive or advanced technology and methods. In addition, prospective research is necessary to evaluate the clinical significance and pathophysiology of enhanced PDW, PCT, and MPV in pediatric cases with PAH. In addition, we showed that O_2 saturation was lower in children with PAH.

This study was conducted in a single center, which may be considered a limitation due to the small sample size. Moreover, it is not possible to arrive at conclusive decisions concerning the intake of iron supplements.

Conclusion

In summary, our research shows a notable elevation in MPV and PDW in pediatric patients with PAH. Furthermore, we have identified a correlation between PDW and MPV in this patient population. Diagnosing PAH presents challenges, but elevated PDW and MPV may offer supportive evidence. These hematological indices serve as simple markers that do not require advanced or expensive technology. Additionally, our research results showed that assessing hematological indicators (such as MPV and PDW) in children can serve as a significant factor in diagnosing cardiovascular diseases, particularly PAH.

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Authors' Contribution

Conceptualization: Mehran Akbari, Masoud Bahrami, Yazdan Ghandi.
Data curation: Mehran Akbari, Masoud Bahrami, Yazdan Ghandi.
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Investigation: Mehran Akbari, Masoud Bahrami, Yazdan Ghandi.
Methodology: Mehran Akbari, Masoud Bahrami, Yazdan Ghandi.
Project administration: Yazdan Ghandi.
Resources: Mehran Akbari, Masoud Bahrami, Yazdan Ghandi.
Software: Masoud Bahrami.
Supervision: Yazdan Ghandi.
Validation: Mehran Akbari, Yazdan Ghandi.
Visualization: Yazdan Ghandi.
Writing–original draft: Mehran Akbari, Masoud Bahrami, Yazdan Ghandi.

Competing Interests

The authors declared no conflict of interest.

Ethical Approval

The Ethics Committee of Arak University of Medical Sciences approved this study, and the study was performed following the approved guidelines (Ethical number: IR.ARAKMU.REC.1399.233). Written informed consent was acquired from the parents of all participants during the initial data collection.

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