Assessment of bronchial asthma exacerbation: the utility of platelet indices

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Background Activated platelets and platelet indices have a vital role in bronchial hyper-responsiveness, bronchoconstriction, bronchial inflammation, airway remodeling, angiogenesis, allergic reactions, and repair and renewal of tissues; platelets contain mediators that lead to inflammatory response.

Aim The aim was to assess the use of platelet indices [mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet large cell ratio (PLCR)] as cheap and readily available biomarkers for bronchial asthma exacerbation.

Patients and methods A case–control study involved 45 bronchial asthma female patients during both stable and exacerbation phases, and 45 age-matched healthy female patients as a control group. Measurements of platelet counts, MPV, PDW, PCT, PLCR, C-reactive protein (CRP), spirometric indices, and arterial blood gases were performed for all participants.

Results The MPV and PDW were significantly lower, whereas the PCT and PLCR were considerably higher in exacerbation phase compared with stable phase and in stable phase in comparison with controls ($P<0.001$). The MPV and PDW were negatively correlated with white blood cells, $PaCO_2$, symptoms duration, and hs-CRP (high sensitive), with positive correlation with forced expiratory volume in the first second and PaO_2 ($P<0.001$). PCT and PLCR were positively correlated with white blood cells, PaO_2, and symptoms duration, and negatively correlated with forced expiratory volume in the first second, symptoms duration, and hs-CRP ($P<0.001$).

Conclusion The platelet indices were altered in exacerbation phase compared with stable phase and control group. Therefore, clinicians should not ignore interpreting platelet indices during asthma exacerbation, especially as these tests are simple, readily available, and of lower cost. It appears that measurement of the platelet indices is a valuable indicator of asthma severity/activity and appears as a useful screening test for asthma exacerbation.

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Introduction

Bronchial asthma exacerbations are episodes described by progressive increase in symptoms of cough, wheezing, shortness of breath, and/or chest tightness, with a progressive decrease in lung function [1]. Few tests are being used for diagnosis of asthma, but at present, no established biomarker is available that may be used for diagnosis and prognosis of asthma [2]. Platelet indices and C-reactive protein (CRP) are markers that reflect a systemic inflammatory response [3]. Activated platelets play a crucial role in bronchial hyper-responsiveness, bronchoconstriction, bronchial inflammation, and airway remodeling. Plasma β-thromboglobulin and platelet factor-4, common markers of platelet activation *in vivo*, have been reported to be elevated in patients with symptomatic asthma [4]. Many biomarkers, such as GP IIb/IIIa, PF4, CD62, CD63, and thromboglobulin, may be used as markers of platelet activation [5]. However, these tests are not routinely performed owing to high costs and necessities for specialized equipment. Platelet indices, which include the mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet large cell ratio (PLCR), have been available in the laboratory routine using blood cell counters for several years; they are related to platelets' morphology and proliferation kinetics [6]. Moreover, their measurement is inexpensive, effective, and easy, and is proposed to be a beneficial method for assessment of platelet function and activation [7]. Because of all of these advantages, the main aim of this study was to investigate the potential of routine use of MPV, PDW, PCT, and PLCR as reliable diagnostic biomarkers of bronchial asthma exacerbation.

Patients and methods

Type and place of the study

This observation case–control study was conducted at Chest Diseases Department of our hospital during the period from October 2016 to January 2018.

Inclusion criteria

The study was conducted on 45 known bronchial asthma female patients during both exacerbation and stable phase of their disease, in addition to 45 age-matched healthy women as a control group.

(1) Bronchial asthma patient group: it included 45 patients who were admitted to our department with symptoms of asthma exacerbation. Asthma exacerbation was defined as the existence of any one of the following events: emergency visits related to asthmatic attack (asthmatic attack-related hospitalization), or the use of systematic corticosteroids for at least 3 days. Inhaled short-acting and long-acting β2-agonist, inhaled steroids, oral steroids, and antibiotics were ordered to treat asthma exacerbation. After 8 weeks following the attack, the patients were re-evaluated when they were symptom free. Stable asthma was defined as no exacerbation of symptoms in the past 8 weeks [1].
(2) Controls: apparently healthy age-matched, nonsmoking women were included as a control group. They had no symptoms suggestive of any chest illnesses, and their spirometry and arterial blood gases (ABG) were in the normal range.

Exclusion criteria

As smoking and inflammatory, infectious, and allergic diseases tend to cause an increase in platelet production by stimulating the bone marrow, therefore, smokers, as well as patients with the following diseases were excluded from the study: diabetes mellitus, hypertension, malignancies, hematological disorder, valvular lesions, coronaries disease, heart failure, autoimmune diseases, liver cell failure, renal failure, those with other chest diseases. Moreover, to avoid possible effect of certain medications on platelet function, patients treated with an anticoagulant, statins, angiotensin-converting enzyme, acetylsalicylic acid, and clopidogrel were excluded from the study.

All participants were subjected to the following:

The BMI was measured as weight (kg)/height (m^2).

(1) Spirometry: it was carried out on MEDISOFT–HYPERAIR compact+flow meter pulmonary function testing (Medisoft Belgium (Headquarter), P.A.E de Sorinnes, 1 Route de la Voie Cuivrée, 5503 Sorinnes, Belgium). It was performed before and after the inhalation of short-acting B2-agonist. The following spirometric indices were recorded: forced vital capacity (FVC%), forced expiratory volume in the first second (FEV_1%), FEV_1\FVC ratio, and forced expiratory flow rate of 25–75 (FEF 25–75%). A postbronchodilator spirometry was done 10–15 min following the inhalation of 400 μg Salbutamol. A rise in FEV_1 greater than 200 ml and/or 12% above the prebronchodilator FEV_1 at time of diagnosis was considered diagnostic [8]. Positive result of reversibility with a bronchodilator is recorded [8]. Spirometric indices were calculated as the best of three technically acceptable performances, in agreement with the ERS recommendations [9].
(2) ABG: it was performed following a 15-min resting period in room air using a Rapid Lab 248 blood (Siemens Medical Solutions, Malvern, Pennsylvania, USA) gases analyzer; O_2 saturation, PaO_2, and $PaCO_2$ were recorded.
(3) Complete blood count: venous samples were collected from participants in the morning between 8.00 and 9.00 a.m. after an 8 h overnight fasting. Venous blood samples were drawn from cubital vein and immediately placed in EDTA-containing tubes (Becton Dickinson Vacuum, Becton, Dickinson and Company, Franklin Lakes, New Jersey, USA) and mixed gently. White blood cells (WBC), platelet count, MPV (fl) and PDW (fl), PCT, and PLCR% were measured within 1–2 h of blood sampling using a hematological analyzer (Sysmex XE-21N, Kobe, Japan). The analyzer was calibrated daily using a standardized, commercially available calibrator kit. Blood samples were placed in standard tubes containing EDTA and analyzed within 1 h after venepuncture. Thus, we had standard EDTA tubes and time window for the analysis [10,11].
(4) High-sensitivity CRP assay: to detect the hs-CRP level, 3 ml of venous blood sample was taken into standard biochemical tubes and centrifuged for 20 min. hs-CRP assay is based on the principle of solid-phase enzyme-linked immunosorbent assay. Expected values were based on published literature, and healthy adult individuals are expected to be 68–8200 ng/ml.

Ethical approval

The study was performed after the Ethical Review Committee approval. Participation was voluntary; an informed oral consent was attained individually from each study participant before enrolment into study.

Statistical analysis of data

Statistics were analyzed by the Statistical Package for the Social Sciences (SPSS) program version 17.0

Table 1 Comparison of spirometric indices and arterial blood gases parameters during exacerbation phase, stable phase, and control group

ABG	Bronchial asthma group (mean±SD)		Control group (mean±SD)	ANOVA test		Post-hoc analysis		
	Exacerbation phase	Stable phase		*F*	*P*	P_1	P_2	P_3
FEV_1/FVC	69.9±4.7	77.8±4.6	87.1±4.0	163.9	0.001*	0.002*	0.001*	0.001*
$FEV_1\%$	53.5±16.0	63.3±14.6	83.7±2.7	67.3	0.001*	0.002*	0.002*	0.001*
FVC%	81.6±3.8	85.4±2.8	85.2±4.2	15.1	0.002*	0.002*	0.85	0.002*
VC%	87.2±2.3	88.6± 3.1	89.6±4.1	6.01	0.003*	0.003*	0.15	0.045
$FEF_{25-75}\%$	59.1±6.4	62.7±4.9	68.8±3.7	40.6	0.002*	0.002*	0.001*	0.002*
O_2 saturation	95.0±2.3	96.0±1.7	96.2±1.3	4.8	0.009*	0.005*	0.70	0.014*
PaO_2	82.5±6.0	84.9±3.6	89.4±3.4	26.9	0.001*	0.001*	0.001*	0.014*
$PaCO_2$	40.6±3.3	38.8±2.2	38.9±2.0	7.0	0.001*	0.002*	0.89	0.001*

ABG, arterial blood gas; ANOVA, analysis of variance; FEF, forced expiratory flow rate; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; P_1, exacerbation phase vs controls; P_2, stable phase vs controls; P_3, exacerbation phase vs stable phase.
*Significant test.

Table 2 Comparison of platelet count, platelet indices, white blood cells, and high-sensitivity C-reactive protein during exacerbation phase, stable phase, and control group

Items	Bronchial asthma group (mean±SD)		Control group (mean±SD)	Test value•	*P* value	Post-hoc analysis		
	Exacerbation phase	Stable phase				P_1	P_2	P_3
WBC	9.1±1.7	7.9±1.1	7.5±1.0	17.6	0.001*	0.001*	0.13	0.001*
Platelet count	366.8±42.5	360.24±37.5	363.5± 57.0	0.23	0.795	0.736	0.73	0.499
MPV	8.39±1.1	10.86±1.1	11.1±1.5	61.8	0.001*	0.001*	0.25	0.001*
PDW	11.3±1.7	11.94±1.3	12.1± 1.2	3.9	0.021*	0.007*	0.449	0.050
PCT	0.25±0.03	0.25±0.02	0.23±0.01	4.1	0.019*	0.011*	0.020*	0.819
PLCR	33.3±7.4	30.5±3.3	25.3± 5.4	23.1	0.001*	0.001*	0.001*	0.023*
hs-CRP ng	12030.44±5736.2	10012.4±4328.2	8086.6±1926.8	9.4	0.001*	0.001*	0.035*	0.028*

hs-CRP, high-sensitivity C-reactive protein; MPV, mean platelet volume (fl); P_1, exacerbation phase vs controls; P_2, stable phase vs controls; P_3, exacerbation phase vs stable phase; PCT, platelet crates; PDW, platelet distribution width (%); PLCR, platelet large cell ratio.
*Significant test.

(SPSS Inc., Chicago, Illinois, USA). Descriptive analysis was performed for each item and the results were stated as mean±SD for quantitative continuous variables, and as percentages for qualitative (categorical and nominal) variables. Comparisons for assessing the difference between the groups were done using the χ^2-test for qualitative data and the analysis of variance test and Student's *t*-test for quantitative data. A linear correlation coefficient was done to detect the correlation between two quantitative variables in the same group. Statistical significance was considered by a *P* value less than 0.05 (with a confidence limit at 95%).

Results

Asthmatic patients and controls were matched regarding age (40.0±15.2 and 41.2±15.1, respectively) and BMI (27.2±3.3 and 26.3±3.6, respectively) ($P>0.05$). Table 1 shows that the FEV_1/FVC, FEV_1, $FEF_{25-75}\%$, O_2 saturation, and PaO_2 were significantly declined in exacerbation phase compared with both stable phase and control group, and in stable phase compared with control group ($P<0.05$). Additionally, FVC% was significantly decreased in exacerbation phase compared with both stable phase and control group. $PaCO_2$ was significantly higher in exacerbation phase in comparison with both stable phase and control group ($P<0.01$). Table 2 shows that there was significant decline in MPV and PDW in exacerbation phase in comparison with stable phase. The MPV, WBC, and PDW were nonsignificantly different between stable phase and control group. Moreover, there is a significant increase in WBC, hs-CRP (ng/ml), PCT, and PLCR in exacerbation phase compared with stable phase. There was no significant change in platelet count in control group compared with both stable phase and exacerbation phase. Table 3 demonstrates that during exacerbation phase and stable phase, both MPV and PDW were positively correlated with FEV_1/FVC, $FEV_1\%$, FVC%, $FEF_{25-75}\%$, O_2 saturation, and PaO_2, whereas it was negatively correlated with hs-CRP, PCT, PLCR, symptoms duration, $PaCO_2$, WBC, and platelet count ($P<0.01$). Moreover, they were negatively correlated with symptom duration during exacerbation phase and positively correlated with each other ($P<0.01$). Table 4 shows that the PCT and PLCR were negatively correlated with FEV_1/FVC, FEV_1, FVC, VC, FEF_{25-75}, O_2

Table 3 Correlation of mean platelet volume and platelet distribution width with other studied variables during exacerbation phase and stable phase

Items	MPV				PDW			
	Exacerbation phase		Stable phase		Exacerbation phase		Stable phase	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
PDW	0.84*	0.002	0.98*	0.001	–	–	–	–
PCT	–0.82*	0.002	–0.97*	0.001	–0.97*	0.001	–0.98*	0.001
PLCR	–0.84*	0.002	–0.97*	0.001	–0.99*	0.001	–0.99*	0.001
Platelet count	–0.84*	0.002	–0.97*	0.001	–0.99*	0.001	–0.99*	0.001
WBC	–0.84*	0.002	–0.97*	0.001	–0.99*	0.001	–0.99*	0.001
hs-CRP ng	–0.84*	0.002	–0.95*	0.001	–0.94*	0.001	–0.85*	0.002
Symptoms duration /day	–0.84*	0.002	–	–	–0.94*	0.001	–	–
$FEV_1/FVC_{\%}$	0.68*	0.001	0.84*	0.002	0.84*	0.001	0.87*	0.001
$FEV_1\%$	0.84*	0.001	0.94*	0.001	0.99*	0.001	0.96*	0.001
FVC%	0.80*	0.002	0.85*	0.002	0.95*	0.001	0.86*	0.001
VC%	0.68*	0.002	0.57*	0.003	0.82*	0.001	0.59*	0.002
$FEF_{25-75}\%$	0.84*	0.002	0.95*	0.001	0.99*	0.001	0.97*	0.001
O_2 saturation	0.81*	0.002	0.92*	0.001	0.96*	0.001	0.94*	0.001
PaO_2	0.84*	0.001	0.97*	0.001	0.99*	0.001	0.98*	0.001
$PaCO_2$	–0.84*	0.001	–0.94*	0.001	–0.98*	0.001	–0.96*	0.001

FEF, forced expiratory flow rate; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; hs-CRP, high-sensitivity C-reactive protein; MPV, mean platelet volume (fl); PCT, platelet crates; PDW, platelet distribution width (%); PLCR, platelet large cell ratio; WBC, white blood cells. *Significant test.

Table 4 Correlation of platelet crates and platelet large cell ratio with other studied variables during exacerbation phase and stable phase

Items	PCT				PLCR			
	Exacerbation phase		Stable phase		Exacerbation phase		Stable phase	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
PLCR	0.98*	0.001	0.98*	0.001	–	–	–	–
Platelet count	0.98*	0.001	0.98*	0.001	0.97*	0.001	0.92*	0.001
WBC	0.97*	0.001	0.98*	0.001	0.99*	0.001	0.93*	0.001
hs-CRP	0.97*	0.001	0.95*	0.001	0.83*	0.002	0.84*	0.002
Symptoms duration/day	0.84*	0.002	0.85*	0.002	0.92*	0.001	0.94*	0.001
FEV_1/FVC	–0.85*	0.001	–0.87*	0.001	–0.81*	0.002	–0.86*	0.002
$FEV_1\%$	–0.97*	0.001	–0.95*	0.001	–0.96*	0.001	–0.93*	0.001
FVC%	–0.95*	0.001	–0.85*	0.001	–0.93*	0.001	–0.84*	0.002
VC%	–0.81*	0.001	–0.57*	0.002	–0.83*	0.001	–0.58*	0.003
$FEF_{25-75}\%$	–0.97*	0.001	–0.95*	0.001	–0.94*	0.001	–0.95*	0.001
O_2 saturation	–0.95*	0.001	–0.93*	0.001	–0.95*	0.001	–0.93*	0.001
PaO_2	–0.98*	0.001	–0.97*	0.001	–0.97*	0.001	–0.93*	0.001
$PaCO_2$	0.97*	0.001	0.95*	0.001	0.93*	0.001	0.94*	0.001

FEF, forced expiratory flow rate; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; hs-CRP, high-sensitivity C-reactive protein; MPV, mean platelet volume (fl); PCT, platelet crates; PDW, platelet distribution width (%); PLCR, platelet large cell ratio; WBC, white blood cells. *Significant test.

saturation, and PaO_2, whereas both were positively correlated with $PaCO_2$, WBC, platelet count, hs-CRP, and PLCR during exacerbation phase and stable phase. Moreover, they were positively correlated with each other.

Discussion

As previously published studies have documented age [12], sex [13], obesity [14], smoking [15], certain diseases [16], and drugs [17] as confounders affecting platelet indices, we selected our participant as females, nonsmokers, in the same age group, having approximately the same BMI, and without co-morbidities that could affect platelet function.

An important finding of our study is that platelet count was nonsignificantly different between asthmatic patients during either exacerbation or stable phase and controls. However, the MPV and PDW were

negatively correlated, whereas PCT and PLCR were positively correlated with platelet count during both exacerbation and stable phase. The results of the previous studies about platelet count in asthmatics are inconsistent. Our finding was in agreement many others published studies, where there was no variance in platelet counts between asthmatic patients and matched control group [18–25]. However, another investigator reported significantly increased platelet count in exacerbated-asthmatic than stable-asthmatics [19]. Same variability in platelet count was found among asthmatic children, as Nacaroglu *et al.* [7] reported that platelet count was significantly increased in the patient group either in exacerbation phase or asymptomatic periods compared with controls. Dogru *et al.* [26] reported that the platelet count was significantly higher in both stable asthmatic and exacerbated asthmatic patients compared with controls, whereas there was no significant difference between stable asthmatic and exacerbated asthmatic patients. In the study by Tuncel *et al.* [24], platelet count in asthmatic children, either during an exacerbation or asymptomatic period, was nonsignificantly higher than control group. These differences in result regarding platelets count between children and adult may be owing to the immaturity of immune system among children.

The main results of this study are that the MPV and PDW were significantly lower, whereas PCT and PLCR were significantly higher during exacerbation phase compared with stable phase and controls ($P<0.001$). These findings indicate that platelet indices are more sensitive biomarkers for asthma exacerbation than platelets count. The alteration of platelet indices indicates increased systemic inflammation during exacerbation; therefore, they could be used as acute-phase reactants for asthma exacerbation. Moreover, the changes of platelet indices in stable phase compared with controls indicate that even in stable phase, the chronic underlying inflammatory process tends to affect platelet indices. The release of small volume platelets by cytokines induces stimulation of megakaryocytes in bone marrow and/or consumption of large volume/active platelets during inflammation have been proposed to be the possible reasons for this finding [27]. These findings are consistent with that reported by Sun *et al.* [18], Ellauriea and Wangb [28], and Yavuz *et al.* [29], as the MPV was reduced in asthmatics in exacerbation and in stable asthmatics in comparison with controls. Additionally, the MPV levels of stable asthmatics were higher than exacerbated asthmatics. Ibrahim *et al.* [22] found that MPV level was significantly lower in asthmatic patients in comparison with non-smoker and smoker healthy controls. However, the PDW levels were significantly higher in asthmatic patients compared with nonsmoker and smoker healthy controls. Not only the inflammation but also its surface area and type of inflammation affects the MPV value, as Akgedik and Yağız [23] reported that the MPV was found to be correlated with the inflamed surface area as the lowest MPV value was detected in bronchial asthma and allergic rhinitis, the moderate MPV was detected in asthma group, and the highest MPV was seen in the allergic rhinitis group, though the variance among the three groups was nonsignificant. However, the PDW was nonsignificantly differed among the three allergic groups and the controls. Additionally, Kara *et al.* [20] and Kin *et al.* [30] found that the percentage of eosinophils, total immunoglobulin E levels, and PDW were significantly higher in atopic asthma group. Different results were reported by other investigators who demonstrated that the neither MPV nor PDW differed between asthmatic patients and controls, or between exacerbated-asthmatics and stable-asthmatics [19,21,24,31,32]. Kepekçi *et al.* [33] found a weak, positive, directional, and meaningful relationship between PDW and MPV variables in patients with nasal polyposis with or without asthma. These controversies between studies regarding the value of platelet indices in bronchial asthma may be owing to different population under concern, different patient age, different attack severity, and different measurement methods.

We found that both MPV and PDW were positively correlated, and PCT and PLCR were negatively correlated with spirometric indices during both exacerbation and stable phases. Therefore, we can suggest that not only the presence of inflammation but also its severity may be a determinant of the platelet indices in asthmatic patients. Therefore, platelet indices may be used to determine exacerbation severity. Similarly, Kara *et al.* [20] found that in the attack, only the group with severe attack had a negative correlation between platelet count and FEV_1, and after the attack, PDW was positively correlated with FEV_1, FVC, FVC, and FEF_{25-75}%. However, Tuncel *et al.* [24] reported that MPV was not correlated with severity of attacks.

ABG analysis is an important parameter in asthma exacerbation and provides the best clues to acuteness and severity of disease and determines the need of ventilator support [15]. Both MPV and PDW had positive correlation with O_2 saturation and PaO_2 and

negatively correlated with $PaCO_2$ ($P<0.001$) during both exacerbation and stable phases. However, both PCT and PLCR were negatively correlated with O_2 saturation and PaO_2 and positively correlated with $PaCO_2$ ($P<0.001$) during both exacerbation and stable phases. The relationship between platelet indices and PaO_2 and $PaCO_2$ could be bidirectional; first direction is that the more alteration in platelet indices, that is, severe the inflammation, the more the deterioration in ABG parameters, and the second direction is that the hypoxemia or hypercapnia could induce platelet consumption with subsequent changes of its indices.

The present study showed that WBC was significantly higher in exacerbation phase in comparison with both stable phase and control group. Moreover, the MPV and PDW were negatively correlated, whereas the PCT and PLCR were positively correlated with WBC during exacerbation and stable phases. These findings indicate that there is a strong link between platelets indices and WBC in underlying inflammation of asthma, which confirm the previously mentioned theory that the processes of airway wall remodeling and leukocyte infiltration fail to occur without the participation of platelets in a murine model [31]. Moreover, increased circulating platelet-leukocyte aggregates have been identified in allergic asthmatics after an antigen challenge [34]. Platelets act as companions in the process of extravasation of leukocytes into bronchopulmonary airways from pulmonary microcirculation and in chemotaxis stimulation [31]. In accordance with our results, Uysal *et al.* [31] and Akgedik and Yağız [23] reported that leukocyte counts were significantly higher in exacerbated asthmatics than in stable asthmatics and controls. Kara *et al.* [20] reported that the WBC was significantly high in asthmatic children during the attack in comparison with those after attack.

Our study revealed that during exacerbation phase, the MPV and PDW were negatively correlated with symptoms duration and hs-CRP. The PCT and PLCR were positively correlated with symptoms duration and negatively correlated with hs-CRP. These findings indicate that during exacerbation, the more severe the underlying inflammation, that is, higher hs-CRP, higher WBC, higher PCT, and PLCR with lower MPV and PDW, the severer attacks, that is, the lower spirometric-indices and worsening ABG parameters. Similar results were reported in previous studies, as MPV was negatively correlated with CRP [18], WBC, and platelet count [23] in exacerbated asthma. Dogru *et al.* [26] described that the MPV had negative correlation with the WBC and platelets and was positively correlated with CRP during exacerbation. In asthmatic children, no correlation was found between MPV and CRP [24].

The negative correlation of hs-CRP with MPV and PDW and its positive correlation with PCT and PLCR during stable asthma phase in our study indicate that the underlying low-grade systemic inflammation in stable asthma has negative effects on platelet indices. Different results were reported by Sun *et al.* [18], as the reduced MPV is negatively correlated with CRP only in acute exacerbations and not in the stable phase. The study by Tuncel *et al.* [24] revealed that MPV was not correlated with CRP in asthmatic children. Our results were also in agreement with Shaaban *et al.* [35] in a population-based study, which suggested that increases in CRP concentration over time are associated with a significant decrease in pulmonary function, consistent with the hypothesis that low-grade systemic inflammation is associated with pulmonary damage.

The main strengths in the current study are as follows: first, the measurement of platelet-indices is a cost-effective test for assessment of asthma exacerbation, which can be applied easily to our patients with limited resources; second, the measurement of platelet counts and platelet indices was done in the same hospital with the same autoanalyzer using the same well-standardized method; and third, patients with other diseases or treated by drugs that could affect platelet function were excluded from the study. Therefore, our results reflect the actual effect of asthma exacerbation on platelet indices. However, this study had limitations that deserve to be mentioned, such as follows: the number of participants was relatively small. Therefore, we did not, classify patients according to exacerbation severity.

The MPV and PDW were found to be lower whereas PCT and PLCR were found to be higher during exacerbation phase compared with stable phase. Therefore, clinicians should not ignore interpreting platelet indices during asthma exacerbation, especially as the tests are simple, readily available, and low cost. It appears that measurement of the platelet-indices is a dependable indicator of asthma severity/activity and appears as a useful screening test for asthma exacerbation.

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Conflicts of interest

There are no conflicts of interest.

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