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ORIGINAL ARTICLE

Usefulness of admission hematologic parameters as diagnostic tools in acute pulmonary embolism

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Abstract The purpose of this study was to determine the role of red cell distribution width (RDW), neutrophil–lymphocyte ratio (NLR), and platelet–lymphocyte ratio (PLR) in the diagnostic phase of acute pulmonary embolism (PE). We screened 248 consecutive patients who were admitted to the emergency service with PE foremost in the differential diagnosis. Based on spiral computed chest tomography, the patients were divided into two groups. There were 112 confirmed cases of acute PE and 138 patients without PE. Blood samples were obtained within 2 hours of presentation and before starting any medication. There were no significant differences between the PE and the non-PE groups with respect to sex, age, frequency of disease, serum creatinine, sodium, and potassium ($p > 0.05$ for all). NLR, RDW, and PLR were higher in patients with PE than those without PE. High-sensitivity C-reactive protein, D-dimer, and troponin levels were also higher in patients with PE. RDW values were positively correlated with troponin levels ($r = 0.147$, $p = 0.021$). There were no correlations between RDW and NLR, PLR, or D-dimer. NLR had a highly positive correlation with PLR ($r = 0.488$, $p < 0.001$). In multivariate logistic regression analysis, troponin I, D-dimer, high-sensitivity C-reactive protein, and RDW were found to be independent predictors of PE [odds ratio (95% confidence interval) respectively: 5.208 (2.534–10.704), 1.242 (1.094–1.409), 1.005 (1.000–1.010), 1.175 (1.052–1.312)]. In receiver operating characteristic analysis of the patients in the study, RDW >18.9 predicted acute PE with a sensitivity of 20.7% and a specificity of 93.4%. In conclusion, RDW can be considered useful as a diagnostic measure for patients with suspected acute PE.

Conflicts of interest: All authors declare no conflicts of interest.

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Introduction

Acute pulmonary embolism (PE) is a life-threatening and common cardiopulmonary disease with a mortality rate of 15—20% [1]. It usually arises from a thrombus formed in the deep venous system of the lower extremities. It can occlude the pulmonary arterial bed and may lead to a chronic disabling condition, an acute life-threatening illness, or even death. As the clinical presentation for pulmonary embolism is often nonspecific and equivocal, clinical physicians need to pay much more attention to its diagnosis [2]. For this reason, it is prudent to pursue a quick and definitive diagnosis in every suspected case of PE. Because of its wide availability and ability to directly visualize thrombus, computed tomography imaging has become the standard imaging technique for the diagnosis of acute PE [3]. Routine laboratory testing is not useful in proving the presence of venous thrombosis or PE, but may contribute to the evaluation of the differential diagnosis. The diagnostic work ought to be as noninvasive and cost-effective as possible in order to minimize health system expenditures. The utility of plasma measurements of circulating D-dimer, a specific derivative of cross-linked fibrin, has been evaluated extensively as a diagnostic test in suspected PE. The recommended diagnostic pathway for outpatients with a low or intermediate probability of PE is to begin by performing a highly sensitive D-dimer assay [4,5]. Another marker, cardiac troponins, has been evaluated in the setting of acute PE and elevated troponin levels correlate with electrocardiographic and echocardiographic findings of right ventricular pressure overload. Elevated plasma troponin levels have been repeatedly reported as associated with worse prognosis and increased mortality in patients with PE [6,7]. Elevated brain natriuretic peptide has also been shown to be a marker of right ventricular pressure overload and one of the predictors of adverse outcome in patients with acute PE [8,9]. Complete blood count is part of the routine laboratory investigations in most hospitalized patients.

The red cell distribution width (RDW) is a measurement that is obtained by automated hematology analyzers. It reflects the range of the red cell sizes that are measured within a sample. RDW is strongly associated with prognosis in cardiopulmonary disorders such as coronary artery disease (CAD), acute myocardial infarction, acute and chronic heart failure, and pulmonary hypertension [10—15]. Recently it was shown that an elevated neutrophil—lymphocyte ratio (NLR) is related to early mortality in patients with PE [16]. An elevated platelet—lymphocyte ratio (PLR) as a risk factor for arterial obstructive diseases has been evaluated in some studies. It has been shown that elevated PLR is a significant independent predictor of long-term mortality after non-ST elevation myocardial infarction [17]. Gary et al demonstrated that a PLR $>$ 150 proved to be at least comparable to a NLR $>$ 3.95, an already established vascular risk factor, in its association with critical limb ischemia in patients with peripheral arterial occlusive disease [18]. However, the diagnostic value of PLR and NLR in acute PE is unknown. The goal of this study is to investigate the diagnostic value of RDW, NLR, and PLR, measured on admission, in patients suspected of having acute PE.

Materials and methods

This study was approved by the ethics review board of Mersin University School of Medicine. In this study, we retrospectively screened 248 consecutive patients over 18 years who were admitted to hospital with a suspect of PE between January 2011 and May 2013. Exclusion criteria were: active or chronic inflammatory or autoimmune diseases; inflammatory rheumatic disease; anemia; clinical evidence of active infection; active cancer; any hematological diseases; recent blood transfusion; chronic renal disease; and history of chronic obstructive pulmonary disease. According to their spiral computed chest tomography, the patients were divided into two groups: 112 of them had acute PE and 138 patients had no PE.

Biochemical analysis

Blood samples were obtained within 2 hours of presentation before starting any medication and were collected in tripotassium EDTA tubes. All measurements were performed 30 minutes after blood collection by an automatic blood counter (A Sysmex XE-2100; Symex, Kobe, Japan). D-Dimer was measured using Sysmex CA-7000 (Symex), troponin I was measured using Access 2 Immunoassay System (Beckman Coulter Inc., Brea, CA, USA), and high-sensitivity C-reactive protein (hsCRP) was measured using a BN2 model nephlometer.

Statistical analysis

Continuous variables are given as the mean $\pm$ standard deviation; categorical variables were defined as a percentage. A value of $p < 0.05$ was considered significant. Comparisons between groups were carried out using an independent-samples t test. Correlation analyses were performed using the Pearson or Spearman coefficient of correlation. Multivariate logistic regression analysis was used to show the independent predictors for PE. The cut-off levels, sensitivity and specificity values of RDW in PE patients were determined using MedCalc 12.7.0.1 (MedCalc Software, Mariakerke, Belgium). A trial version of SPSS 20.0 software was used for basic statistical analysis (Version 20; SPSS Inc., Chicago, IL, USA).

Results

The baseline characteristics of the study participants are summarized in Table 1. There were no significant differences between the PE and the non-PE groups with respect to sex, age, frequency of disease (i.e. diabetes mellitus, hypertension, smoking, and coronary artery disease), serum creatinine, sodium, and potassium ($p > 0.05$ for all). NLR values were higher in patients with PE than without PE (6.2 ± 2.9 in the PE group vs. 5.4 ± 3.0 in the non-PE group, $p = 0.03$). RDW and PLR values were also higher in patients in the PE group compared to the non-PE group (respectively: 15.9 ± 3.8 vs. 14.8 ± 2.2, $p = 0.005$ and 210 ± 131 vs. 178 ± 107, $p = 0.03$).

Table 1 The baseline clinical and biochemical characteristics of study participants.

	PE group (n = 111)	Non-PE group (n = 137)	p
Age (y)	59 ± 16	62 ± 15	0.1
Female (%)	55	54	0.4
Creatinine (mg/dL)	0.93 ± 0.5	0.93 ± 0.2	0.9
Na (mg/dL)	135 ± 4	136 ± 4	0.3
K (mg/dL)	3.7 ± 0.6	3.8 ± 0.7	0.3
AST (U/L)	36 ± 32	37 ± 32	0.7
ALT (U/L)	29 ± 26	32 ± 35	0.4
Hb (mg/dL)	13.3 ± 1.1	13.3 ± 1.2	0.7
RDW (%)	15.9 ± 3.8	14.8 ± 2.2	0.005
NLR	6.2 ± 2.9	5.4 ± 3.0	0.03
PLR	210 ± 131	178 ± 107	0.03
D-Dimer (ng/mL)	3.5 ± 3.0	1.9 ± 2.1	< 0.001
Troponin I (ng/mL)	1.4 ± 1.9	0.1 ± 0.3	< 0.001
HsCRP (mg/L)	76 ± 74	52 ± 64	0.007

A value of $p < 0.05$ was accepted as statistically significant.
ALT = alanine aminotransferase; AST = aspartate aminotransferase; Hb = hemoglobin; hsCRP = high-sensitivity C-reactive protein; NLR = neutrophil lymphocyte ratio; PE = pulmonary embolism; PLR = platelet lymphocyte ratio; RDW = red cell distribution width.

HsCRP, D-dimer, and troponin levels were also higher in patients with PE (Table 1). The correlation analysis is shown in Table 2. RDW values were positively correlated with troponin levels (r = 0.147, p = 0.021). NLR had a highly positive correlation with PLR (r = 0.488, $p < 0.001$). The troponin levels were positively correlated with D-dimer levels (r = 0.231, $p < 0.001$).

In multivariate logistic regression analysis, troponin I, D-dimer, hsCRP, and RDW were found to be independent predictors of PE (Table 3).

In receiver operating characteristic analysis of the patients in the study, RDW predicted acute PE with a sensitivity of 20.7% and a specificity of 93.4% over 18.9 (Fig. 1).

Table 3 Logistic regression analysis of risk factors predicting pulmonary embolism.

Risk factors	OR (95% CI)	p
Troponin I	5.208 (2.534–10.704)	<0.001
D-Dimer	1.242 (1.094–1.409)	0.001
hsCRP	1.005 (1.000–1.010)	0.037
NLR	0.887 (0.780–1.009)	0.068
PLR	0.998 (0.995–1.002)	0.340
RDW	1.175 (1.052–1.312)	0.004

$p < 0.05$ was accepted as statistically significant.
CI = confidence interval; hsCRP = high sensitive C-reactive protein; NLR = neutrophil lymphocyte ratio; OR = odds ratio; PLR = platelet lymphocyte ratio; RDW = red cell distribution width.

Discussion

In our study, we found that RDW, NLR, and PLR values in the PE group were higher than in the non-PE group. In multivariate logistic regression analysis, only RDW was found to be an independent predictor of PE among the hematological parameters. Troponin I, HsCRP, and D-dimer were also found to be independent predictors of PE. We are unaware of any studies that have looked at the diagnostic value of RDW in PE. To the best of our knowledge, this is the first study that specifically evaluates the role of RDW in PE diagnosis among patients admitted to the emergency service suspected of having PE.

RDW is a measure of variability in the size of circulating erythrocytes, with higher values reflecting greater heterogenity in cell sizes (anisocytosis), and it is part of a complete blood count. An increased RDW is usually associated with ineffective red cell production, such as occurs in iron deficiency anemia, vitamin B12, and folic acid deficiency, anemia of chronic disease, and hemoglobinopathies, or red blood cell destruction in the cases of hemolysis and blood transfusion [10]. Felker et al have shown that an elevated RDW is a strong independent predictor of outcome in patients with chronic heart failure (HF) [19]. Several studies

Table 2 The correlation analysis of hematologic parameters with high-sensitivity C-reactive protein (hsCRP), D-dimer, and troponin in all study patients.

		hsCRP	D-Dimer	Troponin I	RDW	PLR	NLR
hsCRP	Pearson correlation	1	0.117	0.092	−0.012	−0.057	0.057
	p		0.066	0.148	0.849	0.375	0.371
D-Dimer	Pearson correlation	0.117	1	0.231*	0.038	0.034	−0.012
	p	0.066		<0.001	0.557	0.593	0.848
Troponin I	Pearson correlation	0.092	0.231*	1	0.147*	−0.027	0.028
	p	0.148	<0.001		0.021	0.670	0.665
RDW	Pearson correlation	−0.012	0.038	0.147*	1	0.041	0.028
	p	0.849	0.557	0.021		0.520	0.662
PLR	Pearson correlation	−0.057	0.034	−0.027	0.041	1	0.488*
	p	0.375	0.593	0.670	0.520		<0.001
NLR	Pearson correlation	0.057	−0.012	0.028	0.028	0.488*	1
	p	0.371	0.848	0.665	0.662	< 0.001	

* Correlation is significant at the 0.05 level.
NLR = neutrophil lymphocyte ratio; PLR = platelet lymphocyte ratio; RDW = red cell distribution width.

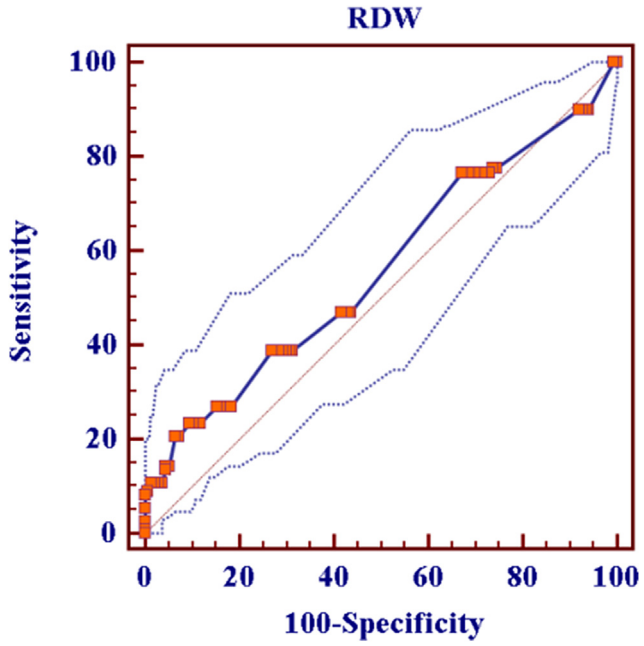

Figure 1. Receiver operating characteristic curve analysis of study patients for predicting pulmonary embolism. The area under the curve is 0.559. Criterion is over 18.9 with 20.7% sensitivity and 93.4% specificity. Standard error = 0.0369, 95% confidence interval = 0.495–0.622, *z* statistic = 1.602, $p = 0.1091$.

have shown the relationship between RDW and CAD. Tonelli et al conducted a *post hoc* analysis in the Cholesterol and Recurrent Events study and demonstrated that baseline RDW in patients with a history of acute myocardial infarction without symptomatic HF is an independent predictor of poor outcomes [12]. These results have been confirmed in many subsequent studies that indicate that RDW is associated with adverse outcomes in cardiovascular and non-cardiovascular disease states. An elevated RDW has been shown to have an association with death due to cardiac causes, nonfatal acute myocardial infarction, recent onset of heart failure, and stroke. These results have been confirmed in many subsequent studies [12–14,20]. The reason for the increase in RDW in cardiovascular and pulmonary diseases is not clearly understood. Nutritional deficiencies, comorbid diseases, impaired renal function, inflammatory cytokines, and erythropoiesis influenced by neurohormonal and adrenergic system activation have been shown to increase the level RDW [10].

In a recent study, Hampole et al showed that RDW is an independent predictor of mortality in patients with pulmonary hypertension due to any cause [15]. Another recent study reports an independent association between elevated RDW and an increased risk of acute PE-related early mortality. They showed that an RDW > 14.6% on admission is associated with an increased risk for acute PE-related early mortality [21]. This study was conducted only in patients with confirmed PE and it showed the importance of increased RDW as a predictor of early mortality.

In our study, we aimed to evaluate the role of the hematologic parameters RDW, NLR, and PLR in the diagnostic phase of PE. All three measures were elevated in patients with PE but only RDW was found to be a predictor of PE diagnosis. In contrast to the study by Fiarresga et al [22], our study included both patients with and without a diagnosis of PE. We included all patients for whom PE was included in the differential diagnosis and divided them into two groups based on the results of thoracic computed tomography. This comparison is one of the strengths of this study, making it very valuable for demonstrating the importance of RDW in evaluating patients suspected of having acute PE. Unfortunately, the receiver operating characteristic analysis of RDW for predicting PE did not adequately support the regression analysis. RDW values > 18.9 predicted PE with very high specificity but very low sensitivity. Therefore, larger studies are needed to demonstrate the diagnostic value of RDW for predicting PE.

In our study, we found that NLR and PLR values were higher in the PE group than in the non-PE group. Both of these measures are associated with inflammatory states. Recent studies have validated the role of NLR as an independent predictor of early mortality in various cardiovascular diseases [22–24]. It has been proposed that elevated serum cortisol levels due to increased sympathetic system activity in acute coronary syndrome (ACS) and acute decompensated HF may account for the alterations in neutrophil and lymphocyte counts in these patients [25]. Moreover, it has been proposed by some researchers that increased cytokine release from stimulated neutrophils may be responsible for the observed vascular injury and increased rate of intracoronary thrombus [26]. A recently published study also reports the prognostic value of NLR in patients with acute PE. Kayrak et al found that NLR on hospital admission may be a predictor of 30-day mortality in acute PE [16]. In our study, we initially demonstrated the diagnostic role of NLR in acute PE patients. NLR levels were higher in the PE group than the non-PE group, but in multivariate logistic regression analysis, it was shown that NLR is not a predictor for PE.

Previous studies have also shown a relationship between high values of peripheral thrombocytes and cardiovascular diseases. Higher platelet counts may indicate underlying inflammation because there are several inflammatory mediators that stimulate megakaryocytic proliferation and produce relative thrombocytosis. Furthermore, other studies have shown that patients with CAD have high levels of platelet-monocyte aggregates in their bloodstream, which is correlated with plaque stability [17,27]. According to these studies, we considered that there may be a relationship between PLR and PE. In fact, while PLR was found to be elevated in patients with PE, the significance was not supported by multivariate logistic regression analysis.

In this study, we found that both of the inflammatory markers NLR and PLR were higher in patients with PE compared to those without PE. We did not, however, find NLR and PLR to be independent predictors of PE. Elevated levels of RDW, NLR, and PLR are important criteria in the differential diagnosis of PE, especially because they can be measured with simple, inexpensive tests at the time of presentation to hospital. Finally, we propose that increased RDW values, along with troponin I and D-dimer levels, may be used to diagnose PE.

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underlying inflammation because there are several inflammatory mediators that stimulate megakaryocytic proliferation and produce relative thrombocytosis. Furthermore, other studies have shown that patients with CADs have high levels of platelet monocyte aggregates in their bloodstream, which is correlated with plaque stability [17,27]. According to these studies, we thought that PLR may related with PE. PLR was found to be significantly higher in PE patients but no significance was seen in multivariate logistic regression analysis.

In the present study we found that both NLR and PLR were higher in PE patients than controls as inflammatory markers. But we did not found NLR and PLR as an independent predictor for PE. Determining RDW, NLR and PLR can be done via easy, inexpensive tests at hospital admission. Increased levels of these three parameters were important for diagnosis for PE. Furthermore, we think that increased RDW values might be used to diagnose PE in combination with troponin I and D-dimer levels.

References

[1] Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999;353:1386—9.

[2] Roy PM, Meyer G, Vielle B, Le Gall C, Verschuren F, Carpentier F, et al. Appropriateness of diagnostic management and outcomes of suspected pulmonary embolism. Ann Intern Med 2006;144:157—64.

[3] Remy-Jardin M, Remy J, Deschildre F, Artaud D, Beregi JP, Hossein-Foucher C, et al. Diagnosis of pulmonary embolism with spiral CT: comparison with pulmonary angiography and scintigraphy. Radiology 1996;200:699—706.

[4] Wells PS, Anderson DR, Rodger M, Stiell I, Dreyer JF, Barnes D, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. Ann Intern Med 2001;135:98—107.

[5] Perrier A, Roy PM, Aujesky D, Chagnon I, Howarth N, Gourdier AL, et al. Diagnosing pulmonary embolism in outpatients with clinical assessment, D-dimer measurement, venous ultrasound, and helical computed tomography: a multicenter management study. Am J Med 2004;116:291—9.

[6] Becattini C, Vedovati MC, Agnelli G. Prognostic value of troponins in acute pulmonary embolism: a meta-analysis. Circulation 2007;116:427—33.

[7] Giannitsis E, Muller-Bardorff M, Kurowski V, Weidtmann B, Wiegand U, Kampmann M, et al. Independent prognostic value of cardiac troponin T in patients with confirmed pulmonary embolism. Circulation 2000;102:211—7.

[8] Pieralli F, Olivotto I, Vanni S, Conti A, Camaiti A, Targioni G, et al. Usefulness of bedside testing for brain natriuretic peptide to identify right ventricular dysfunction and outcome in normotensive patients with acute pulmonary embolism. Am J Cardiol 2006;97:1386—90.

[9] Kostrubiec M, Pruszczyk P, Bochowicz A, Pacho R, Szulc M, Kaczynska A, et al. Biomarker-based risk assessment model in acute pulmonary embolism. Eur Heart J 2005;26:2166—72.

[10] Förhécz Z, Gombos T, Borgulya G, Pozsonyi Z, Prohászka Z, Jánoskuti L. Red cell distribution width in heart failure: prediction of clinical events and relationship with markers of ineffective erythropoiesis, inflammation, renal function, and nutritional state. Am Heart J 2009;158:659—66.

[11] Van Kimmenade RR, Mohammed AA, Uthamalingam S, van der Meer P, Felker GM, Januzzi Jr JL. Red blood cell distribution width and 1-year mortality in acute heart failure. Eur J Heart Fail 2010;12:129—36.

[12] Tonelli M, Sacks F, Arnold M, Moye L, Davis B, Pfeffer M, et al. Relation between red blood cell distribution width and cardiovascular event rate in people with coronary disease. Circulation 2008;117:163—8.

[13] Fukuta H, Ohte N, Mukai S, Saeki T, Asada K, Wakami K, et al. Elevated plasma levels of B-type natriuretic peptide but not C-reactive protein are associated with higher red cell distribution width in patients with coronary artery disease. Int Heart J 2009;50:301—12.

[14] Dabbah S, Hammerman H, Markiewicz W, Aronson D. Relation between red cell distribution width and clinical outcomes after acute myocardial infarction. Am J Cardiol 2010;105:312—7.

[15] Hampole CV, Mehrotra AK, Thenappan T, Gomberg-Maitland M, Shah SJ. Usefulness of red cell distribution width as a prognostic marker in pulmonary hypertension. Am J Cardiol 2009;104:868—72.

[16] Kayrak M, Erdoğan HI, Solak Y, Akıllı H, Gül EE, Yıldırım O, et al. Prognostic value of neutrophil to lymphocyte ratio in patients with acute pulmonary embolism: a retrospective study. Heart Lung Circ 2014;23:56—62.

[17] Azab B, Shah N, Akerman M, McGinn Jr JT. Value of platelet/lymphocyte ratio as a predictor of all-cause mortality after non-ST-elevation myocardial infarction. J Thromb Thrombolysis 2012;34:326—34.

[18] Gary T, Pichler M, Belaj K, Hafner F, Gerger A, Froehlich H, et al. Platelet-to-lymphocyte ratio: a novel marker for critical limb ischemia in peripheral arterial occlusive disease patients. PLoS One 2013;8:e67688.

[19] Felker GM, Allen LA, Pocock SJ, Shaw LK, McMurray JJ, Pfeffer MA, et al. Redcell distribution width as a novel prognostic marker in heart failure: data from the CHARM Program and the Duke Databank. J Am Coll Cardiol 2007;50:40—7.

[20] Ani C, Ovbiagele B. Elevated red blood cell distribution width predicts mortality in persons with known stroke. J Neurol Sci 2009;277:103—8.

[21] Zorlu A, Bektasoglu G, Guven FM, Dogan OT, Gucuk E, Ege MR, et al. Usefulness of admission red cell distribution width as a predictor of early mortality in patients with acute pulmonary embolism. Am J Cardiol 2012;109:128—34.

[22] Fiarresga AJ, Ferreira RC, Feliciano J, Feliciano J, Timóteo AT, Pelicano N, et al. Prognostic value of neutrophil response in the era of acute myocardial infarction mechanical reperfusion. Rev Port Cardiol 2004;23:1387—96.

[23] Hoffman M, Blum A, Baruch R, Kaplan E, Benjamin M. Leukocytes and coronary heart disease. Atherosclerosis 2004;172:1—6.

[24] Karabinos I, Koulouris S, Kranidis A, Pastromas S, Exadaktylos N, Kalofoutis A. Neutrophil count on admission predicts major in-hospital events in patients with a non- ST-segment elevation acute coronary syndrome. Clin Cardiol 2009;32:561—8.

[25] Falk E, Shah PK, Fuster V. Coronary plaque disruption. Circulation 1995;92:657—71.

[26] Li DB, Hua Q, Liu Z, Li J, Xu LQ, Wang S, et al. Association between inflammatory mediators and angiographic morphologic features indicating thrombus formation in patients with acute myocardial infarction. Chin Med J (Engl) 2009;122:1738—42.

[27] Iijima R, Ndrepepa G, Mehilli J, Bruskina O, Schulz S, Schömig A, et al. Relationship between platelet count and 30-day clinical outcomes after percutaneous coronary interventions. Pooled analysis of four ISAR trials. Thromb Haemost 2007;98:852—7.