Introduction

Venous thromboembolism (VTE) has an overall annual incidence of 100-200 per 100,000 inhabitants. As estimated on the basis of an epidemiological model, over 317,000 deaths were related to VTE in six countries of the European Union in 2004. Acute pulmonary embolism (PE) is the most serious clinical presentation of VTE and it is a relatively common disease associated with serious short-term and long-term complications, and potentially high morbidity and mortality. Patients older than 40 years are at increased risk compared with younger patients and the risk nearly doubles with each subsequent decade.

The diagnosis of acute PE can be difficult because of the nonspecific signs and symptoms. Some blood parameters may be used as readily available and cost-effective in the diagnosis of PE but at present, there is no reliable diagnostic test in the definitive diagnosis of PE.

In addition, PE is associated with substantial mortality and morbidity as the third leading cause of cardiovascular system-related diseases after myocardial infarction and stroke. Acute PE increases the pressure of the pulmonary arterial system and right ventricle resulting in right ventricular dysfunction (RVD), which may progress to right heart failure and circulatory collapse. Patients with RVD have a higher mortality rate than those without, even if they are
Initially hemodynamically stable.

Circulating platelets are heterogeneous in size, density, and activity. Mean platelet volume (MPV), a measure of platelet size that is available in every blood count, is increasingly recognized as an important marker of platelet activity. The MPV has been investigated as an indicator of inflammation in different diseases. The MPV has also been noted to be increased in cardiovascular disease, peripheral artery disease and cerebrovascular disease. Several studies have been carried out in patients to evaluate MPV levels in PE. The associated between MPV and PE is controversial. Some studies have showed that stable PE patients have higher MPV than controls, whereas a report found that this association was not statistically significant.

Red cell distribution width (RDW) is a quantitative measure of the size variation of circulating erythrocytes with higher values reflecting greater heterogeneity in cell sizes. RDW is strongly associated with prognosis in cardiopulmonary disorders such as coronary artery disease, acute myocardial infarction, acute and chronic heart failure, and pulmonary hypertension. RDW has been associated with PE but whether RDW is a predictor of first event of PE is unknown.

Therefore, the present study aimed to evaluate the levels of MPV and RDW in patients with acute PE, to establish whether these markers are associated with echocardiography (ECHO) findings, cardiac biomarkers and mortality, and to determine their diagnostic value in these patients.

Material and methods

Study design

This study was retrospectively designed and enrolled 212 consecutive patients who were diagnosed and treated with acute PE between January 2013 and September 2014 and controls. This retrospective study was carried out in accordance with the Helsinki declaration, and approval was obtained from the local ethics committee of Firat University (Number: 64237 / 2014.12.02). The study was conducted at a university tertiary care hospital that serves as a primary referral center for patients with suspected PE.

Patients

The study consisted of consecutive patients with PE who were treated in our university hospital. PE patients were identified via the hospital database using the ICD-10 search code “I26.x” or “pulmonary embolism”. The diagnosis of pulmonary embolism was confirmed by examining computed tomography pulmonary angiography or ventilation-perfusion scintigraphy results. The primary end point was in-hospital mortality. Demographic characteristics and laboratory parameters were recruited from our hospital electronic database. Age, gender, arterial blood gases values, hemogram, D-dimer, cardiac troponin-I (cTnI), brain natriuretic peptide (BNP) and ECHO findings taken before treatment were recorded. In addition, the patients were divided into two groups according to ECHO findings: with RVD and without RVD.

Exclusion criteria were hematological disorders, autoimmune diseases, asthma, chronic obstructive pulmonary disease, pneumonia, active pulmonary tuberculosis, valvular diseases, coronary artery disease and peripheral arterial disease, systemic inflammatory diseases, heart failure, hepatic and renal failure, cancer, and use of antiagregant therapy (acetylic salicylic acid, dipyridamole, ticlopidine, and clopidogrel) within the last month.

Control group included 79 healthy subjects who were admitted to our chest outpatient clinic and did not have any pathology according to laboratory findings and physical examination. These 79 controls who matched for age and sex were also randomly chosen from the outpatient clinic records.

Biochemical analysis

We used hemoglobin, hematocrit, platelet count, MPV, RDW, D-Dimer, BNP, cTnI determined at the time of patient presentation.

Measurement of MPV and RDW levels

Samples referred for routine hematological testing over one working day were included in this study. The samples were collected in 2 mL blood tubes containing dipotassium ethylenedinitrotetra-acetic acid (K2EDTA). The RDW, hemoglobin and MPV were measured on Siemens Advia 2120 (Diagnostic Solutions, Milan, Italy). In Siemens Advia, the manufacturer states that the RDW is determined from the RBC volume histogram, in the narrow window comprised between 60 and 120 fl. For MPV determination, the manufacturer states that the cells are made spherical without modifying their volume by diluting with an iso-osmotic solution containing a surfactant. The Advia 2120 analyzers use two-dimensional platelet analysis; volume and refractive index are simultaneously deter-
mined on a cell-by-cell basis by measuring two angles of laser light scatter. The two scatter measurements are converted to volume (platelet size) and refractive index (platelet density) values. The platelet scatter cytogram map resolves volumes between one and 30 fL and refractive index values between 1.35 and 1.44. Large platelets with volumes between 30 and 60 fL are identified in the large platelet area of the red cell map. The reported 2D platelet count is the sum of platelets and large platelets identified in the platelet and red cell scatter cyagrams.

**Measurement of cTnl and BNP levels**

cTnl and BNP assays were performed on the Siemens ADVIA Centaur XP immunoassay analyzer. cTnl was measured with a chemiluminescent immunoassay, based on the 2-step sandwich method. The reactions were performed and detected in the ADVIA Centaur XP Immunoassay System (Siemens Healthcare Diagnostics K.K., Tokyo, Japan). The chemiluminescence generated by adding an oxidizer was measured with the detector, and the data were analyzed automatically.

**Measurement of D-dimer**

All D-dimer levels in plasma specimens [1 part sodium citrate (3.2%) with 9 parts venous blood] from patients were assessed by using the BCS XP coagulation analyzer. D-dimer measurements are expressed in fibrinogen equivalent units (FEU), with a detection range of 0.17-4.40 mg/L.

**Arterial blood gas measurement**

Arterial blood gas samples of PE patients, determined at the time of patient presentation, were used in our study. Arterial blood gas samples were taken at rest, in a sitting position and in room air at the room temperature. A blood gas analyzer device (Rapid lab 348 Biobak., Chiron, Bayer Diagnostic, UK) measured samples.

**Echocardiography**

Review of patient files revealed that echocardiographic images had been obtained from all cases in semi-supine position, apical four chambers, parasternal short-long axis and subcostal positions. According to ECHO findings, the presence of RVD was considered to be the presence of at least one of the following conditions: right ventricle hypokinesia (asymmetrical or delayed contraction), systolic paradoxal movement in the septal wall, right ventricular (RV) dilation (end-diastolic diameter >30 mm, or right/left ventricle diameter ratio >1).[11]

**Statistical analysis**

IBM Statistical Product and Service Solutions version 21.0 (IBM SPSS Statistics 21 program, Armonk, NY, USA) software was used. Descriptive statistics were reported, including mean, standard deviation, and percentage. Percentages were rounded to a whole number. Pairwise group variables were statistically compared using the student-t test. Variables with categorical data were statistically compared using chi-square tests. Correlation between MPV and variables with continuous data were evaluated by the Pearson rank correlation tests. Receiver operation characteristic (ROC) curve analysis was conducted to identify the optimal cut-off values of MPV and RDW to predict PE and RVD. Results are expressed as means ± standard deviations. A p-value < 0.05 was considered significant.

**Results**

Two hundred and twelve consecutive acute PE patients were initially included in our study. Seventy-nine patients who have any exclusion criteria and 11 patients who have insufficient data from hospital records and 14 patients who cannot underwent ECHO within first 24 hours were excluded the study. As a result, the study enrolled 108 patients with PE.

**Baseline demographic and laboratory characteristics**

The demographic and laboratory characteristics of the patients with PE and controls reported in Table 1.

<table>
<thead>
<tr>
<th></th>
<th>PE patients (n=108)</th>
<th>Controls (n=79)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>58.1 ± 16.9</td>
<td>56.7 ± 13.1</td>
<td>0.525</td>
</tr>
<tr>
<td>Sex, male/female</td>
<td>58/50</td>
<td>42/37</td>
<td>0.94</td>
</tr>
<tr>
<td>Hemoglobin, mg/dL</td>
<td>12.9 ± 1.8</td>
<td>13.4 ± 1.3</td>
<td>0.063</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>39.7 ± 6.3</td>
<td>40.3 ± 3.9</td>
<td>0.416</td>
</tr>
<tr>
<td>Platelet, x109/L</td>
<td>257.9 ± 112.2</td>
<td>272.2 ± 76.4</td>
<td>0.303</td>
</tr>
<tr>
<td>MPV, fL</td>
<td>8.9 ± 1.1</td>
<td>8.4 ± 0.8</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>RDW, %</td>
<td>15.7 ± 2.6</td>
<td>14.1 ± 4.3</td>
<td>0.006</td>
</tr>
</tbody>
</table>

Table 1: The demographic and laboratory characteristics of the patients with pulmonary embolism and controls. MPV: Mean platelet volume, RDW: Red cell distribution width.
There was no significant difference between two groups regarding age, sex, and laboratory parameters including hemoglobin, hematocrit, and platelet count. The mean serum MPV and RDW levels were significantly higher in patients with PE than control group (p<0.001, p=0.006).

**Right ventricular dysfunction**

The mean MPV and RDW levels were statistically higher in PE patients with RVD than PE patients without RVD (p=0.026, p<0.001). As expected, the mean cTnI and BNP levels were statistically higher and the levels of partial pressures of arterial oxygen (PaO\(_2\)) and arterial oxygen saturation (SaO\(_2\)) were statistically lower in PE patients with RVD compared with PE patients without RVD (p<0.001, for all parameters). RV diameter and systolic pulmonary artery pressure (sPAP) were significantly higher in PE patients with RVD than PE patients without RVD (p<0.001, for both). All parameters were shown in table 2.

**In-hospital mortality**

The hospital mortality rate in PE patients was 5.9% (n=11). The mean age was statistically higher in non-survivor patients than survivor patients (p=0.033). Similarly, the mean MPV, RDW, cTnI and BNP levels, RV diameter and sPAP were statistically higher in non-survivors than survivors (p<0.001, for all parameters). In addition, the levels of SaO\(_2\) were statistically lower in non-survivors than survivors (p=0.01) (Table 3).

**Correlations**

Serum levels of MPV showed a weak to moderate positive correlation with RV diameter (r=0.221, p=0.021) and BNP (r= 0.277, p=0.004). There was a weak to moderate positive correlation between RDW and RV diameter (r=0.424, p<0.001), sPAP (r=0.386 p<0.001), BNP (r=0.359 p<0.001), and cTnI (r=0.306, p=0.001).

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**Table 2**: The demographic, laboratory, and echocardiographic findings of the pulmonary embolism patients with and without right ventricular dysfunction.

<table>
<thead>
<tr>
<th></th>
<th>PE patients with RVD (n=64)</th>
<th>PE patients without RVD (n=44)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>64.1 ± 15.2</td>
<td>49.4 ± 15.6</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Sex, male/female</td>
<td>29/35</td>
<td>29/15</td>
<td>0.035</td>
</tr>
<tr>
<td>Hemoglobin, mg/dL</td>
<td>13.0 ± 1.7</td>
<td>12.9 ± 1.9</td>
<td>0.668</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>39.9 ± 6.8</td>
<td>39.4 ± 5.6</td>
<td>0.688</td>
</tr>
<tr>
<td>Platelet, x10⁹/L</td>
<td>244.3 ± 111.2</td>
<td>277.7 ± 111.9</td>
<td>0.13</td>
</tr>
<tr>
<td>MPV, fl</td>
<td>9.2 ± 1.0</td>
<td>8.7 ± 1.1</td>
<td>0.026</td>
</tr>
<tr>
<td>RDW, %</td>
<td>16.3 ± 2.9</td>
<td>14.7 ± 1.9</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>D-Dimer, mg/L</td>
<td>4.5 ± 3.6</td>
<td>3.8 ± 3.6</td>
<td>0.3</td>
</tr>
<tr>
<td>cTnI, ng/mL</td>
<td>0.19 ± 0.30</td>
<td>0.016 ± 0.028</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>BNP, pg/dL</td>
<td>496.2 ± 553.2</td>
<td>492 ± 53.0</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>ABG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PaO(_2), mmHg</td>
<td>57.0 ± 10.2</td>
<td>64.6 ± 8.1</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>SaO(_2), %</td>
<td>87.2 ± 6.8</td>
<td>92.5 ± 2.6</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>ECHO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVDi, mm</td>
<td>28.1 ± 3.3</td>
<td>22.8 ± 1.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>sPAP, mmHg</td>
<td>59.0 ± 20.4</td>
<td>30.5 ± 4.1</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

**Table 3**: Comparison of the demographic, laboratory and echocardiographic characteristics between survivors and non-survivors of the patients with pulmonary embolism. MPV; Mean platelet volume, RDW; Red cell distribution width, cTnI; Cardiac troponin-I, BNP; Brain natriuretic peptide, ABG; Arterial blood gases, PaO\(_2\); Arterial oxygen pressure, SaO\(_2\); Arterial oxygen saturation, ECHO; Echocardiography, RVDi; Right ventricular diameter, sPAP; Systolic pulmonary artery pressure.

<table>
<thead>
<tr>
<th></th>
<th>Non-survivors (n=11)</th>
<th>Survivors (n=97)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>68.4 ± 13.2</td>
<td>56.9 ± 16.9</td>
<td>0.033</td>
</tr>
<tr>
<td>Sex, male/female</td>
<td>4/7</td>
<td>54/43</td>
<td>0.224</td>
</tr>
<tr>
<td>Hemoglobin, mg/dL</td>
<td>11.7 ± 1.4</td>
<td>13.1 ± 1.8</td>
<td>0.012</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>38.9 ± 6.2</td>
<td>39.8 ± 6.3</td>
<td>0.646</td>
</tr>
<tr>
<td>Platelet, x10⁹/L</td>
<td>219.6 ± 72.6</td>
<td>262.3 ± 115.3</td>
<td>0.234</td>
</tr>
<tr>
<td>MPV, fl</td>
<td>10.2 ± 1.3</td>
<td>8.8 ± 0.9</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>RDW, %</td>
<td>18.7 ± 3.0</td>
<td>15.3 ± 2.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>D-Dimer, mg/L</td>
<td>3.8 ± 1.7</td>
<td>4.2 ± 3.7</td>
<td>0.692</td>
</tr>
<tr>
<td>cTnI, ng/mL</td>
<td>0.41 ± 0.47</td>
<td>0.09 ± 0.19</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>BNP, pg/dL</td>
<td>879.1 ± 562.3</td>
<td>250.0 ± 427.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>ABG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PaO(_2), mmHg</td>
<td>56.1 ± 12.7</td>
<td>60.5 ± 9.7</td>
<td>0.168</td>
</tr>
<tr>
<td>SaO(_2), %</td>
<td>84.9 ± 7.9</td>
<td>89.8 ± 5.7</td>
<td>0.01</td>
</tr>
<tr>
<td>ECHO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVDi, mm</td>
<td>29.6 ± 3.9</td>
<td>25.5 ± 3.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>sPAP, mmHg</td>
<td>71.7 ± 28.1</td>
<td>44.6 ± 18.5</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

**PE**: Pulmonary embolism, **RVD**: Right ventricular dysfunction, **MPV**: Mean platelet volume, **RDW**: Red cell distribution width, **cTnI**: Cardiac troponin-I, **BNP**: Brain natriuretic peptide, **ABG**: Arterial blood gases, **PaO\(_2\)**: Arterial oxygen pressure, **SaO\(_2\)**: Arterial oxygen saturation, **ECHO**: Echocardiography, **RVDi**: Right ventricular diameter, **sPAP**: Systolic pulmonary artery pressure.
The ROC curve analysis of MPV and RDW when predicting acute PE were constructed and the area under the curve (AUC) was found to be 0.645 (95% CI 0.567 to 0.723, p=0.001) for MPV and AUC was found to be 0.749 (95% CI 0.679 to 0.820, p<0.001) for RDW (Figure 1).

The optimal cut-off values for MPV and RDW when predicting acute PE were 8.25 fl (sensitivity 72%; specificity 43%) and 13.45% (sensitivity 79%; specificity 52%) respectively. In addition, the ROC curve analysis of MPV and RDW when predicting for RVD in patients PE were constructed and AUC was found to be 0.635 (95% CI 0.526 to 0.745, p=0.017) for MPV and AUC was found to be 0.687 (95% CI 0.585 to 0.789, p=0.001) for RDW (Figure 2). The optimal cut-off value of MPV and RDW for the determining of RVD was 8.35 fl (Sensitivity 76%; specificity 50%) and 14.05% (Sensitivity 79%; specificity 50%) respectively.

Discussion

Our study shows increased levels of MPV and RDW in PE patients. In particular, the higher serum MPV and RDW levels were determined in PE patients with RVD. In addition, the MPV and RDW levels were higher in non-survivors than survivors in PE patients. The positive relationship was found between MPV and RDW levels and RV diameter, sPAP and cardiac biomarkers (BNP and cTnI).

The thrombotic burden is expected to be large in PE patients. MPV is an indicator of platelet activation. There was a strong evidence indicating that MPV is an important variable and that larger platelets have a higher thrombotic potential.

In previous studies showed that there was a relationship between MPV and acute deep venous thrombosis (DVT). An increasing MPV was identified as a predictor for venous thromboembolism (VTE) in a previous study. The role of MPV in PE is still controversial. Several investigations have assessed the role of MPV in PE patients. Some results showed that there were no significant differences in the MPV levels between the patients with acute PE and controls. Conversely, some study results reported that increased MPV levels in PE patients compared to the controls. Also in a previous study demonstrated that MPV is a helpful parameter for the diagnosis of acute PE in emergency department (Sensitivity 82.2%; specificity 52.3%).

Similar to these results, increased levels of MPV in PE patients were found in our study. In addition we found the best cut-off value for MPV when predicting PE in patients with clinically suspected acute PE were 8.25 fl (sensitivity 72%; specificity 43%). These result interpreted that increased MPV levels may reflect the burden of increased aggregation and MPV may be an indicator for diagnosing acute PE. As has been reported before, the MPV values that reflect significant changes in platelet reactivity and aggregation are increased in arterial and venous thrombus and increased MPV values are risk factors for thrombosis.

In addition, previously studies reported that MPV was higher in non-survivors than survivors. Increased levels of MPV have been recognized as an independent predictor for early death in acute PE and increased MPV value may be used as a marker for early hospital mortality in patients with PE. Moreover, platelet activation was also observed in patients after acute PE, and correlated with the
severity of RVD\textsuperscript{(17,21,22)}. MPV was found to be a significant predictor of 30-day mortality in PE patients and especially MPV levels $>10.9$ fl showed significantly increased mortality. Hypoxemia, RVD and RV failure, in association with impaired left ventricular filling and reduced cardiac output, are potent stimuli of platelet activation\textsuperscript{(17)}. Vasoconstrictor mediators such as thromboxane release from platelets may also augment pulmonary vascular resistance and promote RV ischemia and dysfunction\textsuperscript{(23)}. Similar to previous studies we also found significant correlations between MPV and RV diameter and increased levels of MPV in non-survivors than survivors\textsuperscript{(2,16,17)}.

In addition, PaO\textsubscript{2} and SaO\textsubscript{2} were statistically lower in non-survivors than survivors. Increased troponin levels and their prognostic significance in acute PE were reported previously in many studies\textsuperscript{(9,10)}. Elevated troponin levels presented higher MPV values\textsuperscript{(17)}. Increased cTnI and MPV levels were found in PE patients with RVD in our study. Although we did not find any relationship between MPV and cTnI levels, increased MPV levels may be contributing factor to myocardial injury especially in PE patients with RVD. It seems justified to suggest that in addition to haemodynamic factors such as increased RV overload, hypotension and hypoxemia, platelet activation can contribute to myocardial injury\textsuperscript{(17)}. Moreover increased MPV levels ($>8.35$ fl) may be used to predict the existence of RVD in these patients.

RDW, an inexpensive and simple laboratory variable, was independently and significantly associated with the presence and severity of DVT\textsuperscript{(26)}. Moreover, previous studies have demonstrated the RDW as a prognostic marker in patients with PE\textsuperscript{(9)}. Increased RDW level was an independent predictor of short-term mortality in PE\textsuperscript{(9,26)}. The cut-off values of RDW were found $>14.6\%$ and of $>13.7\%$ for the increased risk for acute PE-related mortality in the early phase of hospitalization.

In patients with PE who have increased levels of RDW had more severe disease state that main pulmonary artery involvement, RV impairment, increased sPAP\textsuperscript{(9,10)}. There was a strong association between increased RDW levels with adverse outcomes in patients with chronic heart failure, pulmonary hypertension and myocardial infarction. Proposed mechanism for the association of the elevated RDW in these patients is inflammation\textsuperscript{(29,31,32)}.

Also, several studies have shown a relationship between inflammation and VTE\textsuperscript{(33-35)}. Inflammation may also play a role in VTE\textsuperscript{(36)}. In a previous study, significant correlation was found between C-reactive protein and RDW in patients with PE. Therefore, their data suggest that high RDW levels may reflect various pathologic conditions, such as inflammatory stress and may be potentially attributed to the poor outcomes in patients with PE\textsuperscript{(36)}. Beside the inflammation and related cytokines that might affect erythropoiesis, other propose etiologies are a subtle anemia and neurohormonal axis (that can cause red blood cell production)\textsuperscript{(37)}.

In a previous study demonstrated that RDW was moderately correlated with hemodynamic parameters and RDW was also found to be related to right-sided cardiac function. For this reason, they think that RDW may reflect increasing severity of acute PE\textsuperscript{(9)}.

In the present study, we found increased levels of RDW in PE patients compared to controls, in PE patients with RVD compared to ones without RVD and in non-survivors compared to nonsurvivors. In addition, RDW positively correlated with RV diameter, sPAP, BNP and cTnI. Moreover we found serum RDW $>13.45\%$ showed $79\%$ sensitivity and $52\%$ specificity for the diagnosis of PE and RDW $>14.05\%$ showed $79\%$ sensitivity and $50\%$ specificity for the existence of RVD in these patients. Therefore, we think that high RDW levels may be use for the diagnosis of PE and to predict of patients who had RVD and hence, it may be use the PE patients who have highest risk of mortality.

Main limitation of our study was retrospective design and the small number of patients. These observations must be confirmed in prospective studies with a larger sample size.

In conclusion, MPV and RDW measurements may provide a non-invasive, sensitive, and immediate way of assessing patients with PE. MPV and RDW may be use in both diagnosis of PE and showing RVD in PE. However, these approaches may be addressed by further studies.
The importance of mean platelet volume and red cell distribution width in acute pulmonary embolism

References


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Corresponding author
ERDAL İN
Department of Chest Diseases, Firat University
School of Medicine
23119, Elazig
(Turkey)