Usefulness of Admission Red Blood Cell Distribution Width as a Predictor of severity of Acute Pulmonary Embolism

Running Head:

Severity of Acute Pulmonary Embolism relationship with the RDW

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Abstract

Background: Previous researches have represented a considerable relation between acute pulmonary embolism (PE) and red blood cell distribution width (RDW). To the authors’ knowledge no research has been informed in subjects with PE severity. Pulmonary arterial obstruction index (PAOI) is associated with the severity of acute PE.

Objectives: In our investigation, we purposed to assess the relation between PAOI and RDW and the benefit of these factors in the detection of PE severity.

Methods: We retrospectively investigated the demographic information, probability of clinical scores, laboratory parameters, serum D-dimer levels, and echocardiographic findings of systolic pulmonary artery pressure (sPAP) in Acute PE individuals who were diagnosed by computed tomography of pulmonary arterial angiography. Right ventricular dysfunction (RVD) on CT was assessed by calculating the right ventricular/left ventricular (RV/LV) diameter ratios on transverse (RV/LVtrans).

Results: The information of 131 patients with acute PE and 51 (64.6%) female and 28 (35.4%) male healthy control were evaluated. Acute PE group’s RDW values were higher than control subjects (P < 0.0001). RDW (%) level was remarkable higher in patients with massive PE than in patients with nonmassive PE. There were statistically considerable differences in terms of PAOI and systolic pulmonary arterial pressure (sPAP) between nonmassive and massive PE patients (P < 0.0001 for all).

Conclusions: PAOI was correlated with PE severity, D-dimer level, sPAP and clinical probability scores. PAOI was correlated with RDW levels. RDW levels, an inexpensive and easily measurable laboratory factor, were considerable associated with the severity and presence of PE.
Key words:
Pulmonary embolism, Red cell distribution width, Pulmonary Artery Pressure, Pulmonary arterial obstruction index, Multidetector Computed Tomography

Introduction

Despite substantial progress in the diagnosis and treatment of acute pulmonary embolism (PE), this event is a frequent cause of mortality. Acute PE might cause a potentially life-threatening situation depending on an increase in the occlusion degree of the pulmonary vascular bed[1]. Acute PE risk assessment includes hemodynamic parameters and specific biomarkers. The usefulness of plasma measurements of the circulating d-dimer has been evaluated usually using a diagnostic assay in suspected acute PE [2]. An alternative marker, cardiac troponin levels, has been evaluated in the setting of acute PE [3]. The medical literature has reported on the computed tomography (CT) pulmonary arterial obstruction index (PAOI), which gauges the clinical validity of the extent and degree of thrombotic pulmonary arterial occlusion in individuals with acute PE [4].

The red blood cell distribution width (RDW), a component of the standard complete blood count in a routine hematology laboratory test, is a measurement of the size differentiation of circulating red blood cells and an index of their heterogeneity. Red blood cells (RBCs) are components in clots and thrombi formed *in vivo* [5]. Moreover, small structural variation in RBCs might have a major effect on pathophysiology[5]. Reliable data from various clinical studies have shown a new and unpredictable scenario in the clinical usefulness of this measure, promoting the hypothesis that the RDW may be an effective parameter for collecting meaningful clinical knowledge, either regarding the diagnosis or type of PE. Different lines of evidence have recently shown that a large RDW is related to a higher probability of various disorders. The RDW is strongly related to prognosis in cardiopulmonary disorders such as acute myocardial infarction, coronary artery disease (CAD), chronic and acute heart failure, and pulmonary hypertension[6-11]. However, the diagnostic and variety value of RDW in massive PE is unknown.

In this study, we assessed the relationships among clinical scores (Genova score and Wells score), D-Dimer, cardiac troponin I (cTnI) levels, RDW, and PAOI, and the implications of these relationships in medical clinical experience.
Materials and Methods

This study was approved by the ethics review board of our local ethics committee. The study was planned according to the Declaration of Helsinki and the rules of the Hospital Ethics Committee. We retrospectively screened 250 patients older than 18 years who were admitted to the hospital with suspected PE between January 2010 and May 2015. Acute PE was diagnosed according to their multidetector pulmonary arterial computed tomography angiography (PCTA) results. In addition, we registered only those whose laboratory data (D-Dimer, CBC, cTnI), clinical information, including symptoms, medical history, clinical scores (Geneva score and Wells score), smoking habits, ECO results, and PCTA images could be accessed.

The control patient group consisted of 79 sex- and age-matched healthy persons without any risk components or accompanying diseases who visited the outpatient clinic departments for routine medical examination. Therefore, blood samples for CBC were also taken from controls.

Exclusion criteria

Individuals with hematological disorders (anemia, WBC <3.0 × 10^9/L or >20.0 × 10^9/L), pneumonia, acute infection, systemic hypertension, diabetes mellitus, congestive heart failure, ischemic heart disease, malignancies, inflammatory diseases, liver and renal disease, a history of malnutrition and smoking, alcohol consumption, and the present use of immunosuppressant medications (such as steroids) were excluded from the study. After excluding 119 participants with PE, 131 patients with acute PE and 79 patients with non-PE were suitable for the study.

Evaluation of the severity of PE

Patients older than 18 years presenting with acute-onset dyspnea, chest pain, or syncope without any another diagnosable reason in whom acute PE was verified were registered in this study. The acute PE patient group was subdivided into three subgroups according to the severity of disease: the nonmassive embolism, submassive embolism, and massive embolism groups[4, 12]. The presence of cardiogenic shock and/or arterial hypotension (systolic arterial pressure <90 mmHg) and thrombus, as demonstrated by PCTA in one or both common pulmonary arteries or greater than 50% of pulmonary vascular bed occlusion by a thrombus, was considered a massive embolism. Submassive events included normotensive patients with PE and proof of RV dysfunction. Whole events that did not meet these criteria were considered nonmassive embolisms[4, 12]
Image studies:

All CTA examinations were implemented on a 16-slice MDCT scanner (Light speed; General Electric Medical System, Milwakuee, WI, USA). Idendification of APE was defined in case of a partial or complete luminal filling deficit in the main pulmonary artery or its branches [13]. The embolic burden was categorized according to the most proximal pulmonary artery branch in which a thrombus was visualized as follows: central, lobar or segmental. RV/left ventricular (LV) for pe axial ratio was calculated by measuring the axial section showing the maximum distance between the interventricular septum (IVS) and ventricular endocardium, the perpendicular to the long axis of the heart for both right and left ventricles.

Blood sample analyses

The complete blood count (CBC) and differentials were analyzed for the peripheral venous blood samples taken on acceptance to the emergency clinical department. Blood samples were collected in a calcium-EDTA tube. CBC was conducted using an auto-analyzer (Abbott Cell-Dyn 3700 Ruby Hematology Analyzer, USA). The RDW level was measured using an automated counter by dividing the standard deviation of the red blood cell volume (MCV) by the mean MCV, and then multiplying the result by 100 to express it as a percentage (RDW% = [standard deviation MCV/mean MCV] × 100) [15]. Other routine laboratory data were registered using the electronic records of the hospital.

D-dimer and TROP test

The D-dimer levels were measured using the latex-enhanced photometric method (Sta Compact, Dade Behring, Marburg, Germany) using D-dimer kits.

For troponin I (cTnI), a quantitative chemiluminescence assay (DRG International Inc., Mountainside, New Jersey, USA; Cat No, EIA) was performed.

Echocardiography

ECO evaluations of acute PE cases on the same day of diagnosis were assessed. The ECO outcome of PE, such as the systolic pulmonary arterial pressure (sPAP), was recorded.

Statistical analysis

Continuous variables are given as the mean ± standard deviation; categorical variables were detected as a percentage. A value of $p < 0.05$ was considered significant. The Kolmogorov–
Smirnov test was used to identify the distribution of continuous variables. The \( x^2 \) test and Fisher’s exact test were used to compare categorical variables. Student’s \( t \)-test and/or Mann-Whitney \( U \)-test were used to compare parametric or nonparametric factors between the PE and the control groups. RDW was not normally distributed the Kruskal Wallis tests were conducted to compare these PE groups. Bonferroni correction was used to adjust for multiple comparisons.

The relations between PAOI and the laboratory values [RDW, D-D-dimer, cTnI], probability of clinical scores were analyzed using the Spearman correlation coefficient analysis. Receiver operating characteristic (ROC) curve analysis was used to detect the optimum cutoff levels of RDW ratio in association with PE. The cut-off levels, sensitivity and specificity values of RDW in massif PE patients were determined. A trial version of SPSS 15.0 software was used for basic statistical analysis (Version 15; SPSS Inc. Chicago, IL, USA).

**Results**

The demographic characteristics and RDW levels of the PE group and control group are shown in Table 1. The mean ages of the acute PE patient group and control group were 63.9±11.8 years and 62.3±8.5 years, respectively \( (P = 0.079) \). The study records of 57 (43.5%) males and 74 (56.5%) females with acute PE and 28 (35.4%) male and 51 (64.6%) female healthy controls were evaluated \( (P = 0.249) \). There were no significant differences among the PE groups with respect to sex and age \( (P = 0.066 \text{ and } P = 0.077, \text{ respectively}) \).

The mean RDW value of the patients was 14.58±1.88. The corresponding value for controls was 13.65±0.66. There were statistically significant differences between the groups in terms of the RDW value \( (P < 0.0001; \text{ as shown in Table 1}) \). There was statistically significant difference between PE patients and control subjects regarding many of the clinical parameter (as shown in Table 1).

We categorized the severity of PE according to the principle of PAOI as mild (<40%), moderate (40–60%), or severe (>60%). The mean RDW, D-dimer level, CTnI level, and clinical scores for the groups determined based on this classification are shown in Table 2. The RDW values increased among the mild (<40%), moderate (40–60%), and severe (60%) groups according to PAOI \( (P < 0.0001) \). Moreover, significant differences were determined between PAOI and the PAB \( (P < 0.0001) \), D-dimer \( (P < 0.0001) \), CTnI \( (P < 0.0001) \), and Genova scores \( (P = 0.003) \).
We also categorized the severity of PE based on RV/LV ratios: mild (RV/LV ratio <1), moderate (RV/LV ratio 1.1–1.4), and severe (RV/LV ratio >1.4). The mean RDW, D-dimer level, CTnI level, and Wells and Genova scores for the groups based on this classification are shown in Table 3. Significant variation was found between the RV/LV ratio and RDW level ($P < 0.0001$). In addition, significant differences were found between the RV/LV ratio and the PAB ($P \leq 0.0001$), D-dimer ($P = 0.003$), CTnI ($P = 0.02$), and Wells ($P = 0.012$) values.

There were statistically significant differences among RDW, PAOI, CTnI, and sPAP in nonmassive and massive PE patients ($P < 0.0001$ for all). RDW, PAOI, CTnI, and sPAP were also higher in patients with massive PE than in those with nonmassive PE (Table 4).

The RDW values were correlated with PAOI ($r = 0.658$, $P < 0.0001$) (Figure 1), sPAP ($r = 0.470$, $P < 0.0001$), RV/LV ratio ($r = 0.389$, $P < 0.0001$), CTnI ($r = 0.234$, $P = 0.0001$), Wells score ($r = 0.234$, $P = 0.008$), and Geneva score ($r = 0.273$, $P = 0.003$). The PAOI was correlated with the sPAP level ($r = 0.635$, $P < 0.0001$), RV/LV ratio ($r = 0.569$, $P < 0.0001$), CTnI ($r = 0.547$, $P < 0.0001$), and Wells and Geneva scores ($r = 0.28$, $P = 0.001$ and $r = 0.36$, $P < 0.0001$, respectively) (Table 5). The average D-Dimer value was positively correlated with the sPAP level ($r = 0.391$, $P < 0.0001$), RV/LV ratio ($r = 0.269$, $P = 0.01$), CTnI ($r = 0.424$, $P < 0.0001$), and Wells scores and Geneva scores ($r = 0.36$, $P < 0.001$ and $r = 0.33$, $P = 0.001$, respectively).

In our study, RDW level was related with an increased risk of massive PE, even when the RDW level was still within the usual reference range (above 14.55%). The area under the ROC curve for RDW level, which was used to determine massive PE was calculated as 14.55%. Its sensitivity and the specificity of the best cut-off level were 92.1% and 55.2%, respectively. The cut-off level of 14.55% for RDW level was found to be sensitive and poorly specific for predicting PE. An RDW level of more than 14.55% elevated the risk of presence of PE by 4.2 times (OR 4.5; 95% CI 2.26–6.35).

**Discussion**

To the best of our knowledge, this is the first demonstration that a higher RDW value (%) is independently and significantly related to the presence and severity of acute PE. We investigated whether there is a correlation between PE severity (comparing massive PE with nonmassive PE) and RDW. We detected higher RDW levels with increasing PAOI. Several studies and a meta-analysis have recently demonstrated that a high RDW is not only related...
to hematological disorders [14, 15] but is also related to cardiovascular disorders including coronary artery disease [8], heart failure [16], conditions requiring percutaneous coronary intervention [17], and a first acute cerebral infarction [18]. Increased RDW has also been reported in patients with venous thromboembolism [19].

The cause for the enhanced RDW in pulmonary diseases is not well understood. Virchow [20] defined the mechanistic role of blood rheology in the pathophysiology of PE. In addition, abnormalities in blood rheology and enhanced blood viscosity are risk components for venous thromboembolism [21]. In the event of venous thrombosis, we hypothesize that greater heterogeneity in cell size could enhance the viscosity and disrupt blood flow, causing stasis, one of the primary risk determinants for venous thrombosis [22]. Enhanced local blood viscosity, induced by red blood cell aggregation, decreases blood flow, triggering the production of thrombosis by promoting the collision, activation, and adhesion of platelets as well as vessel wall adhesion [22].

Few studies have evaluated RDW values in patients with acute venous thrombosis [23]. In a previous study, a high RDW was related to the severity and presence of DVT, particularly proximal DVT [24]. However, no study has evaluated the relationship between the RDW and PE risk and severity. When we investigated this relationship in different subgroups of patients, we found a higher risk for massive PE than nonmassive PE with increased RDW levels. Moreover, RDW was related to an increased risk of massive PE, even when it was still within the usual reference range (above 14.95%). We suggest that RDW may be used in the evaluation of PE risk and severity according to these outcomes.

We also investigated the predictability of erythrocyte indices and PAOI scores in the definition of acute PE severity (nonmassive/massive), and found an association between the PAOI and RDW with acute PE severity. There was also more diffuse disease and proximal involvement in patients with a higher RDW.

Many studies have detected an association between PE severity and radiological scoring methods, interpreting the results using digital subtraction pulmonary angiography (Miller index) and CT [13, 25, 26]. Qanadli et al. [13] suggested that the CT index was associated with medical clinical results, such as systolic blood pressure and ECO evidence [24, 25, 27]. A considerable association between clinical scores and PAOI has been reported [4]. In our study, we found an association between the PAOI and both the Genova and Wells scores, as well as a positive relationship between the PAOI and D-Dimer level ($r = 0.456, P < 0.0001$). In agreement with the literature, there were also associations between RDW and the means of both clinical scores ($P < 0.05$ for both). We determined the degree of
occlusion-PAOI using a formula recommended by Qanadli et al. [5], and demonstrated that the PAOI was considerably enhanced in massive PE patients ($P < 0.0001$). Recently, the RDW was shown to be related to venous thromboembolism in two case-control studies [23, 24]. In our study, we found that the RDW was higher among PE patients and that there was a considerable relationship between RDW and PAOI. The mean RDW was markedly higher in the patients with massive PE. To the best of our knowledge, this is the first study to investigate the relationship between PAOI and an elevated RDW. Our results support our hypothesis that the RDW can predict disease severity in acute PE patients.

According to a previous study, the ratio of the short-axis diameters of the right ventricle to the left ventricle (RV/LV) generates appropriate data to detect deterioration of RV function [28] [29]. The considerable correlation between cTnI levels and both the PAOI and RV/LV ratio has recently been emphasized [30, 31]. Consistent with these studies, we also found a significant correlation between these parameters. Individuals with a higher RDW had more severe disease with involvement of the main pulmonary artery, increased sPAP, right ventricular impairment, and the presence of shock; in addition, elevated release of troponin was more common in individuals with higher RDW levels [15, 32]. We showed that a higher RDW was independently and significantly related to the presence and severity of PE. RDW levels in the massive PE group were higher than those in the nonmassive PE group.

Moreover, the RDW was significantly correlated with the sPAP, RV/LV, and cTnI levels. These outcomes also strengthen our study in that the RDW can predict the severity of disease in PE patients. Furthermore, the mean RDW value was associated with the average value of sPAP, a finding that must be confirmed in further studies. Oh et al. [33] investigated the association between RDW and ECO parameters and concluded that this marker may be related to increased LV filling pressure in patients with acute heart failure.

Regarding D-dimer values and thrombus localization, the D-dimer values may differentiate patients with segmental and larger thrombi from those with subsegmental thrombi [34]. Kucher et al. [35] demonstrated that PE patients with higher PAOI values also have higher D-dimer levels. In our study, we found a significant association between thrombus localization and the serum level of D-dimer. Furthermore, the mean D-dimer values were higher in those with MPA-localized thrombi than in patients with thrombi in other arterial branches. Hence, we suggest that RDW values might be useful for the evaluation of PE and its severity. However, we did not determine a significant correlation between RDW and serum level of D-dimer.
In this study, we found that RDW, sPAP, RV/LV ratio, cTnI and D-Dimer levels were related with the pulmonary arterial occlusion level defined by PAOI. These results recommended that pulmonary arterial occlusion level that was the indicator of the massive PE can be predicted with the increased level of RDW, sPAP, RV/LV ratio, cTnI and D-Dimer. So these markers increase many invasive and costly methods used in establishing disease severity. There was also more severity disease and proximal involvement in patients with higher level of RDW in our research.

Considering the clinical implications of our results, we observed that, chiefly, the RDW levels is a routinely practical test as a component of the automated CBC that is a standard practicable factor of routine medical use. For instance, on admission, standard cardiac biomarkers might be used in patients with high RDW levels in PE. It would be a cost-effective approach. In addition, high RDW values in patients with acute PE might provide us with increased clinical understanding from the point of view of the risk and severity stratification of patients with acute PE. It would be preferable to follow up on the RDW levels in patients who have a high risk for acute PE.

There were some limitations to this study. First, it was a clinical study and thus the underlying pathophysiological mechanisms might only be speculative. Second, we did not investigate the causes of the increased RDW values, such as vitamin B12 or iron deficiency, which could confound the association between RDW and adverse outcomes. Third, the CT parameters were interpreted by one radiologist. Analysis of the reliability (interobserver variability) of the results of the CT parameters by two independent radiologists is more appropriate than evaluation by one radiologist. Fourth, because of the low patient number due to stratification by sex, we could not determine the role of hemoglobin, hematocrit, and erythrocyte levels on the risk of PE. Fourth, a question that our results cannot answer is whether the relationship concerning a high RDW can be explained by the presence of hemoglobinopathies, which could cause changes in blood cell counts. Finally we did not detect RDW level after treatment so we could not compare the changes of RDW before and after treatment.

**Conclusion**

Determining the RDW can be performed via easy, inexpensive tests at hospital admission. Elevated levels of this parameter are associated with an increased risk for massive PE. Furthermore, we believe that elevated RDW values may be used to diagnose PE in combination with cTnI, sPAP and PAOI levels.
Contributors

RA contributed to manuscript preparation and study design.

HK contributed to the literature search and study design.

ABK, HK and ZYG contributed to study design.

RA, ZYG contributed to analysis of data contribution;

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Table 1. Comparison of demographic data, clinical parameters and RDW between PE patients and control subjects

<table>
<thead>
<tr>
<th></th>
<th>PE group</th>
<th>Control group</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patients (n)</strong></td>
<td>131</td>
<td>79</td>
<td></td>
</tr>
<tr>
<td><strong>F/M</strong></td>
<td>74/57</td>
<td>51/28</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td>63.9 ± 11.8</td>
<td>62.3 ± 8.5</td>
<td>0.079</td>
</tr>
<tr>
<td><strong>Dyspnea</strong></td>
<td>99 (75%)</td>
<td>4 (5%)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Chest pain</strong></td>
<td>31 (24%)</td>
<td>2 (2.5%)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Hemoptysis</strong></td>
<td>12 (9%)</td>
<td>0 (0%)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Syncope</strong></td>
<td>12 (9%)</td>
<td>0 (0%)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Heart rate (beats/min)</strong></td>
<td>117 ± 23</td>
<td>78 ± 18</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Systolic blood pressure (mm Hg)</strong></td>
<td>95±15</td>
<td>111±19</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Presence of shock</strong></td>
<td>31 (24%)</td>
<td>0 (0%)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Oxygen saturation (%)</strong></td>
<td>82±10</td>
<td>92±5</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>RDW</strong></td>
<td>14.58 ± 1.88</td>
<td>13.65 ± 0.66</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

Data are numbers or means ± SD. F: female; M: male, PE: Pulmonary embolism, RDW: red cell distribution (P < 0.05, nonparametric two independent samples).
Table 2. RDW, D-dimer, CTnl levels Wells and Genova (Clinical probability scores) score in PE severity groups determined on the according to PAOI.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mild (PAOI&lt;40%)</th>
<th>Moderate (PAOI=40-60%)</th>
<th>Severe (PAOI&gt;60%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>56</td>
<td>23</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>RDW</td>
<td>13.4 ± 0.97</td>
<td>14.5 ± 1.4</td>
<td>15.8 ± 2.0</td>
<td>&lt;0.0001*</td>
</tr>
<tr>
<td>Wells score</td>
<td>4.5 ± 2.4</td>
<td>5.1 ± 2.4</td>
<td>5.5 ± 2.0</td>
<td>0.04**</td>
</tr>
<tr>
<td>Genova score</td>
<td>4.6 ± 1.6</td>
<td>6.3 ± 1.6</td>
<td>6.8 ± 1.8</td>
<td>0.003**</td>
</tr>
<tr>
<td>D-Dimer</td>
<td>2323 ± 1075</td>
<td>3575 ± 1650</td>
<td>3400 ± 980</td>
<td>&lt;0.0001**</td>
</tr>
<tr>
<td>sPAP mmHg</td>
<td>37.3 ± 15.6</td>
<td>54.5 ± 18.2</td>
<td>59 ± 11.9</td>
<td>&lt;0.0001**</td>
</tr>
<tr>
<td>CTnlI</td>
<td>0.28 ± 0.47</td>
<td>0.40 ± 0.74</td>
<td>0.84 ± 1.85</td>
<td>&lt;0.0001**</td>
</tr>
<tr>
<td>RV/RL</td>
<td>1.03 ± 0.16</td>
<td>1.10 ± 0.14</td>
<td>1.28 ± 0.25</td>
<td>&lt;0.0001**</td>
</tr>
</tbody>
</table>

Data are numbers or means ± SD. RDW: red cell distribution width, sPAP: systolic pulmonary arterial pressure, PAOI: pulmonary artery obstruction index. PE: Pulmonary embolism, RV/LV: right ventricle/left ventricle, WBC: white blood cell

* Kruskal-Wallis test was used; P < 0.05

** Kruskal-Wallis test and Mann Whitney U test (Bonferroni correction to adjust for multiple comparisons) were used; P < 0.05

Bold defines P value of statistical significance of differences between massive and nonmassive (significance level <0.05)
<table>
<thead>
<tr>
<th>Variables</th>
<th>Mild (RV/LV &lt; 1)</th>
<th>Moderate (RV/LV =1-1.4)</th>
<th>Severe (RV/LV &gt;1.5)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>33</td>
<td>63</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>RDW</td>
<td>13.6 ± 1.3</td>
<td>14.8 ± 1.8</td>
<td>15.9 ± 2.4</td>
<td>&lt; 0.0001**</td>
</tr>
<tr>
<td>Wells Score</td>
<td>4.0 ± 2.2</td>
<td>5.3±2.1</td>
<td>5.6±2.0</td>
<td>0.012**</td>
</tr>
<tr>
<td>Genova Score</td>
<td>5.5 ± 1.8</td>
<td>6.5±1.7</td>
<td>6.6±1.7</td>
<td>0.029*</td>
</tr>
<tr>
<td>D-Dimer</td>
<td>2187 ± 1234</td>
<td>3352 ± 1268</td>
<td>2975 ± 1232</td>
<td>0.003**</td>
</tr>
<tr>
<td>sPAP mmHg</td>
<td>37.6 ± 12.3</td>
<td>53.1 ± 18.9</td>
<td>62.6 ± 7.2</td>
<td>&lt; 0.0001**</td>
</tr>
<tr>
<td>CTnI</td>
<td>0.73 ± 2.46</td>
<td>0.44 ± 1.05</td>
<td>0.34 ± 0.36</td>
<td>0.02**</td>
</tr>
</tbody>
</table>

Data are numbers or means ± SD. PE: Pulmonary embolism, RDW: red cell distribution width, sPAB: pulmonary artery pressure, PE: Pulmonary embolism, RV/LV: right ventricle/left ventricle, sPAP: systolic pulmonary arterial pressure.

*Kruskal-Wallis test was used; P < 0.05

**Kruskal-Wallis test and Mann Whitney U test (Bonferroni correction to adjust for multiple comparisons) were used; P < 0.05

Bold defines P value of statistical significance of differences between massive and nonmassive (significance level <0.05)
Table 4: Comparison of demographic data, laboratory data, PAOI, CTnI and sPAP data between massive and non-massive PE groups.

<table>
<thead>
<tr>
<th></th>
<th>Non-massive PE</th>
<th>Massive PE</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>64</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>F/M (n)</td>
<td>30/34</td>
<td>22/14</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Age (years)</td>
<td>61.6 ± 15.8</td>
<td>66.5 ± 12.6</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>RDW %</td>
<td>13.8 ± 1.36</td>
<td>15.7 ± 2.48</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>PAOI %</td>
<td>33.8±18</td>
<td>71.46 ± 20.17</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>sPAP mmHg</td>
<td>34.6 ± 10.3</td>
<td>62.2 ± 12.6</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>CTnI</td>
<td>0.9 ± 0.7</td>
<td>1.05 ± 2.18</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

*P value for any differences between

Bold defines P value of statistical significance of differences between massive and nonmassive between (significance level <0.05)

Data are numbers or means ± SD. CTnI: cardiac troponin I, F: female; M: male; PAOI: pulmonary artery obstruction index PE: Pulmonary embolism, RDW: red cell distribution width, sPAP: systolic pulmonary arterial pressure
### Table 5: Correlation between clinical probability scores, RDW, sPAP, d-dimer with PAOI

<table>
<thead>
<tr>
<th>Correlation with PAOI</th>
<th>Rho</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDW</td>
<td>0.658</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Wells Clinical Score</td>
<td>0.287</td>
<td>0.001</td>
</tr>
<tr>
<td>Genova Clinical Score</td>
<td>0.362</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>sPAP mmHg</td>
<td>0.635</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>cTnI</td>
<td>0.547</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>D-dimer</td>
<td>0.456</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

*P value for any differences between parameters and PAOI.

Bold defines P value of statistical significance of correlation between parameters and PAOI (significance value < 0.05)

cTnI: cardiac troponin I, PAOI: pulmonary artery obstruction index PE: Pulmonary embolism, RDW: red cell distribution width, sPAP: systolic pulmonary arterial pressure
Figure 1.  

154x78mm (600 x 600 DPI)

Figure 1: Correlation between Red cell distribution width (RDW) and Pulmonary artery obstruction Index (PAOI)
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