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Predicting Long-Term Mortality in Acute Pulmonary Embolism: The Role of RDW Levels

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ABSTRACT

Objective: Acute pulmonary embolism (PE) is a life-threatening cardiovascular emergency. PE long-term mortality data is scarce. Red cell distribution width (RDW) may predict PE mortality independently. This research examined the association between admission RDW and long-term mortality in acute PE patients.

Material and Methods: A prospective registry-based cohort design was used in this investigation. Included were a total of 115 discharged individuals with acute PEThe documentation of clinical features, test data, cardiovascular risk factors, and comorbidities was conducted for patients who were observed for a median of 140 months (range: 2-168) to determine the occurrence of cardiovascular mortality.

Results: The mean age of the patients was 62±16 years. After follow-up, 52 of 115 patients (45%) died from cardiovascular causes. Those who perished had greater RDW levels than those who survived [16.4±3.1 vs. 14.6±2.1%, p<0.001]. The optimum RDW threshold for predicting long-term mortality was >14.6% (AUC =0.704, 95% CI =0.608-0.808).

Conclusion: Long-term mortality in PE patients was likely associated with elevated RDW levels..

Keywords: pulmonary embolism, red cell distribution width, long-term mortality, risk stratification

INTRODUCTION

High mortality rates are associated with the cardiovascular emergency known as pulmonary embolism (PE). Short-term mortality is around 6.5% in the hospital, 7.4% after 30 days, and 6.8% after 3 months (1). Long-term mortality statistics for PE are few; however, one-year and five-year death rates of 13.6% and 26.7%, respectively, have been recorded (2). Moreover, determining prognostic indicators, monitoring these patients, and preventing both catastrophic and nonfatal consequences are crucial since PE patients have a significant risk of mortality at both the early and late stages of the disease (3). Biomarkers for PE's development, progression, and short- and long-term mortality have been suggested in abundance (4,5). They must, however, be simple to get, commonplace, and reliable.

Red cell distribution width (RDW), often known as a measure of the variability in the size of erythrocytes, is a practical measurement that is commonly included in routine blood cell counts. It is also inexpensive, readily available, dependable, and widely used (6). It has been explored as a possible independent predictor of morbidity and mortality in several illnesses and disorders, including heart failure (HF), pulmonary hypertension, and myocardial infarction, as well as for patients in intensive care units (6–8). RDW levels may be a reliable and independent predictor of a higher death risk among individuals with PE (9).

We demonstrated for the first time that RDW levels are connected with hemodynamic parameters and highly correlated with in-hospital mortality in patients with acute PE (10). In spite of this, the relationship between RDW and long-term mortality in persons with PE is poorly known. This study aimed to explore the correlation between admission RDW and long-term mortality in patients with PE.

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MATERIAL and METHODs

Patient selection

In the previous study (10), 21 of 136 hospitalized patients died due to hospital mortality. Including the remaining 115 participants in this prospective registry-based cohort study. Upon admission, we evaluated the patient's medical history and administered a lifestyle and risk factor questionnaire to each patient or immediate family member (if the patient was in a poor clinical condition). Six patients with chronic liver disease, five patients with chronic kidney disease, four patients with malignancy, eight patients who had been previously treated for anemia, six patients who had received erythrocyte suspension for any reason within the previous six months, and fifteen patients who were not diagnosed with acute PE on scintigraphy and/or tomography were excluded from the previous study (10). After obtaining informed consent in the previous investigation, the remaining 136 patients whose diagnoses of acute PE were confirmed by ventilation-perfusion scintigraphy and/or multislice spiral computed tomography based on guideline criteria (11) were enrolled in the present experiment (10). On average, patients were tracked for 140 months (range: 2-168 months). Hospital records, patient interviews (in person or over the phone), patient families, and primary care physicians were used to collect data on patient follow-up. The patients were separated into two groups: those who survived and those who did not. The study was conducted in accordance with the Helsinki Declaration's standards. The local ethics committee authorized the study.

Blood pressure >140/90 mmHg on >2 office measurements or treatment with antihypertensive medication is considered as hypertension. Blood glucose levels of more than 126 mg/dL or treatment with an anti-diabetic medication suggest diabetes. Coronary artery disease was indicated by a clinical history of coronary artery disease, abnormal stress test results with ischemia, or documented coronary stenosis >50%. The hypercholesterolemia was more than 200 mg/dl. During right ventricular (RV) loading, S1Q3T3, the right bundle branch block pattern, and the right precordial T-wave modifications were evaluated.

Biochemical measurements

At the time of admission, the RDW was determined using a Beckman Coulter Automatic CBC Analyzer (Beckman Coulter, Inc., Fullerton, California, USA). The reference range for RDW at our research facility is between 11.5% and 14.5%.

Echocardiography

All participating sites used the Vivid 7 system (GE Healthcare, Wauwatosa, Wisconsin, USA) with 2.5 to 5-MHz probes for admission echocardiograms. A modified Simpson approach calculated the ejection fraction. Contemporary chamber proportions were established (13). To assess RV dysfunction, echocardiography investigated RV dilatation, tricuspid regurgitation jet flow rate, and systolic pulmonary artery pressure. In accordance with the latest guideline (13), RV dimensions >3.4 cm at the basal plane or >3.8 cm at the midplane indicated RV dilatation (13). The minor axis from the right atrial lateral border to the interatrial septum assessed right atrial size (13). The recommendations proposed classifying valvular regurgitations as severe or non-severe based on color flow jet Doppler signal intensity and vena contracta width (14). Systolic pulmonary artery pressure was calculated before (15). A skilled sonographer reviewed unidentified transthoracic echocardiographic data at the main facility.

Statistical analysis

In the event of a non-normal distribution, continuous variables were given as mean±SD or median (range). Percentages represented categorical variables. The chi-square test, the t-test with independent samples, and the Mann-Whitney U test were used to compare patient groups. Spearman's correlation determined correlations. Long-term mortality connections were assessed using univariable regression analysis. Using a multivariate Cox proportional-hazards model with the backward stepwise technique, statistically significant variables and potential confounding factors, such as systolic and diastolic blood pressure and anemia, were used to assess long-term mortality prognostic factors. The cumulative survival curves of Kaplan-Meier revealed survival in two patient groups with a constant (group 1) or rising (group 2) distribution and categorical characteristics shown as percentages. Receiver-operating characteristic (ROC) curve analysis was used to find the RDW cut-off point with the highest sensitivity and specificity for long-term mortality prediction. All statistical calculations were performed using SPSS 22.0 (SPSS Inc., Chicago, IL, USA). The significance level was a two-sided $p < 0.05$.

RESULTs

The patients had a mean age of 62±16 years, with 50% male and 50% female. Patients were monitored for a duration of a median of 140 months (range: 2-168), during which time 52 patients (45%) passed away. The RDW level of >14.6% was shown to be the optimum cut-off point for predicting long-term death, with a sensitivity of 67% and a specificity of 70% (area under the curve =0.704, 95% confidence interval = 0.608–0.800, Figure 1).

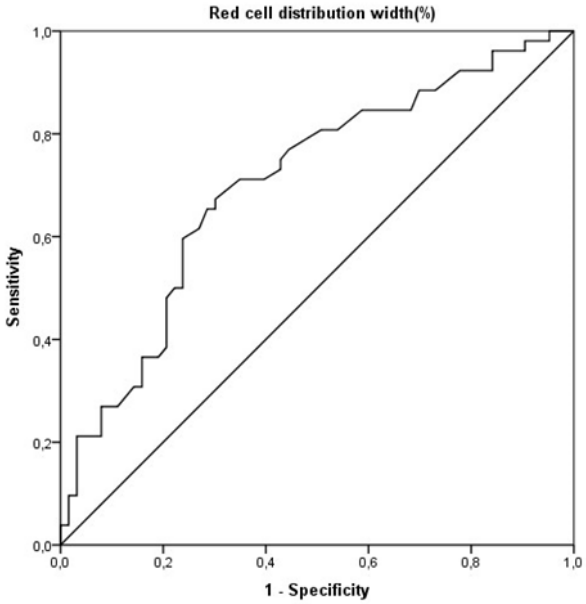

Figure 1. ROC curve for red cell distribution width to predict long term mortality

The patients were divided into two categories: those who successfully beat the illness and those who did not. The patients that passed away were of a more advanced age. Patients who passed away were more likely to have used thrombolytic agents and more likely to have a chronic obstructive pulmonary disease (COPD). This was in comparison to individuals who had lived. Patients who passed away were more likely to be female and to have had a history of surgery in the past, despite the fact that these factors did not reach statistical significance (Table 1).

Table 1. Baseline parameters of study patients

Variable	All patients (n=115)	Patients who survived (n=63)	Patients who died (n=52)	p
Mean age (years)	62±16	56±16	70±1	< 0.001
Women	57 (50%)	36 (57%)	21 (40%)	0.074
Admission symptoms				0.776
Dyspnea	78 (68%)	41 (65%)	37 (71%)	0.622
Chest pain	36 (31%)	22 (35%)	14 (27%)	0.472
Hemoptysis	4 (4%)	2 (3%)	2 (4%)	1.000
Syncope	10 (9%)	5 (8%)	5 (10%)	0.753
Symptom duration (hours)				0.478
< 6	6 (5%)	3 (5%)	3 (6%)	1.000
6-12	7 (6%)	2 (5%)	5 (10%)	0.241
12-24	26 (23%)	16 (25%)	10 (19%)	0.574
> 24	76 (66%)	42 (67%)	34 (65%)	1.000
Hypertension	45 (39%)	21 (33%)	24 (46%)	0.226
Diabetes mellitus	25 (22%)	17 (27%)	8 (15%)	0.203
Coronary artery disease	26 (24%)	11 (18%)	15 (31%)	0.153
Chronic obstructive pulmonary disease	16 (14%)	4 (7%)	12 (24%)	0.018
Immobilization	20 (18%)	13 (21%)	7 (14%)	0.478
Previous history of pulmonary embolism	8 (7%)	8 (8%)	3 (6%)	0.730
Previous history of deep venous thrombosis	6 (5%)	4 (7%)	2 (4%)	0.690
Previous history of surgery	24 (21%)	18 (29%)	6 (12)	0.051
Length of stay (days)	11±7	12±6	11±8	0.483
Usage of thrombolytic agent	10 (9%)	2 (3%)	8 (15%)	0.041
Cardiopulmonary resuscitation	4 (4%)	1 (2%)	3 (6%)	0.327

Table 2. Comparison of patients with acute pulmonary embolism grouped into two categories according to long term mortality along with current hemodynamic, electrocardiography, echocardiography, and laboratory findings

Variable	All patients (n=115)	Patients who survived (n=63)	Patients who died (n=52)	p
Hemodynamic findings				
Heart rate (beats/minute)	107±20	101±20	112±19	0.009
Systolic blood pressure (mm Hg)	108±17	108±19	107±15	0.800
Diastolic blood pressure (mmHg)	67±15	69±15	65±13	0.394
Presence of SHOCK	10 (9%)	4 (6%)	6 (12%)	0.344
Oxygen saturation (%)	87±9	88±8	85±9	0.167
Electrocardiography parameters				
Atrial fibrillation	23 (21%)	8 (13%)	15 (31%)	0.040
Right bundle branch block	25 (23%)	14 (23%)	11 (23%)	1.000
S1Q3T3	19 (17%)	9 (15%)	10 (20%)	0.599
T wave changes	37 (34%)	19 (31%)	18 (37%)	0.679
Echocardiography parameters				
Left ventricle ejection fraction (%)	55±10	56±8	52±11	0.052
Right ventricular dilatation/hypokinesia	63 (68%)	30 (58%)	33 (81%)	0.035
Severe tricuspid regurgitation	48 (53%)	21 (42%)	27 (69%)	0.015
Systolic pulmonary artery pressure (mmHg)	47±19	41±15	55±22	0.001
Laboratory findings				
Red cell distribution width (mean±SD) (%)	15.4±2.8	14.6±2.1	16.4±3.1	< 0.001
Red cell distribution width > 14.6%	54 (47%)	19 (30%)	35 (67%)	< 0.001
Hemoglobin (gr/dl)	13±2.0	13.5±2.0	12.9±1.9	0.793
Presence of anemia	43 (37%)	22 (35%)	21 (40%)	0.682
Troponin I (ng/mL)	0.14±0.3	0.2±0.5	0.07±0.1	0.371
Glomerular filtration rate (mL/min/1.73 m2)	74±26	81±24	66±25	0.001
Spiral computerized tomography findings				0.156
Main pulmonary artery involvement	6 (6%)	3 (5%)	3 (6%)	1.000
Main pulmonary artery branch involvement	64 (58%)	32 (52%)	32 (67%)	0.164
Main pulmonary artery segmental involvement	50 (46%)	33 (53%)	17 (35%)	0.095
Main pulmonary artery sub-segmental involvement	17 (16%)	13 (21%)	4 (8%)	0.121
Deep venous thrombosis	41 (36%)	21 (34%)	20 (39%)	0.695

Table 2 provides a comparison of the present hemodynamic, electrocardiographic, echocardiographic, and laboratory data between the two groups of patients with acute PE based on long-term mortality. Those who passed away had significantly greater levels of RDW than those who made it [16.4±3.1 vs. 14.6±2.1%, p<0.001]. Patients who did not make it to the end had lower baseline RDW levels (14.6%) than those who did [35 (67%), vs. 19 (30%, p<0.001]. Patients who did not make it to hospital discharge often had atrial fibrillation (AF), right ventricular dilatation/hypokinesia, and significant tricuspid regurgitation. Those who did not survive had a higher heart rate, elevated systolic pulmonary artery pressure, decreased left ventricular ejection fraction, and impaired glomerular filtration rate. Neither group differed significantly from the other in terms of demographics or laboratory measurements taken at the outset (**Table 2**).

Patients with acute PE had a positive correlation with heart rate and systolic pulmonary artery pressure as well as a negative correlation with systolic and diastolic blood pressure, left ventricular ejection fraction, and hemoglobin levels. The RDW level was not associated with any of the other test results (p>0.05, **Table 3**).

Long-term mortality analysis results from univariate and multivariate Cox proportional hazards models are shown in Table 4. In univariate analysis, prognostic significance was found for right ventricular dysfunction, age, female gender, history of surgery, chronic obstructive pulmonary disease, atrial fibrillation, thrombolytic agent use, anemia, severe tricuspid regurgitation, right ventricular dilatation/hypokinesia, heart rate, left ventricular ejection fraction, systolic pulmonary artery pressure, and glomerular filtration rate.

Multiple Cox regression analysis using the backward stepwise technique revealed that RDW, age, heart rate, systolic pulmonary artery pressure, and right ventricular dilatation/hypokinesia were independently connected to an increased mortality risk (**Table 4**).

After applying a cutoff of 14.6%, the Kaplan-Meier analysis produced survival curves that were significantly different for the two previously described subgroups of RDW (p <0.001, **Fig. 2**).

Table 3. Spearman's correlation coefficients for Red cell distribution width

	Red cell distribution width, %	P value
Heart rate (beats/minute)	0,242	0,023
Systolic blood pressure (mm Hg)	-0,257	0,022
Diastolic blood pressure (mmHg)	-0,243	0,031
Left ventricle ejection fraction (%)	-0,272	0,008
Systolic pulmonary artery pressure (mmHg)	0,374	< 0.001
Presence of chronic obstructive pulmonary disease	0,261	0,005
Presence of atrial fibrillation	0,359	< 0.001
Hemoglobin (gr/dl)	-0,281	0,002
Presence of anemia	0,315	0,001

Table 4. Univariate and multivariate analyses of long term mortality

	Univariate			Multivariate		
Variable	**p**	**HR**	**(95% CI)**	**p**	HR	(95% CI)
Red cell distribution width	< 0.001	1,212	1.111-1.322	0,016	1,158	1.027-1.306
Age (years)	< 0.001	1,064	1.038-1.090	< 0.001	1,09	1.043-1.144
Women	0,084	1,63	0.936-2.838			
Chronic obstructive pulmonary disease	0,013	2,303	1.194-4.441			
Previous history of surgery	0,048	2,367	1.008-5.558			
Usage of thrombolytic agent	0,026	2,363	1.109-5.034			
Hemodynamic findings						
Heart rate (beats/minute)	0,01	1,019	1.005-1.035	0,012	1,037	1.008-1.066
Systolic blood pressure (mm Hg)	0,696	0,996	0.978-1.015			
Diastolic blood pressure (mmHg)	0,286	0,988	0.966-1.010			
Electrocardiography parameters						
Atrial fibrillation	0,017	1,451	1.069-1.970			
Echocardiography parameters						
Left ventricle ejection fraction (%)	0,011	0,966	0.940-0.992			
Severe tricuspid regurgitation	0,006	2,623	1.326-5.186			
Systolic pulmonary artery pressure (mmHg)	< 0.001	1,032	1.017-1.047	0,006	1,047	1.013-1.082
Right ventricular dilatation/hypokinesia	0,016	2,591	1.195-5.618	0,025	10,895	1.344-88.293
Laboratory findings						
Glomerular filtration rate (mL/min/1.73 m2)	<0.001	0,98	0.969-0.991			
Presence of anemia	0,301	1,34	0.769-2.333			

All the variables from Table 1-2 were examined, and only those significant at a P<0.1 and those with a correlated RDW level are shown in univariate analysis. The multiple Cox regression analysis included all univariate predictors and those correlated with RDW levels. HR: Hazard ratio, CI: Confidence interval.

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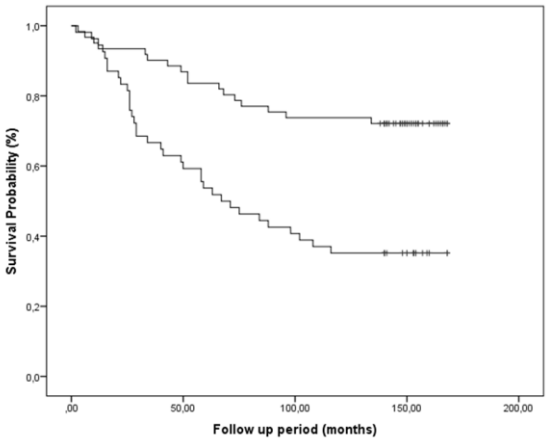

Figure 2. Kaplan-Meier Curve for long term mortality.

DISCUSSION

Acute PE is a significant cardiovascular issue with respect to both early and late consequences. As critical as identifying these individuals is the subsequent monitoring of complications. Reperfusion damage in the lung caused by pulmonary embolism increases oxidative stress, the myeloperoxidase enzyme, and reactive oxygen radicals (16). In addition, severe hypoxia resulting from pulmonary artery occlusion may boost neurohormone and adrenergic system activity and trigger the release of inflammatory cytokines (16). In addition, PE may cause RV dysfunction through cardiac inflammation that adds to myocyte damage (5). Based on coagulation-fibrinolysis processes, immune response mechanisms, and oxidative stress, the majority of traditional PE biomarkers for predicting mortality were examined. Biochemical biomarkers have been proposed for use in risk stratification and prognosis prediction in PE. A reliable predictor may significantly improve PE diagnosis, follow-up, and long-term consequences. N-terminal proB-type natriuretic peptide (NT-proBNP), D-dimer, and troponin I was previously used to predict the long-term prognosis of PE patients (4). Increased levels of NT-proBNP, D-dimer, and troponin I have been linked to worse outcomes in patients with PE (4). Furthermore, C-reactive protein (CRP) may be utilized to evaluate the risk and prognosis of other illnesses, including PE. Abul et al. (5) showed that CRP is related to right ventricular dysfunction, a prognostic indicator for PE.

Red cell distribution width is one of the parameters measured during a standard complete blood count. Even within the normal reference range, a high RDW was substantially related to an elevated risk of all diseases (17). Many disorders, including heart failure (HF) and pulmonary hypertension (PH), have been associated with a substantial link between an increase in RDW and unfavorable outcomes (8, 18). Recent research has shown a correlation between RDW and PE's prediction, severity, and prognosis. (19) While the association between RDW and PE is unclear, PE is a complicated cardiac illness with several pathways that may contribute to an increase in RDW. Increases in inflammatory cytokines, inhibition of bone marrow function, reductions in erythrocyte maturation, and increases in oxidative stress are the initial causes of increased RDW levels in PE (20). Second, significant hypoxia may occur owing to vasoconstriction of the pulmonary arteries after PE, leading to activation of the neurohormonal and adrenergic systems and an increase in RDW with the release of inflammatory cytokines (10). There is a high correlation between RDW levels and coagulation, but it is believed to be linked to prothrombotic disorders such as PE (19). In this regard, Lippi et al. (21) discovered a substantial, progressive correlation between RDW and well-known inflammatory indicators such as CRP and erythrocyte sedimentation rate (ESR), irrespective of other confounding variables. While many studies (9, 10, 22, and 23) have evaluated the association between RDW and short-term mortality in patients with PE and found significant findings, there are insufficient data on the long-term mortality between RDW and PE. In a multivariate Cox proportional-hazards model, admission RDW >14.6% was linked with acute PE-related early mortality (10). Ozsu et al. (9) found that an optimum RDW value of 15% predicted in-hospital mortality. In multivariate analysis, RDW ~15% predicted death 1.2-fold better. RDW independently predicted mortality in 199 PE patients (24). RDW <16% predicted 30-day PE mortality, according to Zhou et al. (23). After controlling for the PE severity index score and other variables, Jurin et al. (22) found that a higher RDW was related to increased mortality.

The connection between RDW and long-term mortality in PE patients is poorly understood. Sen et al. (25) found that RDW levels were predictive of death within 100 days after a diagnosis of PE. According to multivariate regression analysis, the mortality rate from PE was 4.08-fold higher in patients with RDW. (25) Predicting six-month mortality in patients with PE using an RDW cut-off value of 15.2% was shown to be sensitive to 73.3% and specific to 73.2% by Kucuk et al. (20). After controlling for other factors, Kheirkham-Sabetghadam et al. (26) discovered a statistically significant trend linking RDW to long-term (median duration of 17 months) death. Our research examined whether RDW was associated with increased mortality in PE patients over the long term. Long-term mortality in individuals with PE was significantly associated with RDW after a mean follow-up of 140 months. Long-term mortality in patients with PE was predicted with a sensitivity of 67% and a specificity of 70% using an RDW cut-off value of >14.6%. We also reported the correlation between higher mortality and heart rate, systolic pulmonary artery pressure, and right ventricular dilatation/hypokinesia in individuals with PE. There were a few issues with our research. First, the number of patients was somewhat low since the long-term mortality research was only performed on those patients who had survived the first investigation. Validation of our findings requires further research that is prospective, multicentric, and includes a substantial number of patients.. In our research, well-known inflammatory indicators like ESR and CRP did not get the attention they deserved. In addition, it was not possible to evaluate BNP levels, which are used as prognostic indicators for pulmonary embolism.

CONCLUSION

In conclusion, the present research findings indicate that a high RDW level may be beneficial as a useful independent predictor of long-term mortality after PE. RDW, in contrast to a number of other inflammatory indicators, is a very affordable and easily accessible marker that, in addition to traditional risk scores, has the potential to provide further risk stratification.

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Ethical approval: All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and/or with the Helsinki Declaration of 1964 and later versions.

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