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Association between RDW and stent thrombosis in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention

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Abstract

Stent thrombosis is a rare but potentially fatal complication of percutaneous coronary interventions (PCIs). In recent years, the predictive and prognostic value of the red cell distribution width (RDW) as an indicator of inflammation has been shown in many cardiovascular diseases. Aim of this study was to examine the predictive value of RDW for stent thrombosis in patients who underwent successful stent implantation for ST-elevation myocardial infarction (STEMI).

In this retrospective study, 146 patients who underwent successful PCI to native coronary artery due to STEMI previously and presented with acute coronary syndrome with stent thrombosis were included (stent thrombosis group). A total of 175 patients who had similar procedural characteristics (type, diameter, and length of stent) and not had stent thrombosis were consisted control group.

Patients were divided into tertiles according to the admission RDW values (12.9±0.4, 14.2±0.4, and 16.3±1.5, respectively). Stent thrombosis developed in 47 (40.9%) patients in the lowest tertile, 39 (37.9%) patients in mid tertile, and 60 (58.3%) patients in the highest tertile ($P=0.006$). Female gender ratio was statistically significantly higher in the 3rd tertile (13 [11.3%], 8 [7.8%], 24 [23.3%], $P=0.003$, respectively). RDW (OR: 1.397 [95% CI 1.177–1.657], $P<0.001$) and platelet count (OR: 1.008 [95% CI 1.004–1.012], $P<0.001$) remained independent predictors of stent thrombosis after multivariate logistic regression analysis. ROC curve analysis demonstrated that, admission RDW values higher than 13.9 can predict the development of stent thrombosis with a sensitivity of 57% and a specificity of 52% (The area under the ROC curve: 0.59 [95% CI 0.53–0.65] $P=0.007$).

High RDW values found to be independently associated with the development of stent thrombosis in patients with STEMI.

Abbreviations: LDL = low-density lipoprotein, PCI = percutaneous coronary intervention, RDW = red cell distribution width, STEMI = ST-elevation myocardial infarction.

Keywords: myocardial infarction, percutaneous coronary intervention, red blood cell distribution width, stent thrombosis

1. Introduction

Targets of treatment in patients with ST-elevation myocardial infarction (STEMI) are alleviating the ischemic symptoms, preventing the complications, and restoring the coronary blood flow.[1] Fibrinolysis and primary percutaneous coronary intervention (PCI) are the reperfusion therapies in STEMI.[1] Primary PCI is a safe, effective, and preferred treatment option for maintaining reperfusion.[1]

Stent implantation is an important milestone in the treatment of ischemic heart disease in interventional cardiology era.[2] Stent implantation was first performed in 1986 and after this time elastic recoil, coronary dissection, and thrombosis due to coronary angioplasty have been prevented with stenting.[3] However, it may cause new problems like occlusion of side branches, stent thrombosis, and restenosis.[4]

Despite major advances in interventional techniques and anticoagulan-antiagregan therapies, stent thrombosis remains a major problem in interventional cardiology.[4] Incidence of stent thrombosis has been reported between 1.4% and 4.4% in patients with acute myocardial infarction and undergoing PCI with stenting.[5] Reasons of stent thrombosis are resistance to aspirin and/or clopidogrel, insufficient anticoagulation, type of stent use (bare metal, drug eluting, and long stents), presentation with acute coronary syndrome, characteristics of coronary lesions and vessel, procedural causes (stent apposition), and inadequate endothelization after stenting.[6]

Red cell distribution width (RDW) is a quantitative measurement of variability and size of erythrocytes.[7] Higher RDW values have been reported with worse prognosis in patients with coronary artery disease, acute myocardial infarction, stroke, acute and chronic heart failure, pulmonary hypertension, and

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acute pulmonary embolism.[7–12] Pathophysiology of elevated RDW in cardiovascular, pulmonary, and thrombotic diseases has not clearly explained yet. However, it has been thought that increased cytokines may inhibit maturation of erythrocytes in bone morrow and may cause increased RDW values in cardiovascular diseases.[7]

In this study, we aimed to investigate the relationship between preprocedural RDW values in patients who underwent PCI and stenting due to STEMI and development of stent thrombosis during follow-up.

2. Methods

We have retrospectively analyzed the data of 146 patients who previously underwent primary PCI with stenting due to STEMI and presented with acute coronary syndrome and detected stent thrombosis during coronary angiography (stent thrombosis group) between 2009 and 2013 in a high volume tertiary center. A total of 175 patients who underwent PCI with stenting due to STEMI before and underwent coronary angiography other than reason of acute coronary syndrome (refractory angina, abnormal treadmill stress test, etc.) and had similar procedural characteristics (type, diameter, and length of stent) but not stent thrombosis consisted the control group. The local ethics committee approved the study protocol.

Patients with decompensated heart failure, cardiogenic shock, severe arrhythmia, chronic obstructive pulmonary disease, history of stent thrombosis, intervention to left main vessel and complex interventions, creatinine >2 mg/dL, hemoglobin < 12 mg/dL, blood transfusion within 3 months, active infection, chronic inflammatory and rheumatic diseases, malignancy, and cirrhosis were excluded from the study.

We have accessed the data of demographic and clinical features of patients via hospital records. Hematological and biochemical data of patients were obtained from the results of preprocedural venous blood sample analyses retrospectively. RDW values were calculated with using an automatic analyzing machine (Abbott Cell-Dyn 3700; Abbott Laboratory, Abbott Park, IL). Other biochemical parameters were calculated with Standard techniques. Patients were divided into tertiles according to the admission RDW values (12.9 ± 0.4, 14.2 ± 0.4, and 16.3 ± 1.5, respectively). Angiographic data including type, diameter, length, and localization of stent were obtained from coronary angiography records.

STEMI is a clinical syndrome defined by characteristics symptoms of myocardial ischemia with persistent electrocardiographic ST elevation and subsequent release of biomarkers of myocardial necrosis. Diagnostic ST elevation in the absence of left ventricular hypertrophy or left bundle-branch block is defined by the European Society of Cardiology/American Heart Association/World Heart Federation Task Force for the Universal Definition of Myocardial Infarction as new ST elevation at the J point in at least 2 contiguous leads of ≥2 mm (0.2 mV) in men or ≥1.5 mm (0.15 mV) in women in leads V_2–V_3 and/or of ≥1 mm (0.1 mV) in other contiguous chest leads or the limb leads.[13]

Time between stent implantation and control coronary angiography due to acute coronary syndrome was calculated and stent thrombosis was defined as acute (between 24 hours), subacute (24 hours–30 days), late (30 days–1 year), and very late (after 1 year) thrombosis.[14] Stent thrombosis was defined as presence of thrombus inside of stent or 5 mm apart from stent whether it cause occlusion or not.[14]

Analyses were performed using SPSS 15.0 (SPSS, Inc., Chicago, IL). Continuous data with normal distribution were presented as mean and standard deviation, and categorical variables were expressed as percentages. The independent sample *t* test or the Mann–Whitney *U* test was used for the continuous variables and the chi-square test for categorical variables. Multivariate logistic regression analysis was used to determine the independent predictors of stent thrombosis. A receiver-operating characteristics (ROCs) curve analysis was performed to identify the optimal cut-off point of RDW to predict stent thrombosis. Results were presented with odds ratio (OR), 95% confidence interval and *P* value. $P < 0.05$ was accepted as statistically significant.

3. Results

Mean duration of follow-up was 16.9 ± 8.7 months. Mean ages of patients in stent thrombosis group and control group were similar (56.3 ± 10.7 vs 54.9 ± 11.8, respectively; $P = 0.253$). There were more hypertensive patients in stent thrombosis group (47% vs. 34%, respectively; $P = 0.025$). However, total cholesterol (167 ± 40 vs 180 ± 35, respectively; $P = 0{,}002$) and low-density lipoprotein (LDL) cholesterol levels (101 ± 36 vs 113 ± 30, respectively; $P = 0.002$) were significantly lower in stent thrombosis group when compared to control group. There was no significant difference in terms of sex, diabetes mellitus, smoking, triglyceride, and high-density lipoprotein cholesterol levels between groups (Table 1).

Figure 1 shows the distribution map of RDW levels of whole group, and Fig. 2 shows the distribution map of RDW levels

Table 1

Clinical and hematological characteristics of patients according to stent thrombosis.

Variables	Stent thrombosis mn = 146	Control n = 175	*P*
Age, years	56.3 ± 10.7	54.9 ± 11.8	0.253
Male sex	120 (82%)	156 (89%)	0.074
Hypertension	68 (47%)	60 (34%)	0.025
Diabetes mellitus	37 (25%)	42 (24%)	0.781
Smoking	61 (42%)	82 (47%)	0.362
Triglycerides, mg/dL	153 ± 95	151 ± 94	0.861
Total cholesterol, mg/dL	167 ± 40	180 ± 35	0.002
HDL-cholesterol, mg/dL	37 ± 16	38 ± 9	0.725
LDL-cholesterol, mg/dL	101 ± 36	113 ± 30	0.002
Occluded coronary artery			
Left anterior descending	87 (60%)	85 (49%)	
Circumflex	16 (11%)	30 (17%)	0.105
Right	43 (29%)	60 (34%)	
Stent type			
Bare metal	125 (86%)	155 (89%)	0.430
Drug-eluting	21 (14%)	20 (11%)	
Stent diameter, mm	2.96 ± 0.44	3.04 ± 0.44	0.122
Stent length, mm	19 ± 7	19 ± 6	0.934
Hemoglobin, g/dL	14.21 ± 1.40	14.35 ± 1.27	0.342
Red cell distribution width	14.8 ± 2.0	14.1 ± 1.2	0.007
Platelet count, $\times 10^9$/L	281 ± 80	244 ± 60	<0.001
Mean platelet volume, fL	8.5 ± 1.4	8.5 ± 1.2	0.901
White blood cell count, $\times 10^9$/L	11.93 ± 3.52	12.14 ± 4.13	0.626
Neutrophil count, $\times 10^9$/L	8.85 ± 3.64	8.97 ± 4.01	0.784
Lymphocyte count, $\times 10^9$/L	2.10 ± 0.93	2.21 ± 1.25	0.388
Neutrophil to lymphocyte ratio	5.54 ± 5.07	5.52 ± 4.41	0.970

Values are mean ± SD or n (%). HDL = high-density lipoprotein, LDL = low-density lipoprotein, RDW = red cell distribution width, SD = standard deviation.

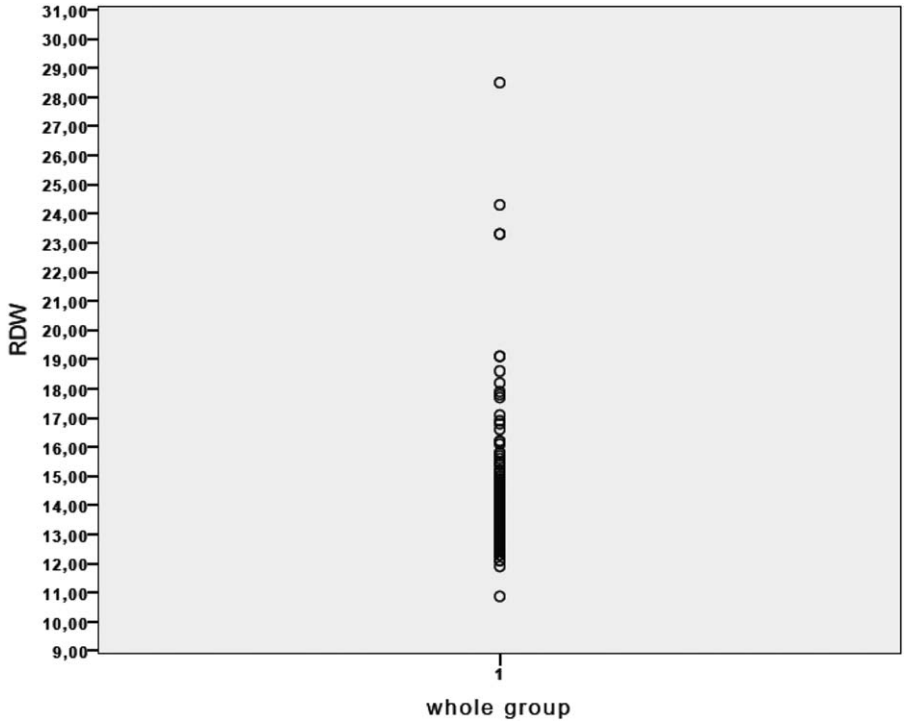

Figure 1. Distribution maps of the red cell distribution width (RDW) values of all patients in the study.

according the presence of stent thrombosis. RDW levels were significantly higher in stent thrombosis group compared to control group (14.8 ± 2.1 vs 14.1 ± 1.2, respectively; $P = 0.007$). Similarly mean platelet count was also significantly higher in stent thrombosis group (281 ± 80 vs 244 ± 60, respectively; $P < 0.001$). There were no significant difference in neutrophil to lymphocyte ratio, mean platelet volume, hemoglobin, and white blood cell count between groups (Table 1). Type of implanted stent (bare metal, drug eluting), location, diameter, and length of stent were similar between groups.

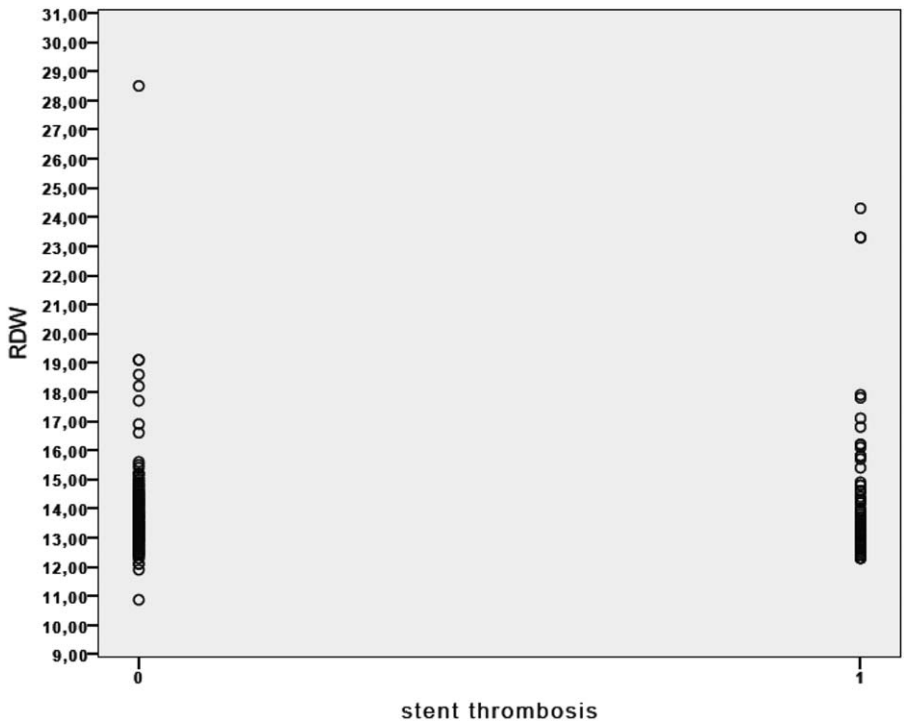

Figure 2. Distribution maps of the red cell distribution width (RDW) values of the patients according to presence of stent thrombosis.

Table 2

Clinical and hematological characteristics of patients according to RDW tertiles.

Variables	RDW Tertile 1	RDW Tertile 2	RDW Tertile 3	*P*
	12.9±0.4 (n=115)	14.2±0.4 (n=103)	16.3±1.5 (n=103)	
Age, years	52.5±10.5	53.8±10.3	60.7±10.5	<0.001
Male sex	102 (89%)	95 (92%)	79 (77%)	0.003
Hypertension	43 (37%)	42 (41%)	43 (42%)	0.786
Diabetes Mellitus	29 (25%)	23 (22%)	27 (26%)	0.797
Smoking	47 (41%)	47 (46%)	49 (48%)	0.589
Glucose, mg/dL	126±66	119±47	143±69	0.018
Creatinine, mg/dL	0.88±0.23	0.86±0.23	0.88±0.23	0.767
Triglycerides, mg/dL	156±97	149±95	152±92	0.854
Total cholesterol, mg/dL	178±38	173±36	171±40	0.439
HDL-cholesterol, mg/dL	39±13	38±15	36±9	0.259
LDL-cholesterol, mg/dL	109±34	107±33	106±35	0.874
Occluded coronary artery				
Left anterior descending	55 (48%)	63 (61%)	54 (52%)	
Circumflex	17 (15%)	15 (15%)	14 (14%)	0.289
Right	43 (37%)	25 (24%)	35 (34%)	
Stent type				
Bare metal	112 (97%)	97 (94%)	71 (69%)	<0.001
Drug-eluting	3 (3%)	6 (6%)	32 (31%)	
Stent diameter, mm	2.97±0.46	3.05±0.46	2.99±0.39	0.409
Stent length, mm	18±5	18±6	22±7	<0.001
Stent thrombosis	47 (41%)	39 (38%)	60 (58%)	0.006
Type of stent thrombosis				
Acute	6 (13%)	9 (23%)	11 (18%)	
Subacute	16 (34%)	9 (23%)	22 (37%)	0.520
Late and very late	25 (53%)	21 (54%)	27 (45%)	
Hemoglobin, g/dL	14.4±1.4	14.5±1.2	14.0±1.4	0.006
Platelet count, $\times 10^9$/L	264±72	252±55	265±86	0.344
Mean platelet volume, fL	8.7±1.4	8.6±1.2	8.2±1.2	0.025
White blood cell count, $\times 10^9$/L	12.8±4.3	11.6±3.6	11.7±3.5	0.040
Neutrophil count, $\times 10^9$/L	9.8±4.3	8.2±3.7	8.6±3.2	0.006
Lymphocyte count, $\times 10^9$/L	2.1±1.3	2.3±1.1	2.1±0.9	0.350
Neutrophil to lymphocyte ratio	6.5±4.9	5.9±5.1	4.9±2.7	0.022

Values are mean±SD or n (%). HDL=high-density lipoprotein, LDL=low-density lipoprotein, RDW=red cell distribution width, SD=standard deviation.

Patients were divided into 3 tertiles according to baseline RDW values (12.9±0.4, 14.2±0.4, and 16.3±1.5, respectively). There was significant difference in terms of age and sex between tertiles (Table 2). Patients in the 3rd tertile were older and there were more woman. Blood glucose levels were significantly higher in 3rd tertile when compared to other tertiles (52.5±10.5 vs 53.8±10.3 vs 60.7±10.5, respectively; $P=0.018$). Hemoglobin (14.4±1.4 vs 14.5±1.2 vs 14.0±1.4, respectively; $P=0.006$) and mean platelet volume (8.7±1.4 vs 8.6±1.2 vs 8.2±1.2, respectively; $P=0.025$) were decreasing when going from lowest to highest tertile.

Rate of stent thrombosis in 3rd tertile was significantly higher than other tertiles (41% vs 38% vs 58%, respectively; $P=0.006$) (Fig. 3). There was no significant difference about type of stent thrombosis between tertiles (acute, subacute, late, and very late). Similarly there was no difference in terms of coronary artery where stent implanted, and diameter of stents between tertiles (Table 2). However, there was significant difference in lengths of stents and types of stents. Implanted stents in tertile 3 were more longer (18.5±5 vs 18±6 vs 22±7, respectively; $P<0.001$), and the rate of drug eluting stents was more higher (3% vs 6% vs 32%, respectively; $P<0.001$) (Table 2).

After multivariate logistic regression analysis, RDW and platelet count remained significant predictor of stent thrombosis (OR: 1.397, 95% CI: 1.177–1.657, $P<0.001$; and OR: 1.008, 95% CI: 1.004–1.012, $P<0.001$, respectively) (Table 3).

ROC curves explored the relation between preprocedural RDW and stent thrombosis. Using a cut point of 13.9, preprocedural RDW predicted development of stent thrombosis with a sensitivity of 57% and specificity of 52% (ROC area under curve: 0.59, 95% CI: 0.53–0.65, $P=0.007$).

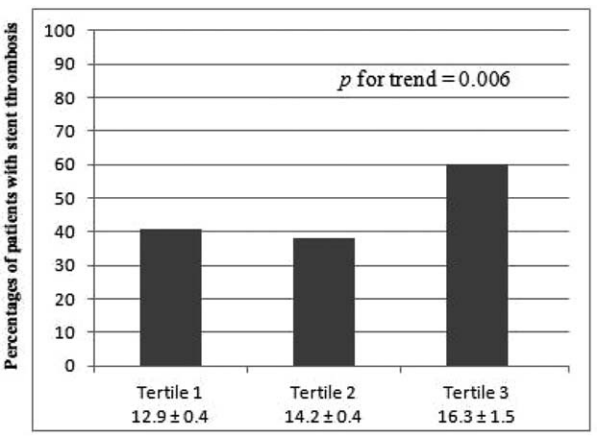

Figure 3. Percentage of patients developing stent thrombosis stratified by tertile of preprocedural red cell distribution width (RDW).

Table 3

Multivariate logistic regression analysis to assess predictors of stent thrombosis.

Variables	Odds ratio (%95 CI)	P
RDW	1.397 (1.177–1.657)	<0.001
Platelet count	1.008 (1.004–1.012)	<0.001
LDL cholesterol	0.991 (0.977–1.005)	0.192
Total cholesterol	0.998 (0.986–1.010)	0.719
Hypertension	1.525 (0.927–2.509)	0.097

CI = confidence interval, LDL = low-density lipoprotein, RDW = red cell distribution width.

4. Discussion

Our study has some fundamental results. First, RDW was significantly higher in stent thrombosis group compared to control group. Second, RDW was found to be independent predictor of stent thrombosis in multivariate logistic regression analyses. Third, rate of stent thrombosis was more higher in 3rd tertile where the patients had the highest mean RDW values.

RDW has emerged as a new risk marker in patients with cardiovascular diseases. High RDW shows expected anisocytosis in nutritional insufficiency, iron, folic acid and vitamin B12 deficiency, chronic liver disease, and blood transfusion.[15,16] It has been thought that chronic inflammation may shorten the half-life of erythrocytes, change the membrane characteristics, and cause to increase of RDW values.[17]

It has been demonstrated in several studies that there is a significant association between prognosis of acute and chronic ischemic heart diseases and RDW values.[7,8,12] Pathophysiological mechanism of relationship between RDW and cardiovascular diseases is not clear yet. Possible mechanisms may be oxidative stress, inflammation, and activation of neurohumoral system.[18–20] C-reactive protein is a well-known marker of cardiovascular diseases, and it has been shown that there was a significant correlation between RDW and C-reactive protein values.[19,21] In a study conducted in 7556 healthy volunteers by Zalawadiya et al,[8] there was strong correlation between RDW values and 10 year Framingham risk score. This correlation was valid after the adjustment of hemoglobin, vitamin B12, folic acid, ferritin, glomerular filtration rate, and body mass index.[8]

We have found significantly higher RDW values in stent thrombosis group, and RDW was found to be an independent predictor of stent thrombosis. Sangoi et al[22] showed that RDW was an independent predictor of in-hospital mortality in 109 patients with acute myocardial infarction. Acet et al[23] demonstrated that there was significant strong correlation between Global Registry of Acute Coronary Events score and RDW values in 800 STEMI patients. Uyarel et al[12] also showed an association between RDW values at presentation and in-hospital and long-term mortality in 2506 STEMI patients who underwent primary PCI. All of these studies have shown the relationship between RDW and worse prognosis in patients with cardiovascular diseases. However, the cause of worse prognosis is not explained yet. There was a significant correlation between RDW and stent thrombosis in our study. Higher RDW values at presentation may be explained with increased ischemia, oxidative stress, neurohumoral activation, and inflammation, and all of these causes may be the reason of stent thrombosis in our study. There was no significant difference about type of stent, diameter, and length of stent, and these similarities exclude the procedural causes of early and late stent thrombosis.

There were no differences in terms of age, diabetes, smoking, triglycerides, and high-density lipoprotein levels between stent thrombosis and control group. There were more hypertensives in stent thrombosis group compared to control group. However, total cholesterol and LDL cholesterol levels were significantly lower in stent thrombosis group, and this need to be explained. Effect of high dose statin in preventing stent thrombosis was shown before.[24] Although we do not have the data about the use of medications, higher preprocedural total cholesterol, and LDL cholesterol levels in stent thrombosis group may be due to nonaggressive use of statins in this group. However, this issue does not go beyond the hypothesis.

In analyses according to RDW tertiles, there were more older and female patients however lower hemoglobin levels in highest tertile. Similar results about the relationship between higher RDW values and female predominancy were shown before.[25,26] This may be explained by increased rate of anemia in female patients.[27] Anemia is known risk factor to cause increase in RDW values.[16] Although many parameters put into the logistic regression analyses, only RDW and platelet count emerged as an independent predictors of stent thrombosis. Stent diameter type and length have been proven to have effects on stent hrombosis.[28] Similarities of type, diameter, and length of stents between groups are explained by the study design before patient selection.

5. Limitations

Our study has several limitations. First it was a retrospective study. The most important limitation of our study is the lack of data about medications that had been taken by patients during the follow-up. Another limitation and a negative impact of our study is we could not find a high sensitivity and specificity cut-off value of RDW to predict stent thrombosis. This may restrict the use of RDW in daily clinical practice to predict stent thrombosis.

6. Conclusion

Higher RDW values may be a predictor of stent thrombosis in patients who underwent stent implantation due to STEMI.

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