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 anticoagulant-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 endothelialization 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
acute pulmonary embolism.\textsuperscript{[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 marrow and may cause increased RDW values in cardiovascular diseases.\textsuperscript{[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 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\textsubscript{2}–V\textsubscript{3} and/or of ≥1 mm (0.1 mV) in other contiguous chest leads or the limb leads.\textsuperscript{[3,13]}

Time between stent implantation and control coronary angiography 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.\textsuperscript{[14]} Stent thrombosis was defined as presence of thrombus inside of stent or 5 mm apart from stent whether it cause occlusion or not.\textsuperscript{[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

\begin{table}[h]
\centering
\caption{Clinical and hematological characteristics of patients according to stent thrombosis.}
\begin{tabular}{lccc}
\hline
Variables & Stent thrombosis & Control & P \\
\hline
Age, years & 56.3 ± 10.7 & 54.9 ± 11.8 & 0.253 \\
Male sex & 120 (82%) & 136 (89%) & 0.074 \\
Hypertension & 68 (47%) & 60 (42%) & 0.025 \\
Diabetes mellitus & 57 (35%) & 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 ± 60 & 180 ± 35 & 0.002 \\
HDL-cholesterol, mg/dL & 37 ± 16 & 38 ± 15 & 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 ± 0.4 & 3.04 ± 0.4 & 0.122 \\
Stent length, mm & 19.7 ± 1.9 & 19.6 ± 1.8 & 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\textsuperscript{12}/L & 281 ± 80 & 244 ± 60 \textsuperscript{<0.001} & \\
Mean platelet volume, fl & 8.5 ± 1.4 & 8.5 ± 1.2 & 0.901 \\
White blood cell count, \times 10\textsuperscript{9}/L & 11.93 ± 3.52 & 12.14 ± 4.13 & 0.626 \\
Neutrophil count, \times 10\textsuperscript{9}/L & 8.85 ± 3.64 & 8.07 ± 4.01 & 0.794 \\
Lymphocyte count, \times 10\textsuperscript{9}/L & 2.10 ± 0.93 & 2.21 ± 2.35 & 0.388 \\
Neutrophil to lymphocyte ratio & 5.54 ± 5.07 & 5.52 ± 4.41 & 0.970 \\
\hline
\end{tabular}
\end{table}

Values are mean ± SD or n (%). HDL = high-density lipoprotein, LDL = low-density lipoprotein, RDW = red cell distribution width, SD = standard deviation.
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.
Table 2
Clinical and hematological characteristics of patients according to RDW tertiles.

| Variables                        | Tertile 1          | RDW Tertile 2          | Tertile 3          | P     |
|---------------------------------|--------------------|------------------------|--------------------|-------|
| Variables                        | Tertile 1          | Tertile 2              | Tertile 3          |       |
| Age, years, 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 (23%)           | 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              | 2.90±0.39          | 0.409 |
| Stent length, mm                 | 18±5               | 18±6                   | 22±7               | <0.001|
| Stent thrombosis                 | 47 (41%)           | 40 (38%)               | 60 (58%)           | 0.006 |
| Type of stent thrombosis         |                    |                       |                    |       |
| Acute                            | 16 (34%)           | 9 (23%)                | 11 (18%)           | 0.520 |
| Subacute                         | 25 (63%)           | 21 (54%)               | 27 (45%)           |       |
| Late and very late               | 14.4±1.4           | 14.5±1.2               | 14.0±1.4           | 0.006 |
| Platelet count, ×10^9/L          | 264±72             | 252±55                 | 269±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, ×10^9/L  | 12.8±4.3           | 11.6±3.6               | 11.7±3.5           | 0.040 |
| Neutrophil count, ×10^9/L        | 9.8±4.3            | 8.2±3.7                | 8.6±3.2            | 0.006 |
| Lymphocyte count, ×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 women. 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 (18.3±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).
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.\[15,26\] 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.\[24\] 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.

References
\[1\] Steg PG, James SK, Ahar D, et al. Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC), ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 2012;33:2569–619.

\[2\] Van Domburg RT, Foley DP, de Jaegere PP, et al. Long term outcome after coronary stent implantation: a 10 year single centre experience of 1000 patients. Heart Be Card Soc 1999;82(Suppl 2):B27–34.

\[3\] Puel J, Joffre F, Rousseau H, et al. [Self-expanding coronary endoprosthesis in the prevention of restenosis following transluminal angioplasty. Preliminary clinical study]. Arch Mal Coeur Vaiss 1987;80:1311–2.

\[4\] Leibundgut G, Niederpisch F, Patt U, et al. Stent thrombosis up to 3 years after stenting for ST-segment elevation myocardial infarction versus for stable angina – comparison of the effects of drug-eluting versus bare-metal stents. Am Heart J 2009;158:271–6.

\[5\] Dansa GD, Caixeta A, Mehran R, et al. Frequency and predictors of stent thrombosis after percutaneous coronary intervention in acute myocardial infarction. Circulation 2011;123:1745–56.

\[6\] Guler A, Karabay C, Kalya A, et al. [Coronary optical coherence tomography in late stent thrombosis: the first application in Turkey]. Türk Kardiyol. Derneci Ars. Türk Kardiyol. Derneği’nin Yayın Organları 2012;40:63–5.

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 | 1.005 (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 (1.927–2.509) | 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 an 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 anisoctyesis in nutritional insufficiency, iron, folic acid and vitamin B12 deficiency, chronic liver disease, and blood transusion.\[15,16\] 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 7536 healthy volunteers by Zalawadiya et al,\[18\] 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.\[19\]

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. Aet 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.
[7] Isik T, Ayhan E, Kurt M, et al. Is red cell distribution width a marker for the presence and poor prognosis of cardiovascular disease? Eurasian J Med 2012;44:169–71.
[8] Zalawadiya SK, Veeranna V, Niraj A, et al. Red cell distribution width and risk of coronary heart disease events. Am J Cardiol 2010;106: 988–93.
[9] Zorlu A, Bektasoglu G, Guven FMK, et al. Usefulness of admission red cell distribution width as a predictor of early mortality in patients with acute pulmonary embolism. Am J Cardiol 2012;109:128–34.
[10] Hampole CV, Mehtrota AK, Thenappan T, et al. Usefulness of red cell distribution width as a prognostic marker in pulmonary hypertension. Am J Cardiol 2009;104:868–72.
[11] Ani C, Ovbiagele B. Elevated red blood cell distribution width predicts mortality in persons with known stroke. J Neurol Sci 2009; 277:103–8.
[12] Uyarel H, Ergelen M, Cicek G, et al. Red cell distribution width as a novel prognostic marker in patients undergoing primary angioplasty for acute myocardial infarction. Coron Artery Dis 2011;22:138–44.
[13] O’Gara PT, Kushner FG, Aschheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions. Catheter Cardiovasc Inter 2013;82:1–27.
[14] Vranckx P, Kint P-P, Morel M-A, et al. Identifying stent thrombosis, a critical appraisal of the academic research consortium (ARC) consensus definitions: a lighthouse and as a toe in the water. Eurointervention 2008;4(Suppl C):C39–44.
[15] Yang W, Huang H, Wang Y, et al. High red blood cell distribution width is closely associated with nonalcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2014;26:174–8.
[16] Evans TC, Jehle D. The red blood cell distribution width. J Emerg Med 1991;9(Suppl 1):71–4.
[17] Weiss G, Goodnough LT. Anemia of chronic disease. N Engl J Med 2003;352:1011–23.
[18] Tsimikas S, Willerson JT, Ridker PM. C-reactive protein and other emerging blood biomarkers to optimize risk stratification of vulnerable patients. J Am Coll Cardiol 2006;47:C19–31.
[19] Libby P, Ridker PM, Maserei A. Inflammation and atherosclerosis. Circulation 2002;105:1135–43.
[20] Ghaifari S. Oxidative stress in the regulation of normal and neoplastic hematopoesis. Antioxid Redox Signal 2008;10:1923–40.
[21] Tsimikas S, Willerson JT, Ridker PM. C-reactive protein and other emerging blood biomarkers to optimize risk stratification of vulnerable patients. J Am Coll Cardiol 2006;47:C19–31.
[22] Lippi G, Targher G, Montagna M, et al. Relation between red blood cell distribution width and inflammatory biomarkers in a large cohort of unselected outpatients. Arch Pathol Lab Med 2009;133:628–32.
[23] Acet H, Ertas F, Akd MA, et al. Relationship between hematologic indices and global registry of acute coronary events risk score in patients with ST-segment elevation myocardial infarction. Clin Appl Thromb 2016;22:60–8.
[24] Brener SJ, Ertelt K, Mehran R, et al. Predictors and impact of target vessel revascularization after stent implantation for acute ST-segment elevation myocardial infarction: lessons from HORIZONS-AMI. Am Heart J 2015;169:242–8.