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Dynamic Monitoring of Erythrocyte Distribution Width (RDW) and Platelet Distribution Width (PDW) in Treatment of Acute Myocardial Infarction

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Background: This study investigated the role of erythrocyte distribution width (RDW) and platelet distribution width (PDW) in evaluating the treatment efficacy for acute myocardial infarction (AMI).





Material/Methods: A total of 120 AMI patients receiving conventional myocardial infarction treatment were included. The patients were divided into an effective group and an ineffective group based on treatment efficacy. The RDW and PDW were measured before and after treatment. We used the independent samples *t* test, chi-square test, logistic regression, and ROC curves for analysis.

Results: The change and change rate of RDW and PDW were significantly improved ($p < 0.01$) and the positive change rate of RDW, PDW, and RDW + PDW were significantly lower in the effective group compared with those in the ineffective group ($p < 0.01$). The change and change rate of RDW and PDW are independent factors for treatment efficacy evaluation ($p < 0.05$). ROC curve analysis showed that the changes and change rate of RDW and PDW were all significant in evaluating treatment efficacy ($p < 0.05$).

Conclusions: The change and change rate of RDW and PDW or their combination can be used to evaluate treatment efficacy; however, the absolute value of RDW and PDW are not as significant.

MeSH Keywords: **Aftercare • Blood Platelets • Erythrocyte Indices • Myocardial Infarction**

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Background

Patient with acute myocardial infarction (AMI) usually present with chest pain, feeling of suffocation, or even death [1]. AMI results from the sharp reduction or interruption of coronary blood caused by coronary atherosclerosis, leading to the corresponding myocardial ischemia and severe acute myocardial necrosis [2]. Pathologically, lipids invade the arterial vessels, accumulate in the smooth muscle cells, collagen, and elastic fibers, and cause smooth muscle hyperplasia [3]. The blood-derived mononuclear cells can engulf a large number of lipids, become foam cells, release active substances, stimulate fibrous tissue proliferation, activate inflammatory responses [4], form atherosclerotic plaque, increase endothelial damage, and promote coronary stenosis, eventually leading to AMI [5].

For AMI, early evaluation and prediction of prognosis are needed to plan individualized treatment [6,7]. In recent years, the prediction value of erythrocyte distribution width (RDW) and platelet distribution width (PDW) in AMI has been reported [8,9]. For example, in 2007, Felker et al. [10] first found that elevated RDW could be used as an independent prognostic factor in patients with heart failure. RDW reflects the size differences of red blood cells and can differentiate the false-negative of mean corpuscular volume in the measure of erythrocyte morphological changes. RDW is significantly correlated with the incidence and development of coronary heart disease (CHD); therefore, it can be used as a sensitive indicator for CHD risk stratification [11,12]. Platelet distribution width (PDW) indicates the heterogeneity of platelet volume, and the elevated PDW indicates a large disparity in the platelet volume [13]. High PDW value suggests hypercoagulability, possibly due to a large amount of platelet adhesion and aggregation, high activation, and subsequent platelet reduction, leading to spontaneous induction of platelets by megakaryocytes [14]. Most relevant studies have focused on the absolute value of RDW and PDW. However, the reference intervals of the normal values of RDW and PDW are too large to accurately distinguish between individual differences between patients. Thus, it is of great importance to develop new indicators that can better distinguish the individual differences.

The change and change rate of RDW and PDW before and after treatment for the early treatment efficacy of AMI patients have seldom been studied. It has been reported [15] that there are little differences between a coronary heart disease group and a healthy control group in RDW and PDW before treatment, indicating its limited prognosis prediction value. It has also been reported that RDW change rate can be used for AMI treatment efficacy and prognosis evaluation, presenting as a significant RDW decrease after treatment [16]. Therefore, instead of the absolute values of RDW and PDW, the change and change rate of RDW and PDW before and after treatment can

be used as important indicators for evaluating the early AMI treatment efficacy.

In the present study, we analyzed the absolute value, change, and change rate of RDW and PDW to determine their value in evaluation of early AMI treatment efficacy.

Material and Methods

Patient recruitment

Patients diagnosed with AMI and admitted to Chongqing Medical University Beibei Affiliated Hospital from April 2014 to March 2015 were included in this study. The patients were diagnosed based on the WHO Clinical Diagnostic Criteria for Coronary Heart Disease, 2003 version. Inclusion criteria were: 1) Patients with “non-ST-elevation myocardial infarction” of first onset and hospitalized within 12 h of onset; and 2) Patients with similar first course treatment strategy. Patients were excluded if they had diabetes, malignancies, chronic respiratory diseases, or coagulation disorders, or received interventional therapy. The study was approved by the Medical Ethics Board of Chongqing Medical University Beibei Affiliated Hospital. Informed consent for participation in a clinical trial was obtained from all patients or their families.

The treatment was based on the Guidelines for the Treatment of Acute Myocardial Infarction, prepared by the Chinese Medical Association Cardiovascular Society in 2001. Patients all received treatment including conventional treatment (sedation drugs, analgesic drugs, statins, rehydration, oxygen, thrombolysis, and nitrates). The first course of treatment lasted for 7–10 days. The RDW and PDW were measured before (within 2 h of hospitalization) and after the first course of treatment (at 7:00 am–8:00 am of the last day of the first treatment course). Patients were divided into an effective group and an ineffective group based on treatment efficacy. Treatments were considered effective if the following 3 conditions were met: 1) serum levels of CTNi, CTNt, and CRP decreased to levels below the diagnostic threshold; 2), ECG significantly improved, especially that ST segment and T wave returned to normal; 3) chest tightness and chest pain of patients disappeared. Otherwise, treatments were considered ineffective. After discharge, all patients were followed up by telephone or clinic visits for 12 months.

Measurement of RDW and PDW

Venous blood samples collected into tripotassium EDTA tubes were analyzed within 2 h after venipuncture. The RDW and PDW values were determined by an Automated Hematology Analyzer (SF-3000, SYSMEX 2001, Kobe, Japan). The reference

range of RDW was 37~50 fl and the reference range of coefficient variation of PDW was 9~16%.

Definitions

The small letter “c” represents the changes before and after treatment. The letters “cr” represent the change rate before and after treatment. Thus, the formulas to calculate the change and change rate of RDW and PDW were as follows: $RDW_c = RDW \text{ after treatment} - RDW \text{ before treatment}$; if the value of $(RDW \text{ after treatment} - RDW \text{ before treatment})$ is larger than or equal to zero, $RDW_cr = (RDW \text{ after treatment} - RDW \text{ before treatment}) / RDW \text{ after treatment}$; if the value of $(RDW \text{ after treatment} - RDW \text{ before treatment})$ is less than zero, $RDW_cr = (RDW \text{ after treatment} - RDW \text{ before treatment}) / RDW \text{ before treatment}$; $PDW_c = PDW \text{ after treatment} - PDW \text{ before treatment}$; if the value of $(PDW \text{ after treatment} - PDW \text{ before treatment})$ is larger than or equal to zero, $PDW_cr = (PDW \text{ after treatment} - PDW \text{ before treatment}) / PDW \text{ after treatment}$; if the value of $(PDW \text{ after treatment} - PDW \text{ before treatment})$ is less than zero, $PDW_cr = (PDW \text{ after treatment} - PDW \text{ before treatment}) / PDW \text{ before treatment}$. To avoid the maximum value, the smaller absolute value of cr obtained by the 2 algorithms was used.

After treatment, if the detected value of RDW or PDW is greater than the upper limit of the reference value, the detected value is defined as positive (P); otherwise, the detected value is defined as negative (N). If the change and change rate of RDW or PDW before and after treatment are larger than or equal to zero, the related value is defined as P; otherwise, the related value is defined as N. If the detected values of RDW and PDW after treatment are both P, the combined detection of RDW and PDW is defined as P; otherwise, it is defined as N. If the change and change rate of RDW and PDW before and after treatment are both P, the combined change and change rate of RDW and PDW are defined as P; otherwise, it is defined as N.

Statistical analysis

SPSS17.00 was used for data analysis. All continuous variables were assessed for normal distribution with the Kolmogorov-Smirnov test. Since all the variable were not distributed normally, the changes and change rates of RDW and PDW before and after treatment were compared between the effective group and the ineffective group by use of the non-parametric independent samples *t* test. The chi-square test was used to compare the positive rate and positive change rate of RDW and PDW before and after treatment between the effective group and the ineffective group.

The effect of age, sex, and length of treatment, as well as positive rate, positive change rate, value, and value change of RDW

and PDW, on treatment efficacy was analyzed by multivariate logistic regression. A total of 4 logistic regressions were performed with different variables. Model I used values of RDW and PDW after treatment. Model II used positive and negative results of RDW and PDW after treatment. Model III used changes of RDW and PDW before and after treatment. Model IV used positive and negative results of RDW and PDW changes before and after treatment. ROC curves were generated based on value change and change rate of RDW and PDW. The area under the curve (AUC) was calculated and the critical point and corresponding sensitivity and specificity points were identified. $p < 0.05$ was considered as statistically significant.

Results

Patient demographics

A total of 120 AMI patients were included in the study. In the effective group, the mean age was 73.15 ± 10.821 years, length of treatment was 8.25 ± 4.938 days, and the sex ratio of males to females was 36/25. In the ineffective group, the mean age was 73.24 ± 10.828 years, length of treatment was 7.41 ± 3.797 days, and the sex ratio of males to females was 34/25. There was no significant difference in patient demographics between the 2 groups.

In the effective group, the follow-up rate was 62.29% (38/61), including 3 cases of AMI and 1 death. In the ineffective group, the follow-up rate was 57.62% (34/59), including 4 cases of AMI and 2 deaths. There were no significant differences between AMI recurrence rate and death rate ($p > 0.05$).

Change and change rate of RDW and PDW

The change and change rate of RDW (RDW_c and RDW_cr) and PDW (PDW_c and PDW_cr) between effective treatment group and ineffective treatment group were compared. There was no significant difference of RDW_before treatment, RDW_after treatment, PDW_before treatment and PDW_after treatment between the 2 groups, as shown in Table 1. RDW_c, PDW_c, RDW_cr and PDW_cr were all significantly different ($p < 0.01$) (Table 2). Results showed that the changes in RDW and PDW significantly decreased in the effective group compared with the ineffective group.

The positive rate and positive change rate of RDW, PDW, and RDW + PDW

The positive rate and positive change rate of RDW, PDW, and RDW + PDW between the effective treatment group and ineffective treatment group were compared. We found that the positive rates of RDW_c, PDW_c, and RDW_c + PDW_c were

Table 1. The value of RDW and PDW in the effective group and ineffective group before and after treatment.

	RDW_before treatment		RDW_after treatment		PDW_before treatment		PDW_after treatment	
	Effective group	Ineffective group	Effective group	Ineffective group	Effective group	Ineffective group	Effective group	Ineffective group
Number of case (n)	61	58	61	58	58	54	58	54
Mean ±SD	47.2±5.1	46.6±7.6	46.2±4.9	48.3±8.8	14.6±3.1	13.4±2.3	13.5±2.8	13.6±2.5
Z(t)	0.547		0.673		1.937		0.245	
P	0.585		0.501		0.053		0.807	

Table 2. The differences, change and change rate of RDW and PDW between the effective group and ineffective group before and after treatment.

	RDW_c		PDW_c		RDW_cr		PDW_cr	
	Effective group	Ineffective group	Effective group	Ineffective group	Effective group	Ineffective group	Effective group	Ineffective group
Number of case (n)	61	58	58	54	61	58	58	54
Mean ±SD	-1.08±3.78	1.72±4.5	-1.04±2.4	0.22±1.5	-2.14±7.2	3.12±7.3	-6.5±14.1	1.35±9.9
Z(t)	4.567		3.559		4.484		3.442	
P	0.000		0.000		0.000		0.001	

RDW_c = RDW after treatment–RDW before treatment; RDW_cr = min [(RDW after treatment–RDW before treatment)/RDW after treatment or (RDW after treatment–RDW before treatment)/RDW before treatment]; PDW_c = PDW after treatment–PDW before treatment; PDW_cr = min [(PDW after treatment–PDW before treatment)/PDW after treatment or (PDW after treatment–PDW before treatment)/PDW before treatment].

Table 3. The difference and positive rate of RDW, PDW, RDW with PDW of the effective and ineffective groups before and after treatment.

	RDW_after treatment_PN		PDW_after treatment_PN		RDW_after treatment + PDW_after treatment_PN		RDW_c_PN		PDW_c_PN		RDW_c + PDW_c_PN	
	N	P	N	P	N	P	N	P	N	P	N	P
Effective	48	13	47	11	58	3	45	16	41	17	56	5
Ineffective	42	16	45	9	53	1	13	45	22	32	28	25
Chi-square value	0.635		0.101		0.802		31.386		10.192		22.215	
P value	0.425		0.751		0.370		0.000		0.001		0.000	
Odds ratio	1.407		0.855		0.365		9.736		3.508		10.000	
95%CI	Lower	0.607	0.324		0.037		4.201		1.602		3.458	
	Upper	3.261	2.257		3.615		22.561		7.682		28.920	

P – positive; N – negative.

Table 4. The impact of gender, age, treatment length, RDW and PDW indicators on treatment efficacy.

	Model I			Model II			Model III			Model IV		
	B	Wals F	P	B	Wals F	P	B	Wals F	P	B	Wals F	P
Gender	0.121	0.94	0.759	0.179	0.204	0.652	0.158	0.129	0.72	-0.195	0.17	0.68
Age	-0.006	0.109	0.741	-0.003	0.033	0.855	-0.06	0.091	0.763	-0.016	0.578	0.447
Length of treatment	-0.054	1.445	0.229	-0.055	1.46	0.227	-0.4	0.606	0.436	-0.074	1.883	0.17
RDW_after treatment	0.26	0.437	0.509	-	-	-	-	-	-	-	-	-
PDW_after treatment	-0.014	0.035	0.851	-	-	-	-	-	-	-	-	-
RDW_after treatment_PN	-	-	-	0.087	0.033	0.855	-	-	-	-	-	-
PDW_after treatment_PN	-	-	-	-0.365	0.481	0.488	-	-	-	-	-	-
RDW_c	-	-	-	-	-	-	0.222	9.066	0.003	-	-	-
PDW_c	-	-	-	-	-	-	0.359	8.388	0.004	-	-	-
RDW_c_PN	-	-	-	-	-	-	-	-	-	2.189	20.291	<0.001
PDW_c_PN	-	-	-	-	-	-	-	-	-	1	4.842	0.028

The four regression models are all logistic regression models with gender, age, duration of treatment, RDW and PDW as independent variables, and efficacy evaluation (effectiveness and ineffectiveness) as dependent variables. Model I used values of RDW and PDW after treatment. Model II used positive and negative results of RDW and PDW after treatment. Model III used changes of RDW and PDW before and after treatment. Model IV were positive and negative results of RDW and PDW changes before and after treatment.

significantly different ($p < 0.01$), but there was no significant difference in the positive rate of RDW_after treatment, PDW_after treatment, and RDW_after treatment + PDW_after treatment between the 2 groups ($p > 0.05$), as shown in Table 3. These results indicate that the positive change rate of PDW and RDW can better reflect the treatment effects.

The impact of sex, age, length of treatment, RDW, and PDW index on treatment efficacy

To analyze the impact of sex, age, length of treatment, RDW, and PDW index on the treatment efficacy, multivariate logistic regression was used. The results are shown in Table 4. The total effective rate was 49.5% for model I and 54.1% for model II. The above 5 indicators were not independent factors for both model I and model II ($p > 0.05$). The total effective rate of model III was 69.4%, and the changes of RDW and PDW were independent factors ($p < 0.01$). The total effective rate of model IV was 72.1%, and the positive change rates of RDW and PDW were independent factors ($p < 0.05$).

ROC for RDW and PDW index on treatment efficacy

The RDW_after treatment, RDW_c, and RDW_cr values were used to draw the ROC curve of RDW after treatment (Figure 1). The AUC for RDW after treatment was 0.536 [95% CI (0.431–0.640), $p > 0.05$]. At the critical value of 45.750 fl, the corresponding prediction sensitivity was 55.2% and the specificity was 57.4%. The AUC of RDW_c was 0.743 [95% CI (0.651–0.834), $p < 0.001$]. At the critical value of -0.050fl, the corresponding predictive sensitivity was 77.6% and specificity was 73.8%. The AUC of RDW_cr showed the AUC was 0.738 [95% CI (0.646–0.831), $p < 0.001$]. At the critical value of -0.216%, the corresponding predictive sensitivity was 79.3% and the specificity was 73.8%.

The PDW_after treatment, PDW_c and PDW_cr value were used to generate the ROC curve of PDW after treatment. The AUC for PDW_after treatment was 0.513 [95% CI (0.406–0.621), $p > 0.05$]. At the critical value of 12.650%, the corresponding prediction sensitivity was 61.1% and the specificity was 41.3%. The AUC of RDW_c was 0.694 [95% CI (0.595–0.794), $p < 0.001$]. At the

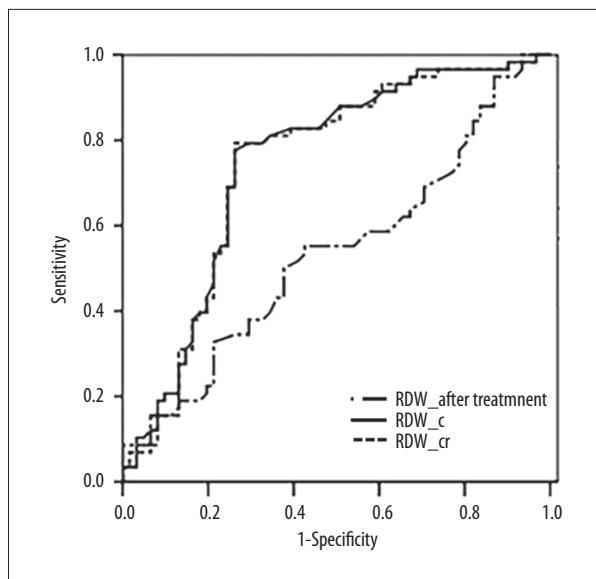


Figure 1. ROC curve of RDW_after treatment, RDW_c and RDW_cr.

critical value of -0.550% , the corresponding predictive sensitivity was 75.9% and specificity was 62.1%. The AUC curve of PDW_cr was 0.687 [95% CI (0.586–0.787), $p < 0.01$]. At the critical value of -4.686% , the corresponding predictive sensitivity was 79.6% and the specificity was 60.3% (Figure 2).

Discussion

AMI and ischemic heart disease have become major causes of emergency death [17]. Epidemiological studies [18, 19] have shown that the ischemic heart disease mortality rate has doubled in the past 2 decades, and the number of deaths has reached more than 1 million each year [20]. According to the World Bank, the MI-related death rate is increasing in China and may reach 230,000 in 2030 [21]. Clinically, patients may show chest pain, acute circulatory dysfunction, acute myocardial ischemia, myocardial damage, and necrosis on electrocardiogram and serum myocardial markers [22]. Its acute onset and high mortality rate leads to early death in the elderly [23].

Because of the acute onset of AMI and early insensitivity of therapeutic effect evaluation, it is of great importance of identifying the early response in AMI [24]. At present, serum enzyme indicators are used, including aspartate aminotransferase (AST), lactate dehydrogenase (LDH), and its isoenzymes [25]. However, AST and LDH are distributed in many organs of the body, and the diagnostic specificity is poor [26]. Abnormal increase of serum creatine kinase isoenzyme (CK-MB) and cardiac troponin (cTn) can also be early predictors of AMI and its treatment efficacy; however, CK-MB is expressed in multiple systems and thus may not accurately represent myocardial

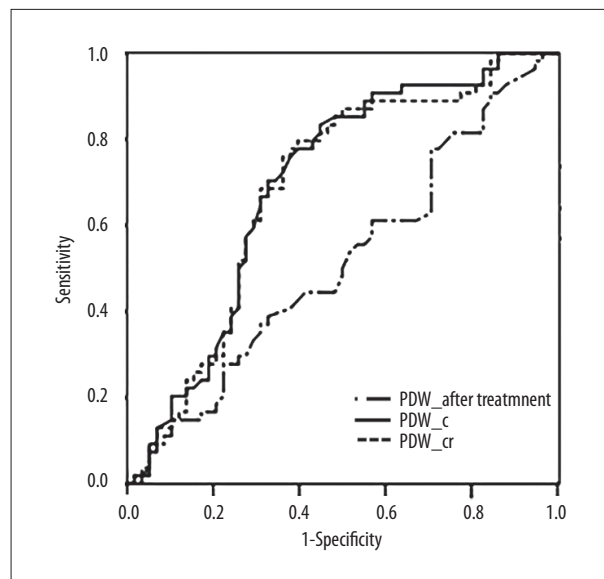


Figure 2. ROC curve of PDW_after treatment, PDW_c and PDW_cr.

injury [27]. In addition, cTn has limited ability to predict treatment efficacy. Peripheral circulating DNA also has some predictive significance on AMI [28], but it is not universally used in clinical practice due to its high cost. Homocysteine (Hcy) can directly or indirectly lead to vascular endothelial cell damage, promote vascular smooth muscle cell proliferation, affect low-density lipoprotein oxidation, enhance platelet function, and promote thrombosis, but it is not widely used clinically due to the difficult procedure involved [29]. In assessing MI extent, aVF lead ST full-spectrum changes have prognostic significance in AMI, but with low sensitivity [30].

In recent years, the relationship between RDW/PDW and AMI has been reported. RDW is a parameter reflecting the difference in the size of red blood cells. The high RDW value reflects the large degree of red blood cell volume dispersion, and can also be used as an indicator of ischemia [31]. A high RDW value may be associated with cerebral vein thrombosis. PDW is a reflection of platelet volume heterogeneity, and the increase in platelet volume may indicate the disparity [8,32]. PDW is closely related to coronary artery diseases [33]. The PDW value of activated platelet may further change, presenting as increased number of pseudopods and morphology types [34]. A higher PDW value suggests that the blood is hypercoagulable and may further promote thrombosis [35]. The ischemia and hypoxia of corresponding tissue can accelerate platelet formation, induce the release of reticulated platelets by bone marrow [36], and further raise the PDW value. Ozyurtlu et al. [37] found that compared with the normal control group, there were no significant differences in PDW before and after treatment in patients with coronary heart disease. Similarly, in our study, there were no differences in RDW and PDW between

the effective group and ineffective group before and after treatment, suggesting that the value of RDW and PDW cannot predict early treatment efficacy. Consistent with the report by Ozdemir et al. [15], our study found that there were significant differences in the amount of change and change rate of RDW and PDW before and after treatment between the effective and the ineffective groups, suggesting that they may be good indicators for treatment efficacy. At the same time, there were significant differences in the positive change rate of RDW and PDW between the effective and ineffective groups. We performed further analyses and found significant differences in the changes in RDW and PDW between the effective and ineffective group. Muhlestein et al. [16] suggested that the change and change rate are of high value in determining the effect of early AMI treatment. In addition, multivariate logistic regression analysis in the present study showed that the RDW and PDW changes and change positive rates were independent risk factors for AMI. The ROC curve showed that the treatment evaluation value of RDW change and change rate was better than the RDW. Similarly, the treatment evaluation value of PDW change and change rate was better than the PDW. Therefore, we believe that the introduction of change and change rate as indicators for dynamic changes of RDW and PDW can better represent AMI treatment efficacy to guide individualized treatment. On the contrary, Wang Xiao et al. [38] showed that when compared with the control group, the RDW value did not significantly decrease in patients with successful treatment.

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They also found that RDW first increased and then decreased in the first 7–9 days of the AMI treatment. Wang et al. [9] reported that PDW change was not significant during the course of MI treatment, and PDW was lower in Killip Class 3. These results indicate that more in-depth study is needed to elucidate the role of RDW and PDW in evaluation of treatment efficacy.

This study has some limitations. First, the sample size was relatively small, and studies with larger sample sizes are needed. Second, complete data on biochemical indexes were lacking. Furthermore, long-term follow-up data were not collected; therefore, the effect of RDW and PDW on long-term efficacy was not analyzed. Finally, the effect of antiplatelet drug on RDW and PDW was not investigated in this study.

Conclusions

RDW and PDW should be widely used due to their simplicity and low price. Compared with the absolute values of RDW and PDW, their changes or change rates can better reflect the treatment outcome for AMI.

Conflict of interest

The authors declare no conflict of interest.

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