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Relation between red blood cell distribution width (RDW) and all-cause mortality at two years in an unselected population referred for coronary angiography

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Abstract

Background: Red blood cell distribution width (RDW), a numerical measure of the variability in size of circulating erythrocytes, has recently been shown to be a strong predictor of adverse outcomes in patients with heart failure and in patients with prior myocardial infarction but no symptomatic heart failure at baseline, even after adjustment for hematocrit. However, there are no data in other cardiac populations, including patients with acute coronary syndromes (ACS).
Methods: The present study investigated the long-term prognostic significance of baseline RDW in a well-characterized cohort of 389 male patients who were referred to coronary angiography for a variety of indications. All patients were followed prospectively for all-cause mortality, and data regarding this endpoint was available for 97% of the population at 24 months.
Results: After controlling for a variety of baseline variables (including hemoglobin and the presence of heart failure), RDW (analyzed as a categorical variable comparing the upper tertile of baseline values to the lower two levels combined) was a strong and independent predictor of all-cause mortality using a Cox proportional hazards model [hazard ratio (HR) 2.69, 95% confidence interval (CI) 1.50–4.84, $p=0.0008$]. In addition, baseline RDW was also an independent predictor of all-cause mortality in the non-anemic (HR 4.73, 95% CI 2.06–10.86, $p=0.0003$) and ACS (HR 2.90, 95% CI 1.32–6.38, $p=0.0082$) subpopulations of patients.
Conclusions: These data demonstrate that elevated RDW is a strong and independent predictor of all-cause mortality in an unselected population of male patients across a broad spectrum of risk (including ACS) referred for coronary angiography.

Keywords: RDW; Anemia; Coronary angiography; Prognosis

1. Introduction

Anemia has been shown to be a powerful and independent predictor of adverse cardiovascular outcomes in multiple patient populations [1–6]. More recently RDW, a numerical measure of the variability in size of circulating erythrocytes, has also been shown to be a strong predictor of adverse outcomes in patients with heart failure [7] and in patients with prior myocardial infarction but no symptomatic heart failure at baseline [8], even after adjustment for hematocrit. Accordingly, we tested the hypothesis that higher values of RDW are associated with the risk of all-cause mortality in a broad and unselected population of male patients referred to coronary angiography for a variety of indications, including ACS.

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2. Methods

2.1. Study design

This was an observational study derived from a cohort of patients prospectively entered into a database for the purpose of examining the prognostic significance of various plasma biomarkers in patients with known or suspected coronary artery disease. The study population and design have been previously described in detail elsewhere [9]. Briefly, 389 male patients undergoing coronary angiography for a variety of indications constituted the study population. Patients presenting with ST-segment elevation MI (23 patients), non-ST-segment elevation MI (84 patients), or unstable angina pectoris (86 patients) were classified as ACS. For the purpose of the present analysis, all other patients were classified as non-ACS (196 patients), whether or not the principal indication for coronary angiography was related to chest pain. Patients were also classified as either anemic or non-anemic, based on the WHO definition of anemia [10]. The primary endpoint of the study was all-cause mortality at 24 months for the entire cohort of patients. The secondary

Table 1
Baseline characteristics of the entire cohort stratified by the upper tertile of baseline RDW values (≥14.4% versus <14.4%)

Characteristics	Statistics	RDW<14.4% (*N*=240)[a]	RDW≥14.4% (*N*=130)[a]	*p*-value
Age (years)	Mean (std)	64.7 (10.2)	66.6 (9.7)	0.0991
Race				0.0214
Black		68 (28.3%)	54 (41.5%)	
Hispanic		78 (32.5%)	29 (22.3%)	
White		94 (39.2%)	47 (36.2%)	
Unstable angina pectoris		55 (23.0%)	28 (21.5%)	0.7460
Congestive heart failure on presentation		51 (21.3%)	48 (36.9%)	0.0011
Myocardial infarction on presentation		75 (31.3%)	31 (23.9%)	0.1326
Family history of premature CAD		63 (26.3%)	27 (20.8%)	0.2408
Diabetes mellitus		104 (43.3%)	53 (40.8%)	0.6338
Hypertension		193 (80.4%)	116 (89.2%)	0.0292
Any history of tobacco use		193 (80.4%)	109 (83.9%)	0.4162
Active tobacco use		69 (28.8%)	44 (33.9%)	0.3096
Body mass index (kg/m^2)	Mean (std)	28.38 (5.21)	29.11 (6.73)	0.9430
	Median	28.2	27.5	
	(25th, 75th)	24.6, 31.2	24.1, 32.0	
Hyperlipidemia[b]		137 (57.1%)	62 (47.7%)	0.0837
Aspirin use		209 (87.1%)	107 (82.3%)	0.2142
Beta blocker use		179 (74.6%)	78 (60.0%)	0.0036
ACE-inhibitor use		144 (60.0%)	80 (61.5%)	0.7725
Statin use		134 (55.8%)	58 (44.6%)	0.0392
Fibrate use		13 (5.4%)	2 (1.5%)	0.0966
Prior coronary artery bypass grafting		21 (8.8%)	12 (9.2%)	0.8769
No. of diseased coronary arteries[c]				0.0906
0		41 (17.1%)	28 (21.5%)	
1		41 (17.1%)	19 (14.6%)	
2		55 (22.9%)	39 (30.0%)	
3		87 (36.3%)	42 (32.3%)	
4		16 (6.7%)	2 (1.5%)	
Left ventricular ejection fraction				0.0827
≥55%		93 (40.4%)	34 (28.3%)	
45–54%		53 (23.0%)	30 (25.0%)	
31–44%		53 (23.0%)	30 (25.0%)	
≤30%		31 (13.5%)	26 (21.7%)	
Hemoglobin (g/dl)	Mean (std)	13.73 (1.51)	12.91 (1.87)	*p*<0.0001
	Median	13.80	12.85	
	(25th, 75th)	12.9, 14.7	11.7,14.2	
Serum creatinine (mg/dl)	Mean (std)	1.11 (0.33)	1.52 (2.04)	0.3610
	Median	1.0	1.10	
	(25th, 75th)	0.9, 1.2	0.9, 1.3	
Troponin-I (ng/ml)	Mean (std)	12.53 (53.7)	8.33 (38.3)	0.6559
	Median	0.3	0.3	
	(25th, 75th)	0.2, 4.3	0.2, 1.0	

Data are presented as frequencies (percentages) for categorical variables and as means±SD and medians (25th, 75th percentiles) for continuous variables.
[a] The total number is 370 (not 389) due to missing RDW values in 19 patients.
[b] Hyperlipidemia was diagnosed in patients who had been given lipid-lowering medication or had a history of total cholesterol levels >240 mg/dl [1].
[c] Takes into account the left main, left anterior descending, left circumflex, and right coronary arteries.

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endpoints of the study were all-cause mortality in the ACS and the non-anemic subpopulations.

2.2. Laboratory methods

After an overnight fast of at least 12 h, blood was obtained from all patients enrolled in the study. Blood was collected from the arterial sheath (after a 5-mL discard) at the time of angiography but before the injection of contrast material. Hemoglobin and RDW were determined by the central laboratories of the Bronx Veterans Administration Medical Center (Bronx, New York) with the use of the Beckmann Coulter Automated CBC Analyzer (Beckman Coulter, Inc., Fullerton, California). The normal reference range for RDW in this laboratory is 11.5 to 14.5%.

2.3. Statistical methods

The study population was divided into 2 groups using the upper tertile of baseline RDW values as a pre-specified cutoff. Summary statistics for the continuous variables were presented as mean+/−SD or medians with interquartile ranges, and comparisons between the 2 groups were performed with the non-parametric Wilcoxon rank-sum test. Categorical data were summarized as frequencies and percentages, and comparisons between groups were performed with Pearson's chi-square test or Fisher's exact test.

The predictors of all-cause mortality at 24 months were identified by univariate Cox regression for the entire cohort, as well as for the ACS, non-ACS and non-anemic subpopulations. The results were presented as hazard ratios (HR) and 95% confidence intervals (CI). The same baseline variables were studied by univariate analysis for each of the groups studied: RDW, age, family history of premature coronary artery disease, diabetes mellitus, hypertension, active tobacco use, any history of tobacco use (past or present), hyperlipidemia, serum creatinine, hemoglobin, body mass index, congestive heart failure (CHF) on presentation, myocardial infarction (MI) on presentation, troponin I, previous coronary artery bypass graft surgery, number of diseased coronary arteries, left ventricular systolic function, aspirin use, statin use, beta blocker use, and angiotensin-converting enzyme inhibitor use. In all of the analyses, RDW was analyzed as a categorical variable, comparing the upper tertile of RDW values to the lower two tertiles combined. Only those univariate predictors with $p<0.05$ were subsequently entered into multivariate Cox proportional hazards regression models. The independent predictors were identified using the backward elimination procedure.

Time-to-event at 24 months was presented with Kaplan–Meier curves for the endpoint of all-cause mortality. Comparison between the 2 groups defined as the upper tertile of RDW values and the combined lower two tertiles was performed with the log rank test.

All analyses used 2-sided tests with an overall significance level of $\alpha=0.05$. All statistical analyses were performed using SAS version 8 (SAS Institute Inc., Cary, North Carolina).

3. Results

3.1. Baseline characteristics

A total of 389 patients constituted the study population. The baseline clinical, laboratory and angiographic characteristics of the study population stratified by the upper tertile of RDW values are shown in Table 1. Higher RDW values were seen in association with hypertension and CHF on presentation, while lower RDW values were seen in association with baseline use of beta blockers and statins. RDW was also negatively correlated with hemoglobin. In the entire study population, there was a moderate negative correlation between RDW and hemoglobin ($R=-0.24$ by Spearman rank correlation; $p<0.0001$).

3.2. Association of RDW with mortality

3.2.1. Entire cohort

Two year follow up data were available for 97% of the patients. In the entire cohort, there were a total of 51 deaths. On univariate analysis, RDW was a significant predictor of all-cause mortality at 24 months, with a HR of 2.76 (95% CI 1.56 to 4.90, $p=0.0005$). After adjustment for covariates that were significant for their association with all-cause mortality on univariate analysis (hemoglobin, CHF on presentation, the number of diseased coronary arteries, age, serum creatinine, and left ventricular systolic function), RDW was found on multivariate analysis to be a strong and independent predictor of all-cause mortality at 24 months (HR 2.69, 95% CI 1.50–4.84, $p=0.0008$) when comparing the upper tertile to the combined lower two tertiles (Table 2).

Using the upper tertile of RDW values as a pre-specified cutoff (i.e., ≥14.4% vs. <14.4%), Kaplan Meier curves were derived for all-cause mortality. Kaplan–Meier plots demonstrated a significant increase in all-cause mortality for patients with RDW values in the highest tertile (i.e., ≥14.4%). At 24 months, the survival rate was 78.1% for the highest tertile,

Table 2
Multivariate Cox proportional hazard analysis for all-cause mortality at 24 months for the entire population

Baseline variable	*p*-value	HR (95% CI)
RDW	0.0008	2.69 (1.50, 4.84)
CHF on presentation	0.0007	2.72 (1.53, 4.85)
Number of diseased coronary arteries	0.0005	1.64 (1.24, 2.17)

Twenty-one clinical, laboratory and angiographic variables were initially studied (see Methods) by univariate analysis. Only those predictors with $p<0.05$ (RDW, hemoglobin, CHF on presentation, LV systolic function, the number of diseased coronary arteries, age, and serum creatinine) were subsequently entered into a multivariate model, the results of which are displayed above.

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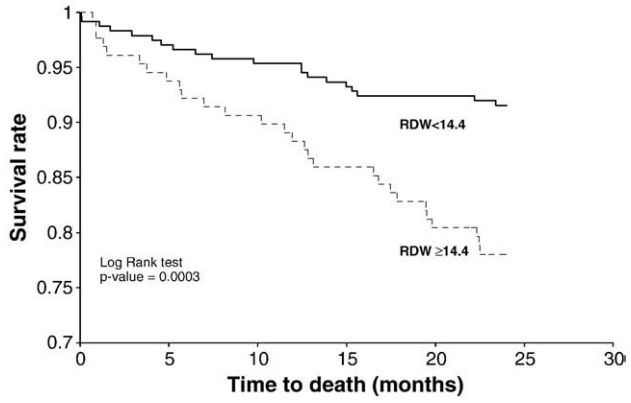

Fig. 1. Kaplan–Meier curves for all-cause mortality comparing the upper tertile of baseline RDW values to the lower two tertiles combined in the entire cohort of patients. At 24 months, the number of patients who had died in the upper tertile was 28 (21.9%), compared with 20 (8.4%) in the lower two tertiles combined ($p=0.0003$ by log rank test).

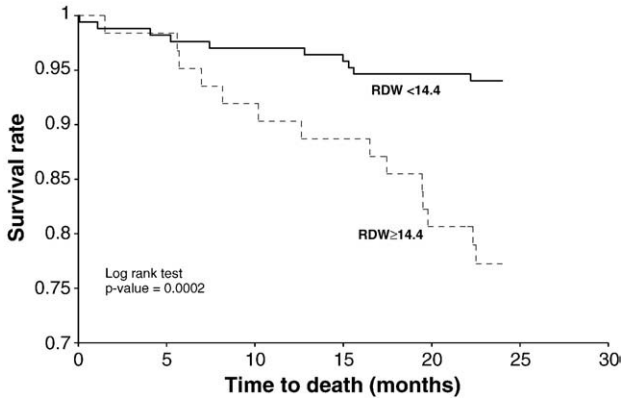

Fig. 2. Kaplan–Meier curves for all-cause mortality comparing the upper tertile of baseline RDW values to the lower two tertiles combined in the non-anemic subpopulation of patients. At 24 months, the number of patients who had died in the upper tertile was 14 (22.6%), compared with 10 (6.0%) in the lower two tertiles combined ($p=0.0002$ by log rank test).

compared with 91.6% for the lower two tertiles combined ($p=0.0003$ by log rank test) (Fig. 1).

3.2.2. The non-anemic subpopulation

Given the moderate negative correlation between RDW and hemoglobin, as well as the known association between anemia and adverse cardiovascular events, we performed additional analyses in the subpopulation of patients who were not anemic on presentation in an attempt to determine if the predictive power of RDW was either related to or affected by baseline hemoglobin levels. Using the WHO definition of anemia for men (hemoglobin < 13 g/dl), there were a total of 247 patients who were classified as non-anemic. In this subgroup of patients, there were a total of 26 deaths by 24 months. In the non-anemic subgroup of patients, RDW was a significant predictor of all-cause mortality on univariate analysis, with a HR of 4.06 (95% CI 1.80 to 9.15, $p=0.0007$) when comparing the upper tertile to the combined lower two tertiles. After adjustment for covariates that were significant for their association with all-cause mortality on univariate analysis (CHF on presentation, the number of diseased coronary arteries, and serum creatinine), RDW was found on multivariate analysis to be a strong and independent predictor of all-cause mortality at 24 months (HR 4.73, 95% CI 2.06–10.86, $p=0.0003$) when comparing the upper tertile to the combined lower two tertiles (Table 3).

Similar to the total population of patients, Kaplan–Meier survival analysis for the non-anemic subpopulation showed a significantly reduced survival in patients in the highest RDW tertile. At 24 months, the survival rate was 77.4% for patients in the highest tertile, compared with 94% for patients who were in the lower two tertiles combined ($p=0.0002$ by log rank test) (Fig. 2).

3.2.3. The ACS subpopulation

To determine if the predictive power of RDW was either related to or affected by baseline risk, we performed additional analyses in the subpopulation of patients who had presented with ACS. In the ACS subset of patients, there were a total of 26 deaths by 24 months, while in the non-ACS subset of patients there were a total of 25 deaths. In patients presenting with ACS, RDW was a significant predictor of all-cause mortality at 24 months on univariate analysis, with a HR of 2.55 (95% CI 1.16 to 5.59, $p=0.0194$) when comparing the upper tertile to the combined lower two tertiles. After adjustment for covariates that were significant for their association with all-cause mortality on univariate analysis (age and the number of diseased coronary arteries), RDW was found on multivariate analysis to be a strong and

Table 3
Multivariate Cox proportional hazard analysis for all-cause mortality at 24 months for the non-anemic subpopulation

Baseline variable	p-value	HR (95% CI)
RDW	0.0003	4.73 (2.06, 10.86)
CHF on presentation	0.0298	2.50 (1.09, 5.73)
Number of diseased coronary arteries	0.0114	1.64 (1.12, 2.39)

Twenty-one clinical, laboratory and angiographic variables were initially studied (see Methods) by univariate analysis. Only those predictors with $p<0.05$ (RDW, CHF on presentation, the number of diseased coronary arteries, and serum creatinine) were subsequently entered into a multivariate model, the results of which are displayed above.

Table 4
Multivariate Cox proportional hazard analysis for all-cause mortality at 24 months for the ACS subpopulation

Baseline variable	p-value	HR (95% CI)
RDW	0.0082	2.90 (1.32, 6.38)
Number of diseased coronary arteries	0.0099	1.80 (1.15, 2.81)

Twenty-one clinical, laboratory and angiographic variables were initially studied (see Methods) by univariate analysis. Only those predictors with $p<0.05$ (RDW, age, and the number of diseased coronary arteries) were subsequently entered into a multivariate model, the results of which are displayed above.

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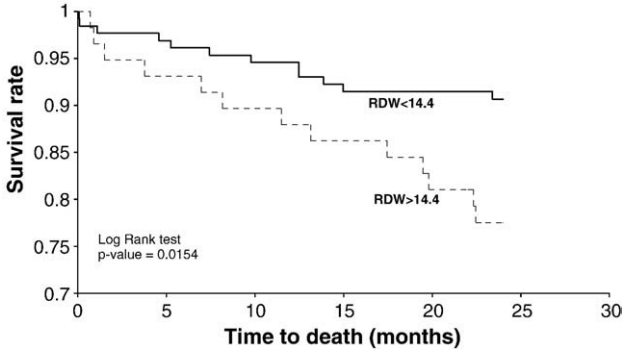

Fig. 3. Kaplan–Meier curves for all-cause mortality comparing the upper tertile of baseline RDW values to the lower two tertiles combined in the ACS subpopulation of patients. At 24 months, the number of patients who had died in the upper tertile was 13 (22.4%), compared with 12 (9.3%) in the lower two tertiles combined ($p=0.0154$ by log rank test).

independent predictor of all-cause mortality at 24 months (HR 2.90, 95% CI 1.32–6.38, $p=0.0082$) when comparing the upper tertile to the combined lower two tertiles (Table 4).

Similar to the total population of patients, Kaplan–Meier survival analysis for the ACS subpopulation showed a significantly reduced survival in patients in the highest RDW tertile. At 24 months, the survival rate was 77.6% for patients in the highest tertile, compared with 90.7% for patients who were in the lower two tertiles combined ($p=0.0154$ by log rank test) (Fig. 3).

RDW was also a significant predictor of all-cause mortality in the non-ACS subpopulation of patients on univariate analysis, with a HR of 3.11 (95% CI 1.32 to 7.30, $p=0.0096$) when comparing the upper tertile to the combined lower two tertiles. However, after adjustment for other significant covariates, RDW was no longer an independent predictor of this outcome.

4. Discussion

The primary finding of this study was that increased RDW was a strong and independent predictor of mortality in an unselected population of males referred for coronary angiography. This association remained significant even after adjustment for a wide variety of clinically relevant covariates. These covariates included not only important laboratory and clinical parameters (such as hemoglobin and CHF on presentation), but also angiographic variables such as the extent of coronary artery disease and LV systolic function which are known to be powerful determinants of mortality. In our study, we also demonstrated that RDW was a powerful and independent predictor of mortality in the subpopulation of patients who had presented with ACS. To our knowledge, this is the first report in the literature demonstrating the prognostic significance of RDW in ACS.

Despite the well-known and established association between anemia and adverse cardiovascular outcomes in multiple patient populations, we do not believe that our findings were confounded by anemia. Not only was the association between RDW and mortality unaffected after adjustment for hemoglobin, but additional analyses restricted to patients who were not anemic demonstrated that RDW was still a powerful predictor of mortality in this subpopulation of patients.

Importantly, the magnitude of increased risk associated with higher values of RDW in our study was clinically meaningful. For example, the HR for all-cause mortality associated with having an RDW value in the upper tertile of baseline levels compared to having a value in the lower two tertiles was almost 3 in both the entire study population and in the ACS subpopulation, and close to 5 in the non-anemic subpopulation. These hazard ratios were higher than those associated with the other independent predictors of mortality. Thus, overall our findings extend the observations of two earlier studies demonstrating the prognostic significance of RDW in patients with heart failure [7] and in patients who were post-MI without heart failure [8] to a broad and unselected population referred for coronary angiography. Our population included patients with and without heart failure, patients with and without ACS, and patients with and without anemia. Thus, RDW appears to be a powerful and independent predictor of mortality in a broad population of patients across a spectrum of risk.

The mechanism by which elevated values of RDW are associated with increased mortality is unknown. RDW is a quantitative measure of anisocytosis, the variability in size of the circulating erythrocytes. It is a routine component of the complete blood count performed by automated hematology analyzers. An elevation in RDW may be seen in conditions of ineffective red cell production (such as iron deficiency, B12 or folate deficiency, and hemoglobinopathies), increased red cell destruction (such as hemolysis), or after blood transfusion [7,8]. Importantly, proinflammatory cytokines have been found to inhibit erythropoietin-induced erythrocyte maturation, which is reflected in part by an increase in RDW. Accordingly, some have proposed that higher values of RDW may reflect an underlying inflammatory state, [8] which is known to be associated with adverse clinical outcomes [11].

There are several limitations to this study. First, the study was exploratory and observational in nature and as such is subject to the limitations of this type of analysis. However, we had formulated the hypothesis that elevated RDW values were associated with adverse outcomes (based on review of the available literature in this regard) before performing analyses on our database. We therefore feel that this approach reduced the risk of spurious conclusions. Second, the size of our population was small and the study was not designed with a priori calculations with respect to sample size or statistical power. As such, the findings need to be confirmed in larger and prospectively designed studies. Third, the population was a high-risk cohort as evidenced by both clinical and laboratory parameters as well as by the high event rate for death. Therefore, it is unknown whether RDW values would be similarly predictive of events in a low-risk population. Fourth, although RDW was predictive of

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mortality in the non-anemic subpopulation, we did not perform similar analyses in the anemic cohort due to small sample size of this subset of patients. Therefore, it is unknown whether RDW values would be similarly predictive of mortality in anemic patients. Finally, this study was conducted in an exclusively male population and the results cannot be extrapolated to women, in whom the factors associated with anemia and anisocytosis may be different.

In conclusion, we found that elevated RDW was a strong and independent predictor of all-cause mortality in a broad population of male patients across a broad spectrum of risk who were referred for coronary angiography. Our findings are concordant with, and extend, the previous reports in the literature about the utility of RDW as an independent prognostic marker in patients with cardiovascular disease.

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The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [12].

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