Red Cell Distribution Width Predicts Long-Term Cardiovascular Outcomes in Patients with Chronic Coronary Syndrome

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Summary

Red cell distribution width (RDW) has been shown to be an independent risk factor for increased cardiovascular mortality, heart failure, and cardiovascular disease. However, the association between RDW and long-term clinical outcomes in patients with chronic coronary syndrome (CCS) remains uncertain. In this study, a total of 2,881 CCS patients who underwent their first percutaneous coronary intervention (PCI) and who had available data on pre-procedural RDW between 2002 and 2016 were enrolled. Of these, 1,827 without anemia and severe renal dysfunction were divided into quartiles based on their RDW values. The primary endpoint was a composite of all-cause death and non-fatal myocardial infarction. As a result, patients in the higher RDW quartile groups were more likely to be older and have chronic kidney disease. During a median follow-up of 6.2 years, 209 (11.4%) events were identified. Kaplan-Meier curves showed the highest RDW quartile group had a clearly higher incidence of the primary endpoint (log-rank $P = 0.0002$). The highest RDW group had a significantly higher risk of cardiovascular events compared with the lowest RDW group, even after adjustment for other risk factors (hazard ratio 1.95, 95% confidence interval 1.04-3.67, $P = 0.04$). Increasing RDW as a continuous variable was also associated with the incidence of the primary endpoint (hazard ratio 1.46 per 1% increase, 95% confidence interval 1.24-1.69, $P < 0.0001$). In conclusion, this study demonstrated that increased RDW was associated with worse clinical outcomes after elective PCI. Assessing pre-PCI RDW may be useful for risk stratification of CCS.

(Int Heart J 2022; 63: 1041-1047)

Key words: Coronary artery disease, Percutaneous coronary intervention, Atherosclerosis, Asian population

Red cell distribution width (RDW) is a parameter reflecting variability in circulating erythrocyte size, which can be easily measured during routine complete blood counts.1) Traditionally, RDW and mean corpuscular volume (MCV) are used in the differential diagnosis of anemia, particularly anemias that are microcytic (caused by iron deficiency) or macrocytic (due to vitamin B12 or folate deficiency).2) In addition to its traditionally known role, RDW is of interest for its potential impact on coronary artery disease (CAD), heart failure, and peripheral artery disease.3-5) Atherosclerosis is a chronic inflammatory disorder with a years long asymptomatic period and usually manifests as acute coronary events with occlusion of coronary arteries.6) RDW has also been reported to be related to underlying chronic inflammation, which induces red blood cell membrane deformability and changes in erythropoiesis.7) In fact, an epidemiologic study has suggested that RDW is associated with increased levels of high-sensitivity C-reactive protein (hs-CRP), a biomarker of inflammation.8,9) Therefore, RDW has been reported to have a possible causal relationship with CAD.10) Recently, the relationship between RDW and acute coronary syndrome (ACS) was initially studied.11-14) However, the association between RDW and long-term clinical outcomes in patients with chronic coronary syndrome (CCS) remains uncertain. The present study sought to examine the association between RDW levels and cardiovascular outcomes in CCS patients who underwent elective percutaneous coronary intervention (PCI).

Methods

Study population: In this observational study, the data of consecutive patients who underwent their first-time elective PCI at Juntendo University from January 2002 to December 2016 were analyzed. Patients without available data for RDW ($n = 1$) and patients with anemia (male Hb < 13.0 g/dL, female Hb < 12.0 g/dL, $n = 849$), or severe renal deficiency (estimated glomerular filtration rate (eGFR) < 30 mL/minute/1.73 m², $n = 205$) were excluded.13,14) Patients were divided into 4 groups according to their RDW values (< 12.7%, 12.7-13.0%, 13.1-13.5%, 13.6% and above).
The demographic data and information on coronary risk factors, medications, revascularization-related factors, and co-morbidities were prospectively collected and analyzed. Hypertension was defined as systolic blood pressure > 140 mmHg or diastolic blood pressure > 90 mmHg, or treatment with antihypertensive drugs. Dyslipidemia was defined as low-density lipoprotein cholesterol ≥ 140 mg/dL, high-density lipoprotein cholesterol ≤ 40 mg/dL, triglycerides ≥ 150 mg/dL, or treatment with lipid-lowering drugs. Diabetes mellitus was defined as hemoglobin A1c ≥ 6.5% or current treatment with insulin or oral hypoglycemic agents.16) Chronic kidney disease (CKD) was defined as eGFR < 60 mL/minute/1.73 m², calculated using the equation of the modification of diet in renal disease, modified with a Japanese coefficient using baseline serum creatinine.17) A current smoker was defined as a participant who currently smoked or who had stopped smoking within 1 year before PCI. Whole blood counting parameters were measured after overnight fasting by automated hematology analyzers [XE-2100 (2002-2008) and XE-5000 (2008-2016); Sysmex Corporation, Kobe, Japan]. Blood was collected in standardized dipotassium ethylenediaminetetraacetic acid tubes for these tests.

This study was performed in accordance with the Declaration of Helsinki and with the approval of our institutional review board. Written informed consent was obtained from all patients before undergoing PCI.

Clinical outcome: The primary endpoint was the composite of all-cause death and non-fatal myocardial infarction (MI). The secondary outcome was all-cause death. MI was defined as clinical evidence of myocardial necrosis that was consistent with myocardial ischemia. Clinical event data were collected during the follow-up period for all patients by reviewing clinical charts, telephone contacts, and questionnaires. Mortality data were collected from the medical records of patients who died or who were treated at our institution. The details and causes of death were obtained from the other hospitals where the patients had been admitted.

Statistical analysis: Quantitative variables are presented as means and standard deviation (SD). Non-normally distributed variables are presented as medians with interquartile ranges (IQRs). A comparison of continuous numeric values between groups was performed using one-way analysis of variance or the Kruskal-Wallis test. Categorical variables were compared by the chi-squared test. Simple linear regression analysis was used for RDW and biochemical and clinical measurements. Pearson’s correlation coefficients were used to examine the relationships between RDW and other variables. The unadjusted cumulative event rates were compared across groups using Kaplan-Meier curves and the log-rank test. The effects of RDW values on clinical outcomes after PCI were determined using Cox multivariable proportional hazards regression analysis. Model 1 was adjusted for age and sex. Model 2 was adjusted for the variables in model 1 plus hypertension, dyslipidemia, diabetes mellitus, body mass index (BMI), CKD, and hemoglobin level. The RDW values were included in the multivariable model, and hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated. A \( P < 0.05 \) was considered significant. All analyses were performed using JMP 14.2.0 software (SAS Institute, Cary, NC, USA).

Results

Baseline characteristics: From among the entire population who underwent elective PCI between 2002 and 2016 at Juntendo University Hospital (\( n = 2,882 \)), after excluding patients with unavailable RDW data (\( n = 1 \)), anemia (\( n = 849 \)) and CKD grade 4 or more (\( n = 205 \)), the remaining 1,827 patients were enrolled in this study (Fig-
The distribution of RDW in the present study population is shown in Figure 2. The median and mean RDW was 13.0% (IQR, 12.7%-13.5%) and 13.2% ± 0.8%, respectively. The baseline characteristics of patients divided into quartiles of RDW values are listed in Table I. Patients in the higher RDW quartile groups were more likely to be older and have a higher prevalence of CKD, higher high-density lipoprotein cholesterol, higher hs-CRP, higher platelet counts, lower triglycerides, and lower fasting blood glucose levels. The prevalences of hypertension, diabetes mellitus, and dyslipidemia were not significantly different among the groups. The associations between RDW and other parameters were examined. The correlation of RDW level with age ($r = 0.16$, $P < 0.0001$), hemoglobin ($r = -0.10$, $P < 0.0001$), and hemoglobin A1c ($r = 0.05$, $P = 0.04$) were significant, but relatively weak. The hs-CRP level was not significantly related to RDW.

Additionally, in individuals in the highest quartile of RDW, comparisons of background demographics with and without subsequent cardiovascular (CV) events showed significantly higher age, lower BMI, higher incidence of CKD, and higher hs-CRP in patients with events, indicating potential associations between these parameters and poor outcomes in patients with elevated RDW.

Clinical outcome: During a median follow-up of 6.2 years (IQR, 2.5-10.7 years), 209 (11.4%) primary endpoints were identified. Kaplan-Meier curves showed that the highest RDW quartile group had clearly the highest incidence of the primary endpoint (Figure 3, log-rank $P = 0.0002$). Similar results were also obtained in Kaplan-Meier analysis for all-cause death (Figure 4, log-rank $P < 0.0001$).

Table II shows the Cox proportional hazard analyses for the primary endpoint. In the unadjusted Cox model, the highest RDW group had a significantly higher risk of cardiovascular events compared with the lowest RDW group ($P = 0.0002$). In Model 2, the highest RDW group had more increased risk than the lowest RDW group (hazard ratio 1.95, 95% confidence interval 1.04-3.67, $P = 0.04$). Increasing RDW as a continuous variable was also associated with the incidence of the primary endpoint, even adjusted for other risk factors (hazard ratio 1.46 per RDW 1% increase, 95% confidence interval 1.24-1.69, $P < 0.0001$).

Discussion

The present study investigated the associations be-
tween pre-procedural RDW levels and long-term clinical outcomes in CCS patients who underwent PCI. The main findings were as follows: (1) patients in the higher RDW groups were more likely to be older and have a higher incidence of CKD, whereas the prevalences of other coronary risk factors, such as hypertension, diabetes mellitus, and dyslipidemia, were not different among the groups; (2) patients in the higher RDW group had a significantly higher incidence of the primary endpoint compared with patients in the lower RDW group; and (3) a higher RDW value was associated with a higher risk of long-term cardiovascular events, even after adjustment for other risk factors.

Table I. Patient Clinical Characteristics

| Baseline characteristic | Overall n = 1,827 | Q1 n = 450 | Q2 n = 468 | Q3 n = 466 | Q4 n = 443 | P value |
|--------------------------|-----------------|------------|------------|------------|------------|---------|
| **RDW, %**               | 13.0 [12.7, 13.5] | 12.4 [12.2, 12.5] | 12.9 [12.8, 13.0] | 13.3 [13.2, 13.4] | 14.0 [13.7, 14.3] | < 0.0001 |
| **Baseline characteristic** |                |            |            |            |            |         |
| Age, years               | 64.9 ± 9.6      | 62.9 ± 9.9 | 64.3 ± 9.5 | 65.0 ± 9.6 | 67.3 ± 9.0 | < 0.0001 |
| Male, n (%)              | 1,576 (86.3)    | 398 (88.4) | 395 (84.4) | 412 (88.4) | 371 (83.7) | 0.06    |
| Hypertension, n (%)      | 1,315 (72.0)    | 315 (70.0) | 348 (74.4) | 338 (72.5) | 314 (70.9) | 0.47    |
| Diabetes mellitus, n (%) | 778 (42.6)      | 190 (42.2) | 195 (41.7) | 202 (43.4) | 191 (43.1) | 0.95    |
| Dyslipidemia, n (%)      | 1,425 (78.0)    | 359 (79.8) | 378 (80.8) | 354 (76.0) | 334 (75.4) | 0.12    |
| Current smoking, n (%)   | 460 (25.2)      | 97 (21.6)  | 123 (26.4) | 127 (27.3) | 113 (25.5) | 0.20    |
| Family history, n (%)    | 536 (29.4)      | 128 (28.4) | 154 (33.1) | 134 (28.8) | 120 (27.2) | 0.23    |
| Multivessel disease, n (%) | 1,073 (57.8)  | 257 (57.1) | 298 (63.7) | 265 (56.9) | 253 (57.1) | 0.10    |
| BMI, kg/m²               | 24.7 ± 3.3      | 24.6 ± 3.2 | 24.9 ± 3.3 | 24.9 ± 3.3 | 24.6 ± 3.3 | 0.34    |
| TC, mg/dL                | 177.4 ± 35.2    | 178.0 ± 35.2 | 177.2 ± 34.9 | 178.0 ± 35.5 | 176.4 ± 35.2 | 0.89    |
| LDL-C, mg/dL             | 104.5 ± 30.9    | 104.9 ± 30.7 | 104.6 ± 32.0 | 104.9 ± 30.7 | 103.5 ± 30.0 | 0.89    |
| HDL-C, mg/dL             | 44.4 ± 12.8     | 43.3 ± 11.8 | 43.4 ± 11.8 | 44.3 ± 11.8 | 44.6 ± 13.2 | 0.001   |
| TG, mg/dL                | 129 [96, 173]   | 131 [98, 179] | 134 [102, 178] | 126 [97, 173] | 120 [90, 162] | 0.003   |
| FBG, mg/dL               | 108.4 ± 31.6    | 110.2 ± 31.2 | 109.8 ± 35.5 | 108.5 ± 29.3 | 104.8 ± 29.7 | 0.04    |
| HbA1c, %                 | 6.3 ± 1.1       | 6.4 ± 1.2   | 6.3 ± 1.1   | 6.4 ± 1.1   | 6.3 ± 0.9   | 0.86    |
| HS-CRP, mg/dL            | 0.08 [0.04, 0.21] | 0.07 [0.03, 0.19] | 0.08 [0.03, 0.21] | 0.08 [0.04, 0.19] | 0.10 [0.04, 0.25] | 0.002   |
| White blood cells, μL    | 5,600 [4,800, 6,700] | 5,550 [4,600, 6,500] | 5,600 [4,700, 6,600] | 5,600 [4,800, 7,000] | 5,700 | 0.07 |
| Hemoglobin, g/dL         | 14.2 ± 1.0      | 14.3 ± 1.0  | 14.3 ± 1.1  | 14.2 ± 1.0  | 14.1 ± 1.1  | 0.22    |
| Platelet count, ×1000/μL | 203.1 ± 52.6   | 198.9 ± 46.0 | 200.6 ± 51.0 | 204.5 ± 53.8 | 208.8 ± 58.6 | 0.02    |
| **ACE-I/ARB, n (%)**     | 860 (47.5)      | 208 (46.9)  | 221 (47.4)  | 217 (47.1)  | 214 (48.8)  | 0.94    |
| **β-Blocker, n (%)**     | 961 (53.1)      | 245 (55.1)  | 255 (54.7)  | 234 (50.8)  | 227 (51.7)  | 0.47    |
| **OHA, n (%)**           | 506 (27.7)      | 123 (27.3)  | 125 (26.7)  | 130 (27.9)  | 128 (28.9)  | 0.90    |
| **Insulin, n (%)**       | 95 (5.2)        | 17 (3.8)    | 22 (4.7)    | 25 (5.4)    | 31 (7.0)    | 0.17    |
| **Statin, n (%)**        | 1,261 (69.7)    | 313 (70.3)  | 329 (70.6)  | 310 (67.4)  | 309 (70.4)  | 0.68    |

ACE-I indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; CKD, chronic kidney disease; FBG, fasting blood glucose; HbA1c, hemoglobin A1c; HDL-C, high-density lipoprotein cholesterol; LAD, left anterior descending artery; LDL-C, low-density lipoprotein cholesterol; HS-CRP, high-sensitive C-reactive protein; OHA, oral hypoglycemic agent; RDW, red cell distribution width; TC, total cholesterol; and TG, triglycerides.

In addition to the diagnosis of different types of anemia, RDW has received attention because of its potential impact on heart failure and myocardial infarction. Roland, et al. found that RDW independently predicts 1-year mortality in acute heart failure. The correlation between RDW and clinical outcomes in patients with ACS has been reported in many studies. Nabais, et al. studied 1,796 patients with ACS admitted to a coronary care unit and found that a high RDW value was an independent predictor of 6-month mortality. Another study showed that a high RDW value was independently associated with the development of stent thrombosis in patients with ST-elevation MI. In a meta-analysis that included 13 studies involving 10,410 ACS patients, it was also demonstrated that a low RDW value was related to a lower risk for cardiovascular events. However, there is a paucity of data concerning the
association between clinical outcomes and RDW values in CCS patients, especially in Asian populations. We have previously reported the association of the RDW value with mortality risk in 560 patients with CCS and diabetes mellitus.23) In the present study, consecutive CCS patients who underwent PCI regardless of diabetes mellitus were enrolled and followed-up for a much longer duration. Therefore, the usefulness of RDW as a predictor of cardiovascular events in CCS patients was able to be better shown than before.

Although the pathophysiological mechanisms explaining the association of RDW with an increase in cardiovascular events are yet to be elucidated, chronic subclinical inflammation may play a significant role.24) Chronic subclinical inflammation brings about changes in red blood cell maturation by disturbing the maturation of the red cell membrane, thereby leading to increased RDW.25) Furthermore, it has already been mentioned that
inflammatory processes play a major role in cardiovascular events, suggesting that inflammation might be a contributing factor in the association between CAD and RDW.\(^3\) While hs-CRP has been reported to be an inflammatory marker of atherosclerosis,\(^27,28\) there was no significant association between RDW and hs-CRP in the present study. Other studies, which showed a correlation between RDW and CCS, also found no confounding effect of hs-CRP on RDW.\(^3\) A possible reason for no clear association between hs-CRP and RDW in the present study could be that RDW is multifactorial, involving factors such as oxidative stress, tissue hypoxia, neurohumoral hyperactivity, and endothelial dysfunction, in addition to inflammation.\(^20,29\) Moreover, increased RDW is thought to be caused by a variety of underlying metabolic abnormalities, low nutritional status, red blood cell fragmentation, and altered erythropoietin function.\(^31\) CKD is one of the diseases contributing to RDW. CKD leads to anemia, endothelial dysfunction, systemic inflammation, malnutrition, and accelerated atherosclerosis. These conditions are likely to increase RDW by promoting microvascular hypoxia and chronic cytokine exposure in the bone marrow.\(^32,33\) CKD has also been reported to be associated with prognosis in patients with CAD.\(^34\) In this study, CKD was also significantly more prevalent in the RDW-Q4 group, both in patients who had an event and in those who did not, suggesting that the combination of CKD and RDW may allow for better risk stratification in the future.

This study has several limitations. First, it was a single-center, observational study with a small patient cohort. Therefore, unknown confounding factors might have affected the outcomes, regardless of the analytical adjustments. Second, we evaluated RDW only once and did not assess changes over time.

**Conclusion**

This study demonstrated that increased RDW was associated with worse clinical outcomes in patients with CCS. Pre-procedural RDW values might be useful for risk stratification of CCS patients scheduled for PCI. Based on these findings, further studies are needed to validate the clinical benefit of RDW.

**Acknowledgments**

The authors are grateful to the staff of the Department of Cardiovascular Medicine and Biology at Juntendo University and the Department of Cardiology at Juntendo University Shizuoka Hospital. The authors also wish to express their appreciation for the secretarial assistance of Ms. Yumi Nozawa.

**Disclosure**

**Conflicts of interest:** The authors declare that they do not have anything to disclose regarding conflicts of interest with respect to this manuscript.

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