Role of elevated red cell distribution width on acute kidney injury patients after cardiac surgery

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Abstract

Background: The aim of the study was to explore associations between elevated red cell distribution width (RDW) and acute kidney injury (AKI) in patients undergoing cardiac surgery (CS-AKI).

Methods: Preoperative, intraoperative and postoperative data of 10,274 patients undergoing cardiac surgery, including demographic data, were prospectively collected from January 2009 to December 2014. Propensity score matching was used on the basis of clinical characteristics and preoperative variables. An elevated RDW was defined as the difference between RDW 24 h after cardiac surgery and the latest RDW before cardiac surgery.

Results: A total of 10,274 patients were included in the unmatched cohort, and 3146 patients in the propensity-matched cohort. In the unmatched cohort, the overall CS-AKI incidence was 32.8% (n = 3365) with a hospital mortality of 5.5% (n = 185). In the propensity-matched cohort, the elevated RDW in AKI patients was higher than in patients without AKI (0.3% (0.0%, 0.7%) vs 0.5% (0.1, 1.1%), P < 0.001) and the elevated RDW incidences were 0.4% (0.1%, 0.9%), 0.6% (0.2%, 1.1%) and 1.1% (0.3%, 2.1%) in stage 1, 2 and 3 AKI patients (P < 0.001). Among propensity-matched patients with CS-AKI, the level of elevated RDW in non-survivors was higher than in survivors (1.2% (0.5%, 2.3%) vs 0.5% (0.1%, 1.0%), P < 0.001) and a 0.1% increase in elevated RDW was associated with a 0.24% higher risk of within-hospital mortality in patients with CS-AKI. Estimating the receiver-operating characteristic (ROC) area under the curve (AUC) showed that an elevated RDW had moderate discriminative power for AKI development (AUC = 0.605, 95% CI, 0.586–0.625; P < 0.001) and hospital mortality (AUC = 0.716, 95% CI, 0.640–0.764; P < 0.001) in the propensity-matched cohort.

Conclusions: An elevated RDW might be an independent prognostic factor for the severity and poor prognosis of CS-AKI.

Keywords: Acute kidney injury, Cardiac surgery, Prognosis, Red cell distribution width

Background

Red blood cell distribution width (RDW), which is a marker that describes the morphology of red blood cells and is routinely reported in complete blood counts, is a measurement of erythrocyte variability and heterogeneity. RDW can be expressed either in absolute values (RDW-SD) or as a percentage (RDW %); the latter approach is more widely used in routine laboratory practice. RDW s together with the value of mean corpuscular volume (MCV) to classify, diagnose and differentiate the causes of anemia, especially iron-deficiency anemia [1]. Recently, several studies have shown that a high RDW level is a strong independent predictor of increased morbidity and mortality in patients with heart failure, myocardial infarction, paroxysmal atrial fibrillation, primary biliary cirrhosis, chronic hepatitis C, pulmonary embolism, chronic obstructive pulmonary disease (COPD), leukoaraisis and drug-eluting stent restenosis [2–9]. However, there is limited research regarding the potential association between elevated RDW and the development, severity and prognosis of AKI associated with cardiac surgery (CS-AKI). The present study investigated the potential association between elevated RDW and the development...
and prognosis of CS-AKI patients in order to identify a novel biomarker for the early diagnosis, clinical severity and prognosis of CS-AKI.

Methods
Study population
We retrospectively analyzed data from patients who underwent cardiac surgery from January 2009 to December 2014 in the Zhongshan Hospital affiliated to Fudan University and matched them 1:1 based on propensity scoring. The criteria for exclusion were: age (< 18 years), anemia (hemoglobin < 120 g/L for males; hemoglobin < 110 g/L for females), solitary kidney or history of kidney transplants, severe renal dysfunction with estimated glomerular filtration rate (eGFR) ≤ 30 mL/min/1.73 m² at baseline; preoperative circulatory assist devices, preoperative renal replacement therapy (RRT) or those undergoing a heart transplant; insufficient examination results at baseline.

AKI and RDW definitions
AKI was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) [10] criteria as described recently [11]: 1) Increase in serum creatinine (Scr) by ≥ 0.3 mg/dL (≥ 26.5 μmol/L) within 48 h; 2) Increase in Scr to ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; 3) Urine volume < 0.5 mL/kg/h for 6 h and staged according to the Scr and urine output. An elevated RDW was defined as the difference between the RDW value 24 h after cardiac surgery and the latest RDW value before cardiac surgery. The reference range for RDW value was 11.0–16.0% at the Zhongshan Hospital affiliated to Fudan University.

Clinical trial design and methods
Informed consent was provided by all of the patients according to hospital guidelines. At admission, baseline blood testing, echocardiography and electrocardiography were performed. Recorded clinical data were preoperative baseline characteristics and blood tests, complications, preoperative renal function (determined by the latest serum creatinine value before cardiac surgery) as well as perioperative data about cardiac functions according to the 1994 New York Heart Association (NYHA) classification and intraoperative variables which included cardiac output data derived from echocardiography, cardiopulmonary bypass (CPB) and aortic cross-clamp (ACC) durations in minutes, as well as types of surgery. The recorded postoperative variables were mechanical ventilation duration and urine output during the first post-surgery 24 h as well as AKI incidence and stage in addition to hospital mortality.

Outcomes
The primary endpoint was the occurrence of AKI. The secondary endpoint was all cause of mortality.

Statistical analysis
All statistical analyses were performed using SPSS Statistics for Windows (Version 22.0. Armonk, NY: IBM Corp). Propensity score matching was used to adjust for observed differences in characteristics of patients with and without AKI. Comparisons of the differences in the baseline characteristics were performed using a Student’s t-test for parametric data and a Mann-Whitney U-test for non-parametric data. Categorical variables were compared using a chi-square test or Fisher’s exact test.

Assessments of pre-, intra-, post-operative and demographic data were tested with Wilcoxon rank-sum or two-sample t-tests for continuous and a Pearson χ² test for categorical variables. Continuous variables are presented as mean ± standard deviation or median (interquartile range [IQR]) and categorical variables are shown as frequency counts (%). A multivariable logistic regression model was used for estimation of unadjusted and adjusted odds ratios (ORs). Pearson’s and Spearman correlation tests were used to determine the correlation between variables. Receiver operator characteristic (ROC) curves were constructed to analyze the discriminating power of elevated RDW for predicting the development of AKI and all-cause hospital mortality. ROC curve analysis statistics were determined with area under the curve (AUC) (95% CI) estimated by bootstrapping. Two-tailed P-values < 0.05 were considered to be statistically significant for all analyses.

Results
Patient characteristics of the study population
Unmatched cohort
A total of 10,274 patients undergoing cardiac surgery were enrolled; most were men (56.7%). The overall CS-AKI incidence was 32.8% (n = 3365) with a hospital mortality of 5.5% (n = 185). The mean age at hospital admission was 53.3 years (± SD 13.9). The mean age, proportion of males mean body mass index (BMI), hemoglobin, white blood cells (WBC), serum creatinine (Scr), blood urea nitrogen (BUN), serum uric acid (SUA), CPB time, elevated RDW, preoperative proportions of hypertension, diabetes mellitus (DM), coronary angiography, chronic heart failure (NYHA > II), aortic cross-clamp (ACC) time were increased in the AKI group, whereas baseline albumin was decreased (Table 1).

Risk factors for the development of CS-AKI
All the variables recorded in Table 1 were put into a univariate logistic regression model and the detailed results are presented in Table 2. The odds ratios of CS-AKI in the unmatched cohort for the independent risk factors that were computed from the multivariate logistic regression model are shown in Table 3. The independent risk factors that were computed from the multivariate logistic regression
model were age (OR = 1.036, 95% CI: 1.029–1.042, P < 0.001), male (OR = 1.873, 95% CI: 1.622–2.164, P < 0.001), BMI (OR = 1.035, 95% CI: 1.016–1.055, P < 0.001), elevated RDW (OR = 1.302, 95% CI: 1.209–1.401, P < 0.001), BUN (OR = 1.076, 95% CI: 1.041–1.112, P < 0.001), SUA (OR = 1.002, 95% CI: 1.001–1.003, P < 0.001), CPB time (additional 30 min) (OR = 1.627, 95% CI: 1.491–1.775, P < 0.001).

The association between elevated RDW and the development and prognosis of CS-AKI
The AKI group had a higher level of elevated RDW than the non-AKI group [0.5% (0.1%, 1.0%) vs 0.3% (0.0%, 0.6%), P < 0.001]. Each 0.1% increment in elevated RDW was associated with a 1.1% higher risk of CS-AKI. The elevated RDW were 0.4% (0.1%, 0.9%), 0.5% (0.2%, 1.0%), 0.8% (0.3%, 1.7%) in stages 1, 2 and 3 of AKI, respectively. An increment in elevated RDW was associated with having a higher stage of AKI (P = 0.02). The patients were divided into two groups according to whether the RDW baseline was ≥16.0%. The incidence of CS-AKI in the RDW group (> 16.0%) was significantly higher than in the RDW group (≤ 16.0%, 33.9% vs 38.4%, P < 0.001). The adjusted odds ratio of CS-AKI development in the RDW group > 16.0% was 1.67-fold, compared with RDW ≤ in the 16.0% group. Among patients with CS-AKI, the level of elevated RDW in non-survivors was higher than survivors [1.1% (0.4%, 2.0%) vs 0.4% (0.1%, 0.9%), P < 0.001]. There was no significant difference in elevated RDW between non-survivors and survivors without CS-AKI [0.4% (0.0%, 0.6%) vs 0.3% (0.0%, 0.6%), P = 0.875]. A 0.1% increase in elevated RDW was associated with a 0.24% higher risk of within-hospital mortality in those patients with CS-AKI.

### Table 1 Comparison of the Demographic and Baseline Characteristics of unmatched and matched AKI and non-AKI groups

|                      | Non-AKI Cohort (n = 10,274) | P-value | Matched Cohort (n = 3,146) | P-value |
|----------------------|-----------------------------|---------|-----------------------------|---------|
|                      | Unmatched Cohort (n = 6,909) |         |                            |         |
| Age (years)          | 51.7 ± 14.0                 | < 0.001 | 54.6 ± 12.0                 | 0.782   |
|                      | 56.8 ± 12.3                 |         | 54.4 ± 12.1                 |         |
| Male, sex            | 3584 (51.9%)                | < 0.001 | 966 (61.4%)                 | 0.059   |
|                      | 2233 (66.6%)                |         | 913 (58.0%)                 |         |
| Comorbidities        |                             |         |                            |         |
| Hypertension         | 1748 (25.3%)                | < 0.001 | 422 (26.8%)                 | 0.177   |
|                      | 1177 (35%)                  |         | 457 (29.1%)                 |         |
| DM                   | 628 (9.1%)                  | < 0.001 | 139 (8.8%)                  | 0.498   |
|                      | 386 (11.5%)                 |         | 151 (9.6%)                  |         |
| Creatinine, μmol/L   | 75.7 ± 20.6                 | < 0.001 | 77.1 ± 17.3                 | 0.325   |
|                      | 83.6 ± 29.5                 |         | 77.8 ± 19.7                 |         |
| eGFR                 | 93.0 ± 22.1                 | < 0.001 | 91.5 ± 20.7                 | 0.566   |
|                      | 87.3 ± 25.3                 |         | 91.0 ± 24.1                 |         |
| BUN                  | 6.1 ± 2.1                   | < 0.001 | 6.2 ± 1.9                   | 0.063   |
|                      | 7.0 ± 2.8                   |         | 6.3 ± 1.8                   |         |
| Albumin baseline     | 40.6 ± 3.3                  | < 0.001 | 40.0 ± 3.4                  | 0.162   |
|                      | 40.1 ± 3.3                  |         | 39.9 ± 3.5                  |         |
| Hemoglobin, g/dL     | 136.5 ± 14.6                | 0.005   | 134.7 ± 17.0                | 0.093   |
|                      | 137.3 ± 15.9                |         | 133.6 ± 18.7                |         |
| WBC, 1000/μL         | 6.4 ± 2.0                   | 0.033   | 6.4 ± 2.4                   | 0.532   |
|                      | 6.5 ± 2.2                   |         | 6.4 ± 2.0                   |         |
| Elevated RDW, %      | 0.3 (0.0, 0.6)              | < 0.001 | 0.3 (0.0, 0.7)              | < 0.001 |
|                      | 0.5 (0.1, 1.1)              |         | 0.5 (0.1, 1.1)              |         |
| Platelet fl          | 188.8 ± 56.7                | < 0.001 | 189.2 ± 62.1                | < 0.001 |
|                      | 177.3 ± 56.4                |         | 178.2 ± 58.7                |         |
| BMI, kg/m²           | 22.9 ± 3.0                  | 0.013   | 22.6 ± 3.2                  | < 0.001 |
|                      | 23.5 ± 3.2                  |         | 23.3 ± 3.7                  |         |
| SUA                  | 351.0 ± 104.7               | < 0.001 | 355.6 ± 101.0               | < 0.001 |
|                      | 394.0 ± 138.6               |         | 377.0 ± 108.6               |         |
| NYHA functional class|                             |         |                            |         |
| I-II                 | 3420 (49.5%)                | < 0.001 | 755 (48.0%)                 | 0.001   |
|                      | 1370 (40.7)                 |         | 610 (38.8%)                 |         |
| III-IV               | 3489 (50.5%)                | < 0.001 | 818 (52.0%)                 | < 0.001 |
|                      | 1995 (59.3)                 |         | 963 (61.2%)                 |         |
| LVEF%                | 62.4 ± 8.3                  | 0.071   | 62.1 ± 8.1                  | 0.092   |
|                      | 61.4 ± 19.3                 |         | 61.3 ± 9.4                  |         |
| CPB time (min)       | 81.0 (65.0, 105.0)          | < 0.001 | 80.5 (65.0, 105.0)          | < 0.001 |
|                      | 101.0 (78.0, 128.0)         |         | 99.0 (78.0, 126.0)          |         |
| ACC time (min)       | 480.0 (36.0, 67.0)          | < 0.001 | 480.0 (37.0, 67.0)          | < 0.001 |
|                      | 58.0 (43.0, 78.0)           |         | 55.0 (41.0, 78.0)           |         |
| Pre-operative coronary angiography n (%) | 2543 (36.8) | < 0.001 | 690 (43.9%) | 0.009 |
|                      | 1528 (45.4)                 |         | 617 (39.2%)                 |         |
| Time interval between coronary angiogram and cardiac surgery (days) | 4 (2, 6) | 0.212 | 3 (2, 6) | 0.981 |

AKI acute kidney injury, BMI body mass index, DM diabetes mellitus, WBC white blood cell, RDW red cell distribution width, BUN blood urea nitrogen, eGFR estimated glomerular filtration rate SUA serum uric acid, NYHA New York Heart Association, LVEF left ventricular ejection fraction, CPB cardiopulmonary bypass, ACC Aortic cross-clamp

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Table 2 Univariate logistic regression analysis of risk factors for CS-AKI

|                | Before Propensity Matching | After Propensity Matching |
|----------------|----------------------------|---------------------------|
|                | OR  | 95% CI          | P-value     | OR  | 95% CI          | P-value     |
| Age            | 1.030 | 1.027–1.034     | < 0.001 | 0.999 | 0.993–1.005     | 0.782 |
| Male           | 1.825 | 1.680–1.994     | < 0.001 | 0.869 | 0.754–1.002     | 0.754 |
| BMI            | 1.066 | 1.051–1.081     | < 0.001 | 1.063 | 1.037–1.090     | < 0.001 |
| HTN            | 1.588 | 1.452–1.736     | < 0.001 | 1.117 | 0.956–1.305     | 0.164 |
| DM             | 1.305 | 1.145–1.487     | < 0.001 | 1.095 | 0.860–1.395     | 0.46  |
| Pre-operative coronary angiography | 1.421 | 1.313–1.553 | < 0.001 | 0.826 | 0.717–0.952 | 0.08  |
| Hb             | 1.004 | 1.001–1.007     | < 0.001 | 0.997 | 0.993–1.001     | 0.093 |
| WBC            | 1.019 | 1.002–1.042     | < 0.001 | 0.990 | 0.958–1.022     | 0.533 |
| Plt            | 0.996 | 0.996–0.997     | 0.633    | 0.997 | 0.996–0.998     | 0.601 |
| Elevated RDW  | 1.497 | 1.419–1.578     | < 0.001 | 1.192 | 1.126–1.262     | < 0.001 |
| BUN            | 1.178 | 1.147–1.193     | < 0.001 | 1.036 | 0.998–1.076     | 0.064 |
| Scr            | 1.015 | 1.013–1.017     | < 0.001 | 1.002 | 0.998–1.006     | 0.325 |
| eGFR           | 0.989 | 0.987–0.991     | 0.002    | 0.999 | 0.996–1.002     | 0.004 |
| UA             | 1.003 | 1.003–1.004     | < 0.001 | 1.002 | 1.001–1.003     | < 0.001 |
| Alb            | 0.954 | 0.941–0.965     | < 0.001 | 0.986 | 0.996–1.006     | 0.162 |
| CPB time (every 30 min) | 1.498 | 1.439–1.559 | < 0.001 | 1.490 | 1.385–1.603 | < 0.001 |
| ACC time (every 20 min) | 1.295 | 1.248–1.343 | < 0.001 | 1.253 | 1.176–1.336 | < 0.001 |

**Bivariate correlation analyses of elevated RDW and various clinical and laboratory parameters of the study population in the unmatched cohort**

The results of bivariate correlation analyses showed that there was a positive correlation between elevated RDW and age (r = 0.188, P < 0.001), HTN (r = 0.080, P < 0.001), DM (r = 0.052, P < 0.001), BUN (r = 0.039, P < 0.001), CPB time (r = 0.159, P < 0.001), ACC time (r = 0.136, P < 0.001), AKI stage (r = 0.171, P < 0.001) and a negative correlation with male (r = -0.096, P < 0.001), BMI (r = -0.045, P = 0.01), hemoglobin (r = -0.147, P < 0.001), albumin (r = -0.047, P < 0.001), and eGFR (r = -0.105, P < 0.001) (see Table 4).

Table 3 Multivariate logistic regression analysis of risk factors for CS-AKI in the unmatched cohort

|                | OR   | 95% CI          | P-value |
|----------------|------|-----------------|---------|
| Age            | 1.036 | 1.029–1.042     | < 0.001 |
| Male           | 1.873 | 1.622–2.164     | < 0.001 |
| BMI            | 1.035 | 1.016–1.055     | < 0.001 |
| Elevated RDW  | 1.302 | 1.209–1.401     | < 0.001 |
| BUN            | 1.076 | 1.041–1.112     | < 0.001 |
| SUA            | 1.002 | 1.001–1.003     | < 0.001 |
| CPB time (every 30 min) | 1.627 | 1.491–1.775 | < 0.001 |

**Propensity-matched cohort**

Propensity score matching created a matched cohort of 1573 in each group. In this matched cohort, few differences remained in non-AKI and AKI groups (Table 1). The results of univariate logistic regression model are presented in Table 2. The odds ratios of CS-AKI in the matched cohort for the independent risk factors that were computed from the multivariate logistic regression model are shown in Table 5. In the matched cohort, the elevated RDW in AKI patients was higher than in patients without AKI (0.3% (0.0%, 0.7%) vs 0.5% (0.1, 1.1%), P < 0.001). The elevated RDW incidences were 0.4% (0.1%, 0.9%), 0.6% (0.2%, 1.1%) and 1.1% (0.3%, 2.1%) in stage 1, 2 and 3 AKI patients (P < 0.001). Among patients with CS-AKI, the level of elevated RDW in non-survivors was higher than in survivors [1.2% (0.5%, 2.3%) vs 0.5% (0.1%, 1.0%), P < 0.001] and a 0.1% increase in elevated RDW was associated with a 0.24% higher risk of within-hospital mortality in patients with CS-AKI.

**Receiver-operating characteristic curve analysis for prediction of the development and prognosis of CS-AKI in the matched cohort by elevated red cell distribution width level**

To assess discrimination of RDW for all causes of hospital mortality, we used receiver-operating characteristic (ROC) analysis and determined the area under the curve (AUC). The cut-off value of elevated RDW for predicting CS-AKI...
Table 4 Bivariate correlation analyses of elevated RDW and various clinical and laboratory parameters of the study population in the unmatched Cohort

| Parameter | r   | p-value |
|-----------|-----|---------|
| Age       | 0.188 | < 0.001 |
| Male      | -0.096 | < 0.001 |
| BMI       | -0.045 | 0.01 |
| HTN       | 0.098 | < 0.001 |
| DM        | 0.052 | < 0.001 |
| Hb        | -0.147 | < 0.001 |
| WBC       | -0.010 | 0.292 |
| Alb       | -0.047 | < 0.001 |
| BUN       | 0.039 | < 0.001 |
| Scr       | 0.020 | 0.040 |
| eGFR      | -0.105 | < 0.001 |
| SUA       | -0.010 | 0.292 |
| CPB time  | 0.159 | < 0.001 |
| ACC time  | 0.136 | < 0.001 |
| AKI stage | 0.171 | < 0.001 |

BMI: body mass index, HTN: hypertension, DM: diabetes mellitus, Hb: hemoglobin, WBC: white blood cell, Alb: albumin, BUN: blood urea nitrogen, Scr: serum creatinine, eGFR: estimated glomerular filtration rate, SUA: serum uric acid, CPB: cardiopulmonary bypass, ACC: Aortic cross-clamp, AKI: acute kidney injury.

Elevated RDW had moderate discriminative power for predicting the death of CS-AKI patients. The cut-off value of elevated RDW for predicting death of CS-AKI patients was 0.75%, AUC value 0.716 (95% CI: 0.640–0.764, P < 0.001) and the sensitivity, specificity, positive likelihood ratio and negative likelihood ratio of elevated RDW were 51.6%, 63.3%, 1.41 and 0.76, respectively.

The main finding of the present study was the establishment of an independent association between elevated RDW and in-hospital mortality with CS-AKI. An elevated RDW remains a significant predictor for the severity and mortality of CS-AKI patients following multivariable adjustments. However, there was no clear evidence to show that the elevated RDW was a significant predictor for the development of CS-AKI and there was no significant effect modification between an elevated RDW and in-hospital mortality in those patients without CS-AKI.

Table 5 Multivariate logistic regression analysis of risk factors for CS-AKI in the matched Cohort

| Parameter   | OR    | 95% CI            | p-value |
|-------------|-------|-------------------|---------|
| BMI         | 1.078 | 1.046–1.111       | < 0.001 |
| Elevated RDW| 1.159 | 1.074–1.251       | < 0.001 |
| eGFR        | 1.006 | 1.001–1.010       | 0.015   |
| SUA         | 1.002 | 1.001–1.003       | < 0.001 |
| CPB time (every 30 min) | 1.493 | 1.363–1.635 | < 0.001 |

AKI: acute kidney injury, BMI: body mass index, RDW: red cell distribution width, eGFR: estimated glomerular filtration rate, SUA: serum uric acid, CPB: cardiopulmonary bypass.
Related research [30–34] has demonstrated that oxidative stress increases anisocytosis by disrupting erythropoiesis and altering blood cell membrane deformability and thus the red blood cell circulation lifetime, ultimately leading to an increase in RDW. In addition, during CPB, ischemia and reperfusion play a pivotal role in oxidative stress by initiating a series of biochemical events [22]. However, in our study, we were not able to adjust the analysis to include inflammatory markers, C-reactive protein (CRP) and other factors, as these were not routinely measured at patient admission.

The renin-angiotensin-aldosterone system (RAAS) is activated by arterial underfilling caused by CPB, heart failure, shock and so on after cardiac surgery. The activation of the RAAS system and adrenocortical hormone release could cause increased RDW with erythropoiesis, resulting in a poor prognosis [34]. There is evidence suggesting that angiotensin II acts as a growth factor for erythroid precursors as well as an erythropoietin secretagogue resulting in an increment in RDW due to macrocytosis from skipped cell divisions [35].

RDW is increased in conditions such as malnutrition and deficiencies such as in vitamin B12 [36], iron [37] or folic acid [38], increased red cell destruction (such as hemolysis), or after blood transfusion. Previous research has reported that RDW was correlated negatively with nutrition in heart failure [39] and HD patients [13]. This latter study showed that elevated RDW was negatively correlated with the albumin level. Patients with malnutrition have a higher risk of infection and adverse outcomes and elevated RDW and poor prognosis may relate to the fact that there is an interaction between malnutrition and inflammation.

The present research work has several limitations. First, it was a retrospective design from a single central pool of patients. Second, we did not measure hematopoietic factors (including iron, vitamin B12, folate, etc.) that may influence RDW. Third, though neurohumoral activation and inflammation may be a mechanistic link between elevated RDW and poor outcomes, we did not assess laboratory markers including CRP, other pro-inflammatory cytokines, norepinephrine and angiotensin II. Despite these limitations, a major strength of the present study is that it had a sufficient number of patients undergoing cardiac surgery to ensure adequate reliability of our incidence and mortality estimates. Future studies of a larger cohort of single center pool patients will be required to confirm the results of the present investigation.

Conclusion

The results of the present study suggest, that an elevated RDW might be an independent prognostic factor for the severity and poor prognosis of CS-AKI.

Abbreviations

ACC: Aortic cross-clamp; AKI: Acute kidney injury; AUC: Area under the curve; BMI: Body mass index; BUN: Blood urea nitrogen; CI-AKI: AKI in patients undergoing cardiac surgery; CVD: Cardiovascular disease; DM: Diabetes mellitus; eGFR: Estimated glomerular filtration rate; ESRD: End stage renal disease; IQR: Interquartile range; KDIGO: Kidney Disease Improving Global Outcomes; LOS: Length-of-stay; MCV: Mean corpuscular volume; ORs: Odds ratios; PCI: Percutaneous coronary interventions; PD: Peritoneal dialysis; RAAS: Renin-angiotensin-aldosterone system; RDW: Red cell distribution width; ROC: Receiver-operating characteristic; RRT: Renal replacement therapy; Scr: Serum creatinine; SUA: Serum uric acid; WBC: White blood cells;
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