Journal of Cardiology 74 (2019) 212–216

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Original article

Red blood cell distribution width as a prognostic factor in patients undergoing transcatheter aortic valve implantation

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ARTICLE INFO

Article history:
Received 12 December 2018
Received in revised form 12 March 2019
Accepted 9 April 2019
Available online 3 May 2019

Keywords:
Aortic valve stenosis
Transcatheter aortic valve implantation
Red blood cell distribution width
Biomarkers
Risk scores

ABSTRACT

Background: Red blood cell distribution width (RDW), which is routinely reported in complete blood counts, is a measure of the variability in size of circulating erythrocytes. RDW is an independent predictor of prognosis in patients with cardiovascular diseases. We evaluated the short- and long-term prognostic value of RDW in a large cohort of transcatheter aortic valve implantation (TAVI) patients.
Methods: The impact of RDW on outcome was determined prospectively in 1029 consecutive patients with severe aortic stenosis (AS) undergoing transfemoral TAVI. The cohort was divided into 2 groups according to RDW above and below 15.5%. Collected data included patient characteristics, medical background, left ventricle ejection fraction (LVEF), frailty score, Society of Thoracic Surgeons (STS) score, periprocedural laboratory results, and long-term (up to 7.5 years) clinical outcomes.
Results: The mean age (±SD) was 83.1 ± 6.3 years, mean STS score was 4.2 ± 3.1% and mean estimated LVEF was 55.7 ± 8.4%. Mean pre-TAVI RDW levels were 15.3 ± 3.2%. Patients with RDW ≤ 15.5% (n = 683) and RDW > 15.5% (n = 346) had a 1-year mortality rate of 6% and 17%, respectively (p = 0.001) and a 5-year mortality rate of 20% and 38%, respectively ($p < 0.001$). Baseline RDW > 15.5% was independently associated with all-cause mortality (hazard ratio 1.83, 95% confidence interval 1.44–2.32, $p < 0.001$).
Conclusions: Elevated RDW is a strong independent marker and predictor of short- and long-term mortality following TAVI, that might present a relevant future supplement to current preprocedural risk scores. Additional research is needed to clarify the mechanisms responsible for this finding.

Introduction

Red blood cell distribution width (RDW) is a numerical measure of the variability in size of circulating erythrocytes. Higher RDW values indicate greater variation in size. The normal RDW range in human red blood cells is 11.5–14.5%. RDW has been used to differentiate between the causes of anemia. It is routinely reported as part of the complete blood count [1]. Anisocytosis is an increase in RDW that may be caused by iron deficiency anemia, vitamin B12 or folate deficiency, chronic liver disease, and myelodysplastic syndrome, all resulting in ineffective red blood cell (RBC) production and/or increased destruction. Elevated RDW levels have been linked to hepatic congestion, kidney injury, malnutrition, and inflammation-induced bone marrow dysfunction [2–4]. A higher RDW has also been shown to be a strong predictor of increased risk of morbidity and mortality in both cardiac patients and in the general population [5–8]. Previous studies from our group have shown that raised RDW correlates with increased risk for preclinical and clinical carotid atherosclerosis [9], higher rates of metabolic syndrome [10], increased long-term all-cause mortality in patients with ST-elevation myocardial infarction [11] and increased risk of cardiovascular morbidity and all-cause mortality in a large community cohort [12].

Transcatheter aortic valve implantation (TAVI) is an alternative for surgical aortic valve replacement in patients suffering from symptomatic severe aortic stenosis (AS) who are considered at intermediate or high risk for surgery [13]. In TAVI patients, surgical risk scores, such as Society of Thoracic Surgeons (STS) score and Logistic EuroSCORE provide only a moderate correlation for short-

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https://doi.org/10.1016/j.jjcc.2019.04.005
0914-5087/

and medium-term outcomes [14]. Dedicated scores are needed to properly tailor the right type and timing of procedure, as well as avoiding futility [15]. Cheap and easily attainable biomarkers, such as RDW, might help develop such risk scores.

The aim of this study was to evaluate the long-term prognostic value of baseline RDW in patients undergoing TAVI.

Methods

Patients

In this single-center study, we included 1029 consecutive patients with severe symptomatic aortic valve stenosis, referred for TAVI as decided by the local heart team, using the Medtronic CoreValve (Medtronic, Dublin, Ireland) (470 patients), Edwards SAPIEN XT (Edwards Lifesciences, Irvine, CA, USA) (290 patients), Medtronic Evolute-R (192 patients), Edwards SAPIEN 3 (42 patients), Lotus (Boston Scientific, Marlborough, MA, USA) (11 patients), or St. Jude Portico (St Paul, MN, USA) (5 patients) prosthesis between March 2009 and March 2017. Informed consent was obtained from each patient. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee.

Study design

Collected data included patient characteristics, medical history, echocardiographic data, surgical risk scores (STS score, EuroSCORE I and II), frailty assessment (according to the Katz index), and outcome variables.

Laboratory tests were performed within 24 h before TAVI. Hemoglobin, mean cellular volume (MCV), and RDW were measured using UniCel DxH 800 Coulter Cellular Analysis System (Beckman Coulter Inc., Brea, CA, USA) on blood collected in ethylenediaminetetraacetic acid (EDTA) tubes. Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease-4 (MDRD-4) formula [16].

In adherence to the VARC-2 consensus [17], we assessed our cohort for short-term and long-term complications, such as need for a pacemaker, vascular complications, bleeding, need for packed RBC transfusion, acute kidney injury (AKI), new stroke or transient ischemic attack, length of hospitalization, in-hospital mortality, 30-day mortality, 1-year mortality, and 5-year mortality.

Statistical analysis

All data were displayed as mean (±standard deviation) for continuous variables, and as the number (percentage) of patients in each group for categorical variables. For normally distributed variables we used Student's *t*-test. For non-parametric variables we used the Mann–Whitney test. For categorical variables we used χ^2 test or Fisher's exact test. Elevated RDW was defined as RDW > 15.5%, in accordance with previous studies done in this field [18,19]. This cut-off value was selected using receiver operating characteristic (ROC) for 30-day mortality with area under the curve (AUC) = 0.55 and was also chosen because it maximized sensitivity − (1 − specificity) in the 30-day mortality term, with a sensitivity of 0.478 and a specificity of 0.667. Odds ratios were calculated with a confidence interval of 95%. As mentioned above, we divided our cohort into two groups according to RDW above and below 15.5%. We created Cox regression models for assessing 1-year, 5-year, and long-term mortality with variables found to be associated with increased mortality in univariable analysis and in previous studies and variables found to be significantly different between the two RDW groups. They included the following variables: age, gender, medical history of diabetes mellitus, hyperlipidemia, atrial fibrillation, myocardial infarction and coronary artery bypass graft surgery, regular use of aspirin or alpha blockers, as well as preprocedural white blood cell levels, hemoglobin levels, eGFR, STS score, frailty, aortic valve mean pressure, ejection fraction, post-procedural paravalvular leak, and RDW levels below and above 15.5% using the Forward method. All of the analyses were considered significant at a 2-tailed *p*-value of <0.05. The SPSS statistical package was used to perform all statistical evaluation (SSPS, Chicago, IL, USA).

Results

Patients were stratified according to their RDW with a cut-off of 15.5% (≤15.5%, n = 683; >15.5%, n = 346). Differences in baseline characteristics between these two groups are shown in Table 1. Patients with elevated RDW had a significantly lower hemoglobin levels (11.3 ± 1.5 g/dL vs. 12.3 ± 1.4 g/dL, $p < 0.001$), lower eGFR (50.1 ± 19.6 mL/min/1.73 m^2 vs. 54.4 ± 16.4 mL/min/1.73 m^2, $p = 0.001$), lower estimated LVEF (53.3 ± 9.8% vs. 57 ± 7.1%, $p < 0.001$), and lower aortic valve mean pressure (44.1 ± 15.1 mmHg vs. 47.15 ± 14.7 mmHg, $p < 0.005$). A medical background of atrial fibrillation or flutter (46% vs. 24%, $p < 0.001$), as well as prior myocardial infarction (24% vs. 15%, $p < 0.001$) and prior coronary artery bypass graft surgery (20% vs. 14%, $p < 0.001$), were more prevalent in patients with elevated RDW. Furthermore, patients with elevated RDW were more frail (27% vs. 22%, $p < 0.05$) and had significantly higher STS score (5 ± 4.1% vs. 3.85 ± 2.4%, $p < 0.001$), EuroSCORE I (18.9 ± 12.8% vs. 14.6 ± 9.2%, $p < 0.001$) and EuroSCORE II (7.3 ± 6.4% vs. 5.4 ± 4.6%, $p < 0.001$).

Baseline RDW and short-term outcome

Patients with elevated RDW had a longer duration of hospitalization (8.1 ± 5.6 days vs. 7.7 ± 5.5 days, $p < 0.05$) and a higher rate of AKI (13% vs. 7%, $p = 0.05$) and were more likely to receive a packed RBC transfusion (21% vs. 14%, $p < 0.05$), but did not significantly differ in rates of need for a permanent pacemaker, new atrial fibrillation or flutter, bleeding or vascular complications, stroke or TIA and infections. There were non-significant higher rates of in-hospital mortality and of 30-day mortality among the elevated RDW group. Differences in periprocedural adverse events, short-term, and long-term outcomes are summarized in Table 2.

Baseline RDW and long-term survival

We found a significantly higher 1-year mortality rate [17% vs. 6%, hazard ratio (HR) 2.18, 95% confidence interval (CI) 1.37–3.47, $p = 0.001$] and 5-year mortality rate (38% vs. 20%, HR = 1.9, 95% CI 1.45–2.49, $p < 0.001$) among patients with elevated RDW. Variables significantly associated with all-cause mortality in univariable and multivariable analysis are shown in Table 3.

A Kaplan–Meier curve showing the survival difference according to RDW, after adjustment for the significant variables, is shown in Fig. 1.

Discussion

In the present study, we demonstrated that an elevated baseline RDW > 15.5% was associated with a more complex baseline clinical patient risk profile and associated with longer hospital stay, increased need for RBC transfusions, and higher rates of AKI. Moreover, we found that RDW > 15.5% is a strong independent predictor of mortality up to 7.5 years following TAVI.

According to a single-center study which included 168 patients with severe symptomatic AS who underwent TAVI using the

Table 1
Baseline characteristics according to RDW 15.5% cutoff.

Variable	RDW ≤ 15.5% (*n* = 683)	RDW > 15.5% (*n* = 346)	*p*-Value
Patient characteristics and medical history			
Age (years)	82.9 ± 5.8 (653)	83 ± 5.9 (337)	0.59
Gender (male)	41%	49%	**0.02**
Diabetes mellitus	36%	41%	0.11
Dyslipidemia	78%	79%	0.59
Hypertension	87%	87%	0.98
Current smoker	5%	4%	0.37
Atrial fibrillation/flutter	24%	46%	<**0.001**
Post MI	15%	24%	<**0.001**
Post CABG	14%	20%	**0.01**
Coronary artery disease	56%	61%	0.12
Laboratory			
WBC ($10^3/\mu L$)	8.1 ± 3.4	8.4 ± 3.6	0.48
Hemoglobin (g/dL)	12.3 ± 1.4	11.3 ± 1.5	<**0.001**
Albumin (g/L)	35.4 ± 3.5	35.1 ± 3.7	0.09
eGFR (mL/min/1.73 m^2)	54.4 ± 16.4	50.1 ± 19.6	**0.001**
Echocardiography			
LVEF (%)	57 ± 7.1	53.3 ± 9.8	<**0.001**
Aortic valve peak pressure (mmHg)	77 ± 22.8	72.9 ± 22.9	0.12
Aortic valve mean pressure (mmHg)	47.15 ± 14.7	44.1 ± 15.1	**0.004**
Pre-procedural risk			
Frailty	22%	27%	**0.048**
STS Score-Mortality (%)	3.85 ± 2.4	5 ± 4.1	<**0.001**
EuroSCORE I (%)	14.6 ± 9.2	18.9 ± 12.8	<**0.001**
EuroSCORE II (%)	5.4 ± 4.6	7.3 ± 6.4	<**0.001**
Medical treatment			
Aspirin	71%	60%	**0.001**
Statins	71%	72%	0.84
ACE-I/ARB	60%	60%	0.99
Beta blockers	59%	59%	0.96
Alpha blockers	46%	55%	**0.01**
Calcium blockers	24%	28%	0.16

ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; CABG, coronary artery bypass graft; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; MI, myocardial infarction; RDW, red cell distribution width; STS, Society of Thoracic Surgeons; WBC, white blood cells.
All P<0.05 are in [bold].

Table 2
Patients outcomes stratified by RDW.

Variable	RDW ≤ 15. 5% (*n* = 683)	RDW > 15.5% (*n* = 346)	*p*-Value
Moderate to severe paravalvular leak	1.5%	2%	0.5
In hospital new onset atrial fibrillation	4%	4%	0.88
In hospital major vascular complications	7%	5%	0.2
In hospital need for pacemaker transplantation	21%	18%	0.24
In hospital AKI	7%	13%	**0.005**
In hospital infection	4%	5%	0.42
In hospital major bleeding	11%	9%	0.38
In hospital need for packed RBC transfusion	14%	21%	**0.016**
In hospital stroke	1.1%	1.5%	0.56
In hospital mortality	2%	4%	0.28
Hospitalization days	7.7 ± 5.5	8.1 ± 5.6	**0.03**
30 days conversion to open surgery	0.6%	0.3%	0.67
30 days coronary obstruction	0.2%	0.3%	1
30 days ventricular septal perforation	0%	0%	1
30 days mitral valve damage	0.3%	0%	0.56
30 days tamponade	1.4%	0.6%	0.35
30 days valve migration/embolization	0.6%	1.5%	0.52
30 days mortality	2%	3%	0.15
1-Year mortality	6%	17%	<**0.001**
5-Year mortality	20%	38%	<**0.001**

AKI, acute kidney injury; RBC, red blood cells; RDW, red cell distribution width.
All P<0.05 are in [bold].

Medtronic CoreValve prosthesis, those who had elevated RDW (RDW > 15.5%) at baseline had worse characteristics at baseline and worse outcomes. The study demonstrated that patients who had elevated baseline RDW levels had significantly more post-procedural major vascular complications (10.3% vs. 1.8%, $p = 0.042$) and significant paravalvular leak (50.0% vs. 18.0%, $p = 0.001$) as quantified by echocardiography. One-year mortality was higher among these patients with RDW being the strongest predictor of 1-year mortality (HR 2.7, 95% CI 1.28–5.70, $p = 0.009$), even when the STS score was added to the model (HR 2.28, 95% CI 1.05–4.95, $p = 0.038$) [18].

In another single-center cohort study of 376 patients who underwent TAVI with either the Medtronic CoreValve system or the Edwards SAPIEN Valve, it has been demonstrated that anemia

Table 3
Cox regression results – all-cause mortality.

Variable	Univariable		Multivariable	
	HR	*p*-Value	HR	*p*-Value
RDW (15.5% cutoff)	2.14 (1.7–2.7)	<0.001	1.98 (1.54–2.56)	<0.001
STS score (per 1%)	1.1 (1.08–1.13)	<0.001	1.08 (1.05–1.1)	<0.001
Atrial fibrillation	1.85 (1.48–2.32)	<0.001	1.42 (1.1–1.84)	0.008
Age (per 1 year)	1.05 (1.02–1.07)	<0.001	1.03 (1.01–1.06)	0.01
Hemoglobin (per 1 g/dL)	0.86 (0.79–0.93)	<0.001		
eGFR (per 1 mL/min/1.73 m^2)	0.99 (0.98–1)	<0.001		
Aortic valve mean pressure (per 1 mmHg)	0.99 (0.98–1)	0.002		
LVEF (<30% vs. preserved)	2.4 (1.27–4.53)	0.007		
LVEF (30–44% vs. preserved)	1.11 (0.69–1.78)	0.664		
LVEF (45–54% vs. preserved)	1.36 (0.99–1.87)	0.054		
Frailty	1.34 (1.02–1.75)	0.033		
History of MI	1.37 (1.05–1.8)	0.022		

eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; MI, myocardial infarction; RDW, red cell distribution width; STS, Society of Thoracic Surgeons.

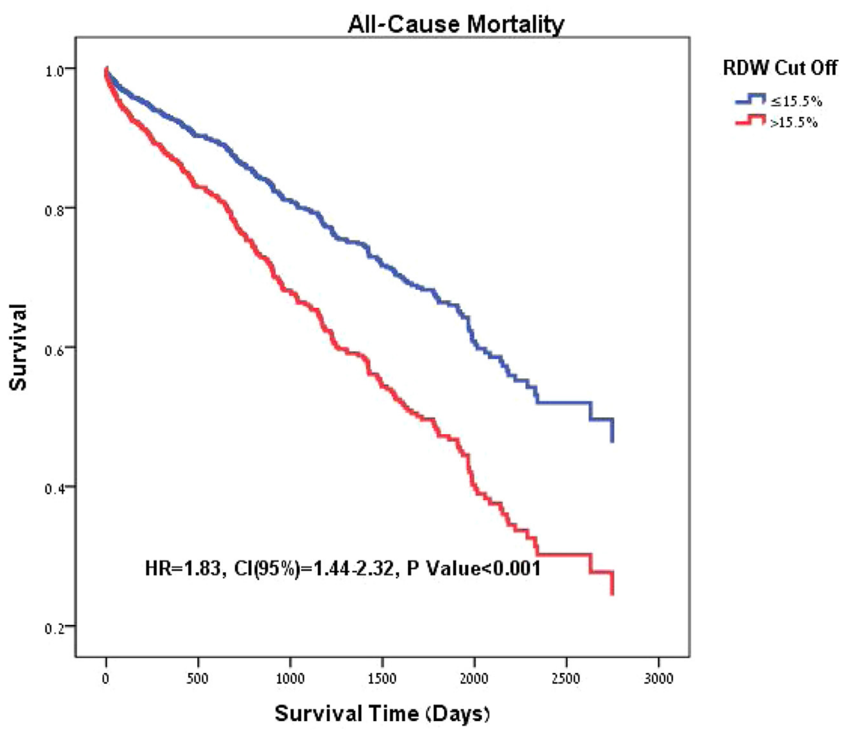

Fig. 1. A Kaplan–Meier curve showing the survival difference according to RDW after standardization for the significant variables. RDW, red blood cell distribution width.

did not predict long-term mortality, but RDW > 14% was a predictor of both short- and long-term mortality [20].

Another retrospective single center observational study of 175 TAVI patients showed similar results, with pre-TAVI RDW > 15.5% predicting death (HR 2.7, 95% CI 1.40–5.22, $p = 0.003$), independently of LVEF, baseline pulmonary artery systolic pressure, moderate/severe mitral regurgitation, and body mass index. It was also shown that a higher rate of increase in RDW over time was associated with increased mortality (adjusted HR 1.11, 95% CI 1.04–1.18, $p = 0.001$) independently of baseline RDW and other temporal changes in kidney and liver function. An increase in RDW > 0.1%/month was associated with a two-fold increased risk of mortality [19].

Our study is unique in its size (over 1000 patients) and in its long-term follow up (up to 7.5 years).

Since contemporary risk scores are not accurate enough in individual prediction of post-TAVI prognosis [21], our observations suggest a supplement that might expand the current risk scores.

The added value of various biomarkers to clinical risk scores was demonstrated in different studies [22]. An earlier study showed that growth differentiation factor (GDF)-15 enhanced prognostic performance of the EuroSCORE II for 1-year mortality and enabled substantial reclassification of patients [23]. The fact that RDW is an inexpensive and readily available biomarker further encourages its use in this context, in an attempt to promote a TAVI-specific risk score, that will allow better risk stratification and patient selection, and as we have showed, might help to estimate the risk of mortality following TAVI.

This study has an intrinsic limitation of being a single-center retrospective cohort study. Furthermore, one should consider the possibility that RDW is merely a marker for sicker patients who have worse prognosis to begin with. We approached this by using a multi-variable regression analysis in an attempt to control for identified confounders, such as variables found to be associated with increased mortality in our univariable analysis and in previous studies, as well as variables found to be significantly different between the two RDW groups. Nevertheless, the existence of unidentified confounders linked both to RDW levels and prognosis cannot be completely ruled out.

In conclusion, this study shows that elevated RDW is a strong independent predictor of short and long-term mortality following TAVI. This might encourage including this index into dedicated pre-TAVI prognostic scores. Additional research is needed in order to shed light on possible mechanisms, which may help elucidate the role of RDW as a causative factor and not simply a general marker for sicker patients. Future studies regarding interventions in an attempt to reduce RDW values might further emphasize the importance of this hematological index.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Funding

None declared.

Conflict of interests

All authors declare that they have no conflict of interest.

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