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Original Research Article

Correlation of red cell distribution width with the severity of coronary artery disease on coronary angiography as assessed by modified Gensini score in acute coronary syndrome patients

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ABSTRACT

Background: Coronary artery disease (CAD) remains a leading cause of global mortality, with Indian populations experiencing earlier onset and higher death rates. While traditional biomarkers aid diagnosis, their limitations in early detection necessitate alternative indicators. Red cell distribution width (RDW), an inexpensive and routinely measured hematological parameter, has emerged as a potential marker for cardiovascular risk stratification. Objectives were to evaluate the association of RDW with the severity of CAD assessed via the modified Gensini score (MGS) in patients presenting with acute coronary syndrome (ACS).
Methods: This cross-sectional observational study enrolled 500 consecutive patients presenting with ACS at a tertiary care center in Jaipur, India, between November 2018 and April 2020. All participants underwent diagnostic coronary angiography (CAG). CAD severity was quantified using the MGS mild: ≤80 and severe: >80. Complete blood count parameters, including RDW-CV and RDW-SD, were measured and analyzed across subgroups defined by disease severity, number of diseased vessels, myocardial infarction subtype, and in-hospital outcomes.
Results: Elevated RDW-CV (>13.7%) was significantly associated with severe CAD (p=0.009). Patients with higher RDW-CV had significantly higher Gensini scores (mean: 13.76±0.53% vs. 13.65±0.39%, p=0.048). However, no statistically significant associations were observed between RDW-CV or RDW-SD and the number of diseased vessels (p>0.05), infarction subtypes (p>0.05), or in-hospital mortality (p>0.05). Receiver operating characteristic (ROC) analysis demonstrated that an RDW-CV cutoff of >13.7% predicted severe CAD with an area under the curve (AUC) of 0.651 (95% CI: 0.549-0.744; sensitivity: 68.7%, specificity: 64.7%).
Conclusions: Elevated RDW-CV correlates significantly with angiographic severity of CAD in patients presenting with ACS. However, its capacity to discriminate multi-vessel involvement or acute in-hospital mortality remains limited.

Keywords: Red cell distribution width, Acute coronary syndrome, Modified Gensini score, Coronary angiography, Biomarkers

INTRODUCTION

Coronary artery disease (CAD) results from atherosclerotic narrowing and occlusion of the coronary arteries, impairing myocardial perfusion.[1] Atherosclerosis represents an insidious, progressive inflammatory condition that often remains clinically silent until presenting as ACS or sudden cardiac death. Globally, CAD is the foremost contributor to cardiovascular mortality, accounting for over 18 million deaths annually, with more than 80% occurring in low- and middle-income countries.[2] In India, the epidemiological burden is

pronounced: the estimated CAD mortality rate is 272 per 100,000 population, substantially exceeding the global average of 235 per 100,000. Furthermore, CAD in South Asian populations manifests prematurely, with over 50% of cardiovascular deaths occurring prior to age 70 compared to approximately 23% in Western cohorts.

Established modifiable risk factors-dyslipidemia, tobacco consumption, arterial hypertension, and type 2 diabetes mellitus-drive disease pathogenesis.[3-5] While cardiac-specific troponins and creatine kinase-MB (CK-MB) are essential for diagnostic confirmation, their clinical utility can be constrained in the ultra-early hours after symptom onset or in resource-limited triage facilities. With ongoing public health initiatives aiming to curb premature non-communicable disease mortality by 25%, identifying accessible, low-cost biomarkers to assist in early disease stratification is of paramount clinical importance.

RDW, reported routinely as part of automated complete blood counts (CBC), quantifies circulating erythrocyte anisocytosis.[6,7] Beyond its classical diagnostic role in differentiating anemias, RDW has gained attention as an integrative surrogate of systemic inflammation, oxidative stress, and neurohormonal activation.[8,10]

Pro-inflammatory cytokines (notably IL-1β, IL-6, and TNF-α) suppress erythropoietin gene expression, impair bone marrow iron utilization, and destabilize erythropoiesis, expanding circulating red cell size heterogeneity.[11]

While elevated RDW has been linked to adverse long-term cardiovascular prognosis, data evaluating its direct relationship with anatomical lesion severity assessed by validated angiographic scoring systems-such as the MGS-warrant comprehensive evaluation.

METHODS

Study design and setting

This observational, cross-sectional study was conducted in the Departments of Medicine and Cardiology at Sawai Man Singh Hospital, Jaipur, spanning from November 1, 2018, to April 30, 2020. Institutional Ethics Committee approval was obtained prior to study initiation, and written informed consent was secured from all participants or their legally acceptable representatives in their preferred language. The study conformed strictly to the ethical standards of the Declaration of Helsinki.

Participant selection

Consecutive adult patients (≥18 years) presenting with a confirmed diagnosis of ACS were included. ACS was categorized based on standard clinical criteria into ST-elevation myocardial infarction (STEMI) and non-ST elevation ACS (NSTEACS). Diagnosis was based on clinical symptoms indicative of myocardial ischemia, ECG abnormalities (e.g., ST changes, T wave inversions, new Q waves), positive cardiac biomarkers, and regional wall motion abnormalities observed on echocardiography. For cases presenting with unstable angina, the diagnosis was substantiated by coronary angiographic findings despite the absence of wall motion changes.

Exclusion criteria comprised baseline anemia, defined as hemoglobin <12.0 g/dl in males and <11.0 g/dl in females. History or clinical signs of heart failure (identified via clinical signs such as pedal edema, raised jugular venous pressure, or echocardiographic evidence of dysfunction). Previous revascularization procedures (PCI or CABG). Known valvular heart disease, pregnant women, individuals receiving estrogen therapy, patients with active malignancies or thrombotic thrombocytopenic purpura and patients who declined consent.

Baseline clinical and laboratory workup

After consent, each participant underwent detailed history-taking and clinical examination using a structured proforma. Demographic data, including age, weight, and height, were recorded. Venous blood samples were drawn upon presentation for complete blood count profiling, from which red cell indices-RDW-CV (%), RDW-SD (fL), MCV (fL), MCH (pg), and MCHC (g/dl)-were obtained. Routine biochemistries, including renal parameters and blood glucose, were documented.

Standard comorbidity definitions were applied:

Hypertension

Systolic BP>140 mmHg, diastolic BP>90 mmHg, or use of antihypertensive therapy.

Diabetes mellitus

Fasting glucose ≥126 mg/dl or the antidiabetic treatment.

Smokers

Those actively smoking or having the quit within the past year.

Positive family history of CAD

A first-degree relative developing CAD before age 55 in men or 65 in women.

CAG and severity scoring

All patients underwent diagnostic CAG, with stenotic lesions visualized in two or more projections. Standard pre-procedure medication included 300 mg of aspirin and 600 mg of clopidogrel, followed by 100 IU/kg of intravenous heparin. CAG was performed via either femoral or radial access using 6-French catheters and

iodinated contrast medium. Lesions causing >50% luminal narrowing were classified as significant.

CAD severity was evaluated using the MGS system.[12] Each coronary segment was scored for maximal luminal stenosis: 1 (1-49%), 2 (50-74%), 3 (75-99%), and 4 (100% occlusion).

Segment-specific multipliers were applied: left main artery (×5), left anterior descending (×20), diagonal branches (×10), septal perforators (×5), left circumflex and its branches (×10), right coronary artery (×20), and posterior descending artery (×10). disease severity was stratified into: Mild CAD the MGS≤80 and severe CAD were MSG>80.

Statistical analysis

Data were entered into Microsoft excel and analyzed using SPSS version 21.0. Categorical variables were reported as frequencies and percentages, while continuous data were expressed as mean±SD or median (interquartile range, IQR). Normality was assessed using the Kolmogorov-Smirnov test.

The Mann-Whitney U test and Kruskal-Wallis tests were used for non-parametric comparisons. Chi-square testing (χ^2) was applied for categorical variables. ROC curves were constructed to determine the predictive accuracy, sensitivity, and specificity of RDW-CV for severe CAD. A two-sided $p<0.05$ was considered statistically significant.

RESULTS

The study cohort comprised 500 patients presenting with ACS who satisfied all eligibility criteria. Table 1 details baseline demographic, clinical, and laboratory profile of cohort.

The mean age was 59.68±10.20 years, with 81.00% (n=405) male participants. Chest pain was the most common symptom (96.00%), followed by breathlessness (73.00%), diaphoresis (71.00%), and palpitations (53.00%). Prevalent comorbidities included hypertension (47.00%), diabetes mellitus (42.00%), and acute kidney injury (65.00%). A family history of CAD was documented in 95.00% of cases.

RDW distribution and relationship to angiographic severity

Overall, 63.00% (n=315) of the patients had an RDW-CV >13.7%, whereas 37.00% (n=185) had values ≤13.7%. The mean RDW-CV was 13.74±0.51%, and the median (IQR) was 13.80% (13.60-13.90%). Average values for RDW-SD, MCV, MCH, and MCHC were 44.89±0.77 fL, 89.65±3.65 fL, 29.49±0.88 pg, and 34.03±0.82 g/dl respectively.

A total of 83.00% (n=415) of patients had a MGS indicative of severe CAD, while 17.00% (n=85) had mild disease. The mean score was 110.10±28.59, with a median (IQR) of 100.0 (90.0-120.0).

A statistically significant difference was observed between RDW-CV groups and Gensini score categories (Table 2). Among patients with RDW-CV ≤13.7%, 29.73% had mild CAD and 70.27% had severe CAD. In contrast, among those with RDW-CV >13.7%, only 9.52% had mild disease and 90.48% had severe disease (χ^2=6.745, p=0.009).

Mean RDW-CV was significantly higher in severe CAD compared to mild CAD (13.76±0.53% vs. 13.65±0.39%, p=0.048). In contrast, the RDW-SD showed no statistically significant difference across the severity categories (44.90±0.78 fL vs. 44.84±0.73 fL and p value=0.528).

Vessel involvement, clinical presentation, and in-hospital outcomes

RDW-CV did not show a statistically significant correlation with the number of affected coronary vessels (p>0.05). Among patients with RDW-CV ≤13.7%, double-vessel disease was observed in 59.46%, single-vessel in 35.14%, and triple-vessel in 5.41%. In those with RDW-CV >13.7%, double-vessel disease occurred in 66.67%, single-vessel in 25.40%, and triple-vessel in 7.94%.

Median RDW-CV values across single-, double-, and triple-vessel disease were comparable at 13.80%. RDW-SD similarly lacked significant association across vessel distribution groups (p>0.05).

No significant link was observed between RDW-CV and MI subtypes (p>0.05). AWMI remained the most common (43.24% in RDW-CV ≤13.7% and 39.68% in RDW-CV>13.7%), followed by NSTEACS, inferior wall variants, and non-ST-elevation angina. Median RDW-CV was 13.80% across subtypes. RDW-SD also showed no statistical association (p>0.05) among subtypes.

Outcome-wise, in-hospital mortality was 5.41% in the RDW-CV≤13.7% group and 11.11% in the RDW-CV >13.7% group, but this difference did not achieve statistical significance (p>0.05). Median RDW-CV and RDW-SD values between survivors and non-survivors exhibited no significant differences.

Diagnostic accuracy and ROC analysis

ROC analysis indicated that RDW-CV had acceptable predictive value for severe CAD (AUC 0.651; 95% CI: 0.549-0.744, p=0.037). At a cutoff value of >13.7%, the sensitivity was 68.67%, specificity was 64.71%, positive predictive value (PPV) was 90.48%, negative predictive value (NPV) was 29.73%, and overall predictive accuracy was 65.10%.

Table 1: Baseline demographic, clinical, and laboratory characteristics (n=500).

Parameters	Value
Demographics	
Age (in years)	59.68±10.20
Male sex	405 (81.00%)
Risk factors and comorbidities	
Arterial hypertension	235 (47.00%)
Diabetes mellitus	210 (42.00%)
Positive family history of CAD	475 (95.00%)
Acute kidney injury	325 (65.00%)
Tobacco smoking	235 (47.00%)
Tobacco chewing	115 (23.00%)
Alcohol consumption	120 (24.00%)
Presenting clinical manifestations	
Chest pain	480 (96.00%)
Breathlessness (Dyspnea)	365 (73.00%)
Diaphoresis	355 (71.00%)
Palpitations	265 (53.00%)
Vitals and laboratory investigations	
Heart rate (beats/min)	84.0±12.0
Blood pressure (mmHg)	110/70
Hemoglobin (g/dl)	14.07±1.52
Total leukocyte count (×103/µl)	9.96±3.78
Serum creatinine (mg/dl)	1.10±0.36
Random blood sugar (mg/dl)	151.52±42.10
Left ventricular ejection fraction (LVEF, %)	44.50±14.60
ACS clinical subtypes	
Anterior wall MI (AWMI)	205 (41.00%)
Non-ST-elevation ACS (NSTEACS)	150 (30.00%)
Inferior/posterior/right ventricular MI (IWMI/IPWMI/PWMI/RVMI)	90 (18.00%)
Unstable angina / CSA presentation	55 (11.00%)
Coronary vessel involvement	
Single-vessel disease (SVD)	145 (29.00%)
Double-vessel disease (DVD)	320 (64.00%)
Triple-vessel disease (TVD)	35 (7.00%)

Table 2: Association of absolute indices with total Gensini score.

Absolute indices	Mild, (n=85)	Severe, (n=415)	Total, (n=500)	Test statistic	P value
RDW-CV stratification					
≤13.7%	55 (29.73%)	130 (70.27%)	185 (100.00%)	X^2=6.745	0.009
>13.7%	30 (9.52%)	285 (90.48%)	315 (100.00%)		
RDW-CV (%), continuous					
Mean±SD	13.65±0.39	13.76±0.53	13.74±0.51	U=492.5	0.048
Median (IQR)	13.70 (13.40-13.80)	13.80 (13.60-13.95)	13.80 (13.60-13.90)		
Range	12.90-14.60	11.60-14.50	11.60-14.60		
RDW-SD (fL), continuous					
Mean±SD	44.84±0.73	44.90±0.78	44.89±0.77	U=637.0	0.528
Median (IQR)	44.80 (44.60-45.20)	45.10 (44.55-45.60)	45.10 (44.58-45.60)		
Range	43.40-45.90	42.30-45.90	42.30-45.90		

DISCUSSION

Patients presenting with ACS are frequently encountered in emergency settings, necessitating a rapid, affordable, and easily accessible method to assess CAD severity for risk stratification. RDW, a routinely available laboratory parameter, has emerged as a potential marker for predicting the severity of CAD, as well as outcomes in stroke and angiographic assessments.

In this study, the MGS was employed to quantify the severity of coronary lesions observed during angiography.[12] RDW was evaluated as part of the complete blood count (CBC) and included absolute red blood cell

indices. The analysis included 500 patients aged over 18 years with a confirmed diagnosis of ACS-either STEMI or NSTEACS-who underwent CAG.

The mean age of patients was 59.68±10.20 years, aligning closely with previous research, such as Nagula et al (53.6±10.3 years), Wu et al (57.8±10.7 years for low RDW and 60.9±10.7 years for high RDW), and Dabbah et al (60±13 years).[13-15] A male predominance (81.00%) was noted, consistent with earlier reports: Nagula et al (75.8%), Wu et al (76.5%), and Dabbah et al (80.0%).[13-15]

Chest pain was the most frequently reported symptom (96.00%), followed by breathlessness (73.00%), diaphoresis (71.00%), and palpitations (53.00%). Comorbidities such as diabetes mellitus (42.00%), hypertension (47.00%), and acute kidney injury (65.00%) were commonly encountered, in line with prior studies.[13-15] Behavioral risk factors included tobacco chewing (23.00%), smoking (47.00%), and alcohol use (24.00%), which are comparable to findings by Nagula et al (smoking 55.5%), Wu et al (42.7%), and Celik et al (20.0%).[13,16]

Vital and laboratory parameters, including a mean heart rate of 84/min, blood pressure of 110/70 mmHg, hemoglobin of 14.07±1.52 g/dl, total leukocyte count of 9.96±3.78, creatinine of 1.10±0.36 mg/dl and random blood sugar of 151.52 mg/dl, were comparable to those reported in prior cohorts.

The mean RDW-CV in our study was 13.74±0.51%. Patients were stratified based on a cutoff of 13.7%, resulting in two subgroups. The mean MCV was 89.65 fL. Regarding angiographic findings, 29.00% of patients had single-vessel disease, 64.00% had double-vessel disease, and 7.00% had triple-vessel disease. The distribution of single-, double- and triple-vessel disease in our cohort (29%, 64% and 7%) reflects inclusion of only angiographically significant CAD in an ACS population.

Among various CAD subtypes, anterior wall myocardial infarction (AWMI) was most prevalent (41.00%), consistent with Nagula et al.[13] The left ventricular ejection fraction (LVEF) in our cohort was 44.50±14.60%, which was slightly lower than reported by Dabbah et al(46±12%) and Wu et al (61±7%).[14,15] The lower LVEF might be attributable to early ischemic cardiomyopathy, which could improve with time.

The mean MGS in our cohort was 110.10±28.59. Direct comparison with other series is limited by differences in case-mix and scoring conventions. Based on severity, 17.00% of patients had mild disease (Gensini ≤80), while 83.00% had severe disease (>80).

When evaluating RDW-CV in relation to disease severity, patients with RDW-CV ≤13.7% had mild CAD in 29.73% and severe CAD in 70.27%. In contrast, those with RDW-CV >13.7% had severe CAD in 90.48%, indicating a significant correlation ($p<0.05$). This supports RDW as a predictor of CAD burden.

Receiver Operating Characteristic (ROC) analysis showed that RDW-CV had an area under the curve (AUC) of 0.651 (95% CI: 0.549–0.744, p=0.037), with 68.67% sensitivity, 64.71% specificity, positive predictive value (PPV) of 90.48%, negative predictive value (NPV) of 29.73%, and overall diagnostic accuracy of 65.10%. These findings are in line with Cemin et al (AUC = 0.61) and Nagula et al, who found an AUC of 0.79, sensitivity of 58.9%, and specificity of 84.8%.[13,17]

Multiple studies support the association between elevated RDW and adverse outcomes. Uyarel et al noted increased in-hospital mortality with RDW values of 16.1%.[18] Gul et al demonstrated RDW at admission to be an independent predictor of poor outcomes in NSTEACS and unstable angina.[19] Isik et al found higher RDW levels in patients with angiographically confirmed CAD compared to those without (14.4% vs. 12.5%).[20] Tenekecioglu et al found an AUC of 0.649 (95% CI: 0.546–0.753, $p<0.01$), with RDW cutoff >14.05% yielding 73.0% sensitivity and 56.0% specificity, reaffirming the utility of RDW in STEMI risk prediction.[21] Our in-hospital mortality difference (5.41% vs 11.11%) was not significant.

Limitations

This study was limited by its sample size. Also, because this was a cross-sectional study confined to the index hospital admission, the prognostic impact of elevated RDW on long-term outcomes and post-discharge mortality in patients with CAD could not be assessed. Only hemoglobin and red cell indices were measured in this study, whereas parameters such as serum ferritin, serum iron, vitamin B12, and folate were not correlated. Hepcidin and TIBC levels were not measured. The peripheral blood smears of cases with anisocytosis were not evaluated.

CONCLUSION

In this cross-sectional study of 500 patients with acute coronary syndrome, an admission RDW-CV above 13.7% was associated with angiographically severe coronary artery disease on the modified Gensini score. Its discriminative ability was modest (AUC 0.651; sensitivity 68.7%, specificity 64.7%). RDW was not associated with the number of diseased vessels, the myocardial infarction subtype or in-hospital mortality. RDW is an inexpensive, routinely available parameter that may serve as an adjunct to, but not a replacement for, clinical assessment and angiography. Larger, multicentre prospective studies that include iron indices and follow-up outcomes are needed.

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