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Original article

Association of Red Cell Distribution Width with Cardiovascular Complications and Risk Factors in Hypertensive and Cardiac Patients: A Retrospective Study

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Red Cell Distribution Width (RDW), Erythrocyte Size Heterogeneity, Cardiovascular Diseases, In-hospital Mortality, ICU Admission, Libya.

ABSTRACT
This study addresses an overlooked clinical gap in Libya by evaluating baseline Red Cell Distribution Width (RDW)—a standard measure of erythrocyte size heterogeneity—as a zero-cost prognostic tool for predicting cardiovascular mortality and critical care escalation. While RDW traditionally classifies anemias, modern clinical pathophysiology demonstrates that fluctuating RDW levels actively reflect systemic inflammation and tissue hypoxia, both of which impair bone marrow function during cardiac stress. Currently, no published regional literature has established specific RDW thresholds for cardiac risk stratification in Libya, where managing cardiovascular crises is heavily complicated by strict budgetary constraints and chronic shortages of expensive diagnostic reagents. To provide an accessible predictive alternative, this retrospective investigation analyzed archived clinical and laboratory records of 350 adult cardiac patients at the Tajoura National Center for Cardiac Treatment. The baseline cohort (mean age 61.42 years, 55.7% male) exhibited high rates of hypertension (61.4%) and diabetes mellitus (56.0%), with an elevated overall mean RDW of 16.60%. RDW profiles remained statistically similar between isolated cardiovascular disease and concurrent hypertension (16.72% vs. 16.52%, $p = 0.505$) but varied significantly with myocardial infarction status ($p = 0.035$) and peaked in mechanical heart failure (17.33% vs. 15.78%, $p < 0.001$). Controlling for baseline age, diabetes, and smoking status via multivariate regression, elevated RDW independently outperformed these traditional covariates, emerging as a powerful predictor of death, with optimal thresholds fixed at 16.25% for mortality screening and 16.35% for ICU admission. In conclusion, this study provides the first definitive domestic data confirming that baseline RDW functions as an independent biometric predictor of patient mortality and critical care escalation, offering resource-strained hospitals an immediate, zero-cost screening marker to help save lives.

Introduction

Cardiovascular diseases (CVD) and arterial hypertension (HTN) remain major drivers of global morbidity, intensive care admissions, and mortality (1). Early risk stratification, alongside the identification of accessible and cost-effective biomarkers, is essential to alter the clinical course of these conditions, optimize therapeutic strategies, and prevent acute, life-threatening complications such as myocardial infarction (MI) and heart failure (HF) (2).

In routine clinical practice, red blood cell distribution width (RDW) is automated and universally reported as a standard component of the complete blood count (CBC). Historically, the importance of RDW was limited to its use in conjunction with MCV in assessing the level of anisocytosis to differentiate between various types of nutritional anemias (3). However, recent advances in biochemical and clinical studies have contributed to a growing understanding of the clinical significance of RDW levels. Elevated RDW levels result from the activity of systemic cytokines (IL-6 and TNF-alpha) and oxidative stress, which affect the erythropoietin response, disrupt iron metabolism, and increase red blood cell death. Thus, elevated RDW indicates an ongoing inflammatory process at the vascular and metabolic levels (4).

Cardiovascular disease outcomes vary drastically between wealthy and resource-constrained nations; in the latter, a deficit in diagnostic infrastructure often delays early screening, driving up preventable complication rates (5). Systemic hypertension acts as the primary catalyst here, directly instigating coronary artery disease, atherosclerosis, heart failure, and stroke (6), which makes structured clinical follow-up an absolute necessity to salvage patient outcomes (7). Emerging pathophysiological data now connect red blood cell distribution width (RDW) to these hypertensive states, pointing toward chronic vascular inflammation as the culprit altering erythrocyte kinetics (8). In Libya, this clinical challenge is exceptionally severe. National epidemiological data show that hypertension affects nearly 45.9% of men and 39.4% of women, making

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cardiovascular disorders a leading cause of death and permanent disability nationwide (9, 10). Yet, a major clinical contradiction persists.

While international cohorts widely link RDW to overall cardiovascular and hypertensive progression (11, 12), published data remain highly inconsistent—and often contradictory—when evaluating how RDW interacts with specific co-existing metabolic or lifestyle risk factors, namely diabetes mellitus, dyslipidemia, and active smoking. Locally, there is a critical lack of hospital-based studies in Libya; no local research has routinely assessed this index for risk evaluation and prediction of severe deterioration in patient health. Our study aims to fill this gap by providing the first clinical data reference in Libya that directly links the index to ICU bed occupancy rates and clinical mortality.

The current study aimed to address the aforementioned problem by analyzing a multi-indicator dataset of cardiovascular patients in Libya to understand how the red blood cell size differential (RDW) index functions in practice. To achieve this, we not only evaluated the RDW index but also integrated it with other routine indicators, including white blood cell count, platelet count, and cholesterol analysis. This will help us understand whether integrating routine indicators improves risk prediction and clinical decision-making.

Methods

Study Design and Setting

The comparative retrospective investigation was conducted at the National Center for Cardiac Treatment and Surgery, located in Tajoura, Tripoli, Libya, which stands as the premier specialized referral institution providing cardiac care and advanced cardiovascular surgery services nationwide. The data acquisition phase involved a comprehensive evaluation of archived patient medical files and institutional laboratory databases registered over a four years spanning from 2020 to 2024. The study targeted clinical records of individuals admitted directly to the hospital's internal medicine departments, cardiology wards, and intensive care units. Eligible registries were restricted to patients firmly diagnosed with either isolated cardiovascular disease, designated as code one, or concomitant hypertension with cardiovascular disease, designated as code two.

Ethical Considerations

The institutional administration of the National Center for Cardiac Treatment and Surgery officially granted formal administrative clearance to access the archived medical records and databases. To ensure absolute patient privacy and data security, all personal identifying details were completely redacted from patient worksheets. This process was executed prior to entering any clinical metrics into the digital coding protocol, thereby maintaining complete anonymity throughout the research process.

Selection Criteria

Clinical eligibility was governed by specific chronological and pathological conditions. The inclusion framework required that the record belong to an adult patient over eighteen years of age with a verified diagnosis of isolated cardiovascular disease or hypertension co-existing with cardiovascular disease, alongside a complete and intact baseline complete blood count profile. Conversely, the exclusion criteria strictly omitted records of patients with documented malignancies, severe anemia defined as hemoglobin levels less than 8 g/dL, or primary hematologic disorders that could inherently alter erythrocyte size heterogeneity and confound the study outcomes. Patient files with incomplete initial data or missing core clinical parameters were likewise excluded from the final analysis.

Sample Size and Data Acquisition

To ensure sufficient clinical representation and adequate statistical power across the comparative groups, a total baseline sample of 350 validated patient cases was collected and distributed across the designated study cohorts. The extracted variables were categorized into three distinct clinical domains. Demographic parameters included the baseline age, gender, and the exact calendar year of admission. Laboratory and biochemical parameters comprised red cell distribution width, hemoglobin concentration, white blood cell count, total platelet count, and baseline serum total cholesterol levels. Clinical profile information encompassed the documentation of conventional metabolic risk factors, specifically diabetes mellitus, smoking status, and dyslipidemia, alongside major cardiac complications and endpoints, including acute myocardial infarction, mechanical heart failure, intensive care unit stay, and all-cause in-hospital mortality.

Statistical Analysis

All clinical data were compiled and processed utilizing the SPSS version 26.0 software package. Continuous variables, including age, red blood cell size anisotropy index, hemoglobin, platelets, white blood cells, and total cholesterol, were evaluated for distribution and summarized as mean values with their corresponding standard deviations. Qualitative data and categorical clinical frequencies were reported as percentage rates.

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Independent samples t-tests and one-way analysis of variance were implemented to evaluate variations across distinct categories of complications, comparing outcomes such as individuals experiencing specific cardiac events against non-affected patients, as well as survivors against non-survivors. Furthermore, multivariate logistic risk modeling was applied to derive odds ratios and their ninety-five percent confidence intervals, allowing a definitive assessment of whether the red blood cell size anisotropy index acts as a powerful, independent indicator for intensive care unit admissions and mortality after adjusting for the confounding effects of baseline age, smoking status, and diabetes mellitus, with statistical significance established at a p-value of less than 0.05.

Results

Baseline Demographic and Clinical Characteristics

A total of 350 patients were included in the study. The baseline demographic and clinical characteristics of the study population are comprehensively summarized in Table 1. The mean age of the cohort was 61.42 years (with a standard deviation of 14.12), and a wide age distribution ranging from 23 to 102 years. The largest age group was 60 to 69 years (29.4%), followed by 50 to 59 years (24.6%). Males represented the majority of the study population (55.7%).

Regarding clinical comorbidities, hypertension and diabetes mellitus were highly prevalent, affecting 61.4% and 56.0% of the patients, respectively. Patients with concomitant hypertension and cardiovascular disease constituted 60.0% of the entire cohort, while 40.0% presented with cardiovascular disease alone. Additionally, only 14.9% of the participants were active smokers. Laboratory profiles showed a mean baseline Red Cell Distribution Width (RDW) of 16.60%, ranging from a minimum of 10.3% to a maximum of 27.8%. The mean hemoglobin concentration was 12.06 g/dL, mean platelet count was 239.45 × 10³/μL, and the mean white blood cell count was 10.13 × 10³/μL. Total cholesterol measurements were available for a subpopulation of 308 patients, yielding a mean value of 134.88 mg/dL.

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population (N = 350)

Variable / Characteristic	Presentation / Statistics	Minimum	Maximum
Continuous Variables (Mean ± SD)			
Age (years)	61.42 ± 14.12	23	102
RDW (%)	16.60 ± 2.72	10.3	27.8
Hemoglobin (g/dL)	12.06 ± 1.88	8.20	16.60
Platelet Count (×10³/μL)	239.45 ± 91.82	31.0	632.0
White Blood Cell Count (×10³/μL)	10.13 ± 4.89	3.10	35.50
Total Cholesterol (mg/dL) *	134.88 ± 46.70	47.0	326.00
Categorical Variables, n (%)			
Age Group			
23–39 years	22 (6.3%)	-	-
40–49 years	42 (12.0%)	-	-
50–59 years	86 (24.6%)	-	-
60–69 years	103 (29.4%)	-	-
70–79 years	56 (16.0%)	-	-
80 years and older	41 (11.7%)	-	-
Gender			
Male	195 (55.7%)	-	-
Female	155 (44.3%)	-	-
Primary Diagnosis			
Cardiovascular Disease (CVD)	135 (38.6%)	-	-
Concomitant HTN + CVD	215 (61.4%)	-	-
Hypertension (HTN)			
Yes	215 (61.4%)	-	-
No	135 (38.6%)	-	-
Diabetes Mellitus (D.M.)			
Yes	196 (56.0%)	-	-
No	154 (44.0%)	-	-
Smoking Status			
Yes	52 (14.9%)	-	-
No	298 (85.1%)	-	-

**Note: Total Cholesterol data available for 308 patients.*

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Comparison of RDW Across Clinical Subgroups and Outcomes

The distribution of RDW values was evaluated across various clinical subsets and adverse outcomes (Table 2). No statistically significant difference in RDW values was observed between patients diagnosed with cardiovascular disease alone and those presenting with concomitant hypertension and cardiovascular disease (16.72% vs. 16.52%, p = 0.505). However, a statistically significant difference in RDW was detected based on myocardial infarction status (p = 0.035), with non-myocardial infarction patients showing slightly higher values (16.79%) than myocardial infarction patients (16.11%).
Crucially, patients who experienced severe clinical complications or adverse outcomes exhibited profoundly higher RDW metrics. Specifically, patients with heart failure had significantly elevated RDW values compared to those without heart failure (17.33% vs. 15.78%, p < 0.001). Similarly, patients requiring ICU admission demonstrated significantly higher RDW levels than non-ICU patients (17.12% vs. 15.96%, p < 0.001). The highest absolute RDW values were documented among patients who died during the study period, showing a stark and significant elevation compared to survivors (18.33% vs. 16.31%, p < 0.001).

Table 2. Comparison of Red Cell Distribution Width (RDW) According to Clinical Characteristics and Outcomes

Variable	Group	N	Mean RDW (%) ± SD	p-value
Disease Status	CVD	135	16.72 ± 2.95	0.505
	HTN + CVD	215	16.52 ± 2.56	
Myocardial Infarction (MI)	Yes	98	16.11 ± 2.64	0.035
	No	252	16.79 ± 2.73	
Heart Failure (HF)	Yes	186	17.33 ± 2.88	<0.001
	No	164	15.78 ± 2.28	
ICU Admission	Yes	192	17.12 ± 2.94	<0.001
	No	158	15.96 ± 2.28	
Mortality Status	Dead	50	18.33 ± 3.13	<0.001
	Alive	300	16.31 ± 2.54	

Relationship Between RDW and Other Laboratory Markers

An evaluation of the relationships between baseline red cell distribution width (RDW) and other laboratory parameters was performed (Table 3). The analysis revealed a statistically significant negative correlation between RDW and hemoglobin levels, indicating that an increase in red blood cell size heterogeneity is closely associated with a decline in hemoglobin concentration.
Conversely, no statistically significant correlations were observed between RDW and white blood cell counts, platelet counts, or total cholesterol levels. These findings demonstrate that RDW functions as an independent hematological index, operating distinctly from pathways involving systemic leukocytosis, thrombocyte dynamics, or lipid profiles.

Table 3. Correlation Between RDW and Hematological Parameters and Total Cholesterol

Variable Associated with RDW	Pearson Correlation (r)	p-value
Hemoglobin (Hb)	-0.244	<0.001
White Blood Cells (WBCs)	-0.035	0.511
Platelets (PLTs)	-0.051	0.345
Total Cholesterol	-0.103	0.072

Prognostic Value of RDW for Adverse Clinical Endpoints

The prognostic capacity of baseline red cell distribution width regarding major clinical outcomes was evaluated in (Table 4). Initial analysis demonstrated that elevated RDW levels are significantly associated with a higher risk of both intensive care unit (ICU) admission and patient mortality.
To determine the independent prognostic value of this biomarker alongside age, diabetes mellitus, and smoking status, RDW maintained a direct relationship with mortality. Under the same evaluated conditions, no direct relationships were observed between mortality and the other factors of age, diabetes mellitus, or smoking status.

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Table 4. Logistic Regression Models Predicting Patient Mortality and ICU Admission

Outcome/Predictor Model	Coefficient (B)	Standard Error (SE)	Wald Value	Odds Ratio (95% CI)	p-value
Univariate models					
ICU Admission: RDW	0.173	0.045	15.118	1.189 (1.090–1.298)	<0.001
Mortality: RDW	0.247	0.054	20.628	1.280 (1.151–1.424)	<0.001
Multivariable model (mortality)					
RDW	0.267	0.057	22.193	1.306 (1.169–1.460)	<0.001
Age	0.010	0.012	0.806	1.010 (0.988–1.034)	0.369
Diabetes Mellitus	0.535	0.342	2.449	1.708 (0.874–3.338)	0.118
Smoking Status	0.158	0.458	0.119	1.171 (0.477–2.875)	0.730

Discriminative Capacity of RDW for Mortality and ICU Admission

The diagnostic performance of baseline RDW in discriminating clinical outcomes was evaluated using Receiver Operating Characteristic (ROC) curve analysis (Table 5). RDW demonstrated a statistically significant capacity to predict patient mortality, yielding an Area Under the Curve (AUC) of 0.697 (95% confidence interval: 0.617–0.777, $p < 0.001$). The optimal cut-off value for mortality prediction was determined to be 16.25%, which provided a sensitivity of 76.0% and a specificity of 52.7% (Figure 1; A).

For the prediction of ICU admission, the discriminative ability of RDW was lower but remained statistically significant, with an AUC of 0.615 (95% Confidence Interval: 0.557–0.673, $p < 0.001$). The optimal cut-off threshold for ICU triage was identified at 16.35%, offering a sensitivity of 57.6% and a specificity of 62.3% (Figure 1; B).

Table 5. Receiver Operating Characteristic (ROC) Curve Diagnostics of Baseline RDW for Clinical Endpoints

Predicted Outcome	Area Under Curve (AUC)	95% Confidence Interval	p-value	Optimal Cut-off (%)	Sensitivity (%)	Specificity (%)
Mortality	0.697	0.617–0.777	<0.001	16.25%	76.0%	52.7%
ICU Admission	0.615	0.557–0.673	<0.001	16.35%	57.6%	62.3%

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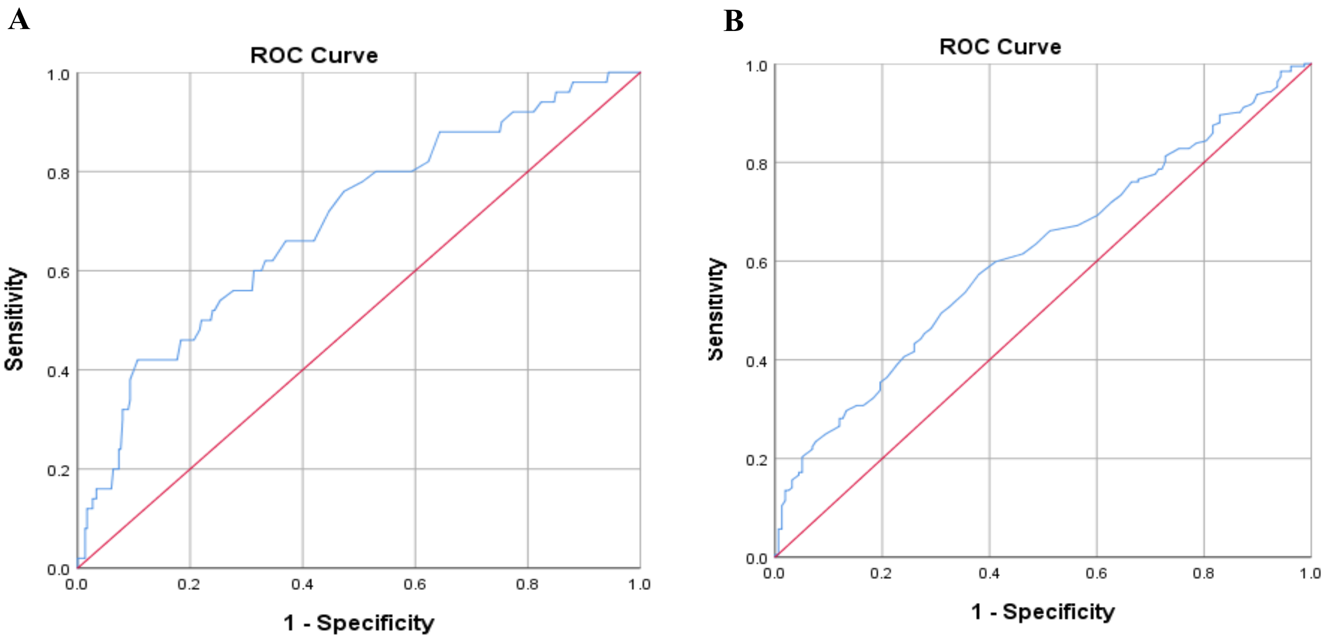

Figure1 . Receiver Operating Characteristic (ROC) curve analysis of baseline Red Cell Distribution Width (RDW). (A) ROC curve for the prediction of patient mortality (Area Under the Curve = 0.697). (B) ROC curve for the prediction of Intensive Care Unit (ICU) admission (Area Under the Curve = 0.615).

Discussion

This retrospective study was specifically designed to investigate the association between RDW and cardiovascular complications, as well as traditional risk factors, in hypertensive and cardiac patients. Furthermore, this work aimed to evaluate the potential role of RDW as a simple, universally accessible biomarker for cardiovascular risk stratification. The baseline characteristics of the investigated cohort (N = 350) revealed a mean age of 61.42 years, with a notable male predominance (55.7%), reflecting the established global epidemiological patterns where advanced age and male gender serve as non-modifiable risk factors for accelerated vascular aging, endothelial injury, and cumulative arterial stiffness (13).
The clinical profile of the study population underscored a heavy burden of comorbidities, characterized by remarkably high rates of hypertension (61.4%) and diabetes mellitus (56.0%). In this high-risk vascular environment, the baseline mean RDW was found to be notably elevated at 16.60% (± 2.72%), significantly exceeding standard physiological reference thresholds. Structurally, an elevated RDW reflects pronounced anisocytosis, which represents an underlying state of systemic disruption rather than a mere hematological anomaly (3). In patients with established cardiovascular disease, this expansion in red blood cell size heterogeneity serves as a biological mirror reflecting ongoing systemic inflammation, heightened oxidative stress, neurohormonal overactivation, and blunted bone marrow responsiveness (14).

RDW Dynamics Across Cardiovascular Complications

A compelling finding emerged when analyzing structural disease phenotypes: no statistically significant difference in RDW metrics was observed between patients diagnosed with cardiovascular disease alone and those presenting with concomitant hypertension and cardiovascular disease (16.72% vs. 16.52%, p = 0.505). This similarity indicates that once advanced cardiovascular disease is established, the co-existence of hypertension does not cause any further or additional damage to the red blood cell production process (15). Both groups of patients likely share a similar level of chronic tissue inflammation and vascular stress, which affects the stability of red blood cell membranes and bone marrow response to the same extent (3, 16).
In contrast, RDW values exhibited significant variations based on myocardial infarction status (p = 0.035). During an acute ischemic coronary event, the sudden onset of profound myocardial hypoxia triggers a rapid systemic surge of pro-inflammatory cytokines, such as interleukin-6 and tumor necrosis factor-alpha (17). These inflammatory mediators directly impair the bone marrow's response to erythropoietin, preventing proper erythrocyte maturation and leading to the premature release of larger, immature cells into the circulation, which shifts the RDW threshold (18).
The most clinically profound divergence was documented in relation to mechanical heart failure, where patients suffering from heart failure exhibited markedly higher RDW values compared to non-heart failure individuals. From a pathophysiological standpoint, heart failure initiates a state of chronic systemic hypoperfusion combined with continuous venous congestion. This mechanical insufficiency triggers a

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sustained compensatory activation of both the renin-angiotensin-aldosterone system and the sympathetic nervous system (19).
In the bone marrow microenvironment, this chronic ischemic stress reduces tissue oxygenation and accelerates oxidative damage to emerging red blood cell progenitors. Furthermore, the ongoing cascade of systemic inflammatory reactions leads to increased hepcidin expression, which restricts iron availability and induces a state of functional iron deficiency, impairing efficient hemoglobin synthesis and altering the structural development of red blood cells (20). Therefore, the marked increase in RDW in heart failure patients is a direct pathophysiological reflection of severe mechanical and metabolic stress on the hematopoietic system, making this indicator a reliable indicator of advanced cardiac dysfunction (21).

Biological Isolation of RDW and Risk Triage

The biological uniqueness of RDW was further validated through relationship assessments with other laboratory markers. A statistically significant, moderate negative correlation was identified between RDW and hemoglobin levels ($r = -0.244$, $p < 0.001$). This inverse relationship highlights that a progressive shift toward variation in cell size is intrinsically tied to a decline in oxygen-carrying capacity, reflecting subclinical anemia of chronic disease. Crucially, RDW displayed no significant relationship with white blood cell counts ($p = 0.511$), platelet counts ($p = 0.345$), or total cholesterol levels ($p = 0.072$). This clinical independence is highly meaningful and fully comports with the landmark reference study conducted by Danese et al. (8). Their rigorous laboratory evaluation established that the only consistent, direct inverse correlation for RDW is with hemoglobin concentration as a consequence of impaired erythropoiesis, while remaining biologically isolated from systemic leukocytosis, thrombocyte dynamics, or baseline lipid profiles. Consequently, these findings confirm that the prognostic signals embedded within RDW constitute a distinct and isolated biological pathway, completely unconfounded by general immune activation, platelet-driven thrombosis, or lipid status (3, 8, 22).

Independent Prognostic Strength of RDW

The clinical findings demonstrated that a high baseline RDW is directly associated with a greater likelihood of both intensive care unit admission and patient mortality. Even when considering other major cardiovascular risk factors—such as advanced age, diabetes mellitus, and smoking status—RDW maintained its powerful, direct connection to patient mortality. Strikingly, under these same conditions, traditional factors like age, diabetes, and smoking status did not show a direct or independent link to mortality outcomes. This indicates that an elevated RDW serves as a centralized indicator of true systemic damage and tissue hypoxia, carrying a stronger predictive value for patient survival than conventional risk parameters (23, 24).
These outcomes are in perfect agreement with a comprehensive clinical study conducted by Topf et al. (25), which evaluated real-world cardiovascular cohorts and confirmed that RDW remains a superior, independent indicator of death even when traditional metabolic and demographic risks are present. Similarly, our findings strongly align with a clinical trial published by Oba et al. (2025), which concluded that while factors like diabetes and smoking represent common baseline vulnerabilities, an elevated RDW acts as a common final pathway that reflects the true, real-time physiological frailty and survival risk of the cardiac patient (25) (26).

Clinical Thresholds and Strategic Applications

To establish the practical utility of this biomarker for bed-side risk stratification, the receiver operating characteristic curve analysis was utilized. RDW demonstrated a significant discriminative capacity for patient mortality, yielding an Area Under the Curve of 0.697, with a highly significant p-value of less than 0.001. The calculated optimal cut-off threshold was fixed at 16.25%, which achieved a robust sensitivity of 76.0% paired with a moderate specificity of 52.7%. In clinical screening applications, prioritizing high sensitivity is essential. An RDW tracking above 16.25% can effectively serve as an early clinical red flag, allowing healthcare providers to identify high-risk individuals who require intensive monitoring or aggressive therapeutic optimization (27) .
Similarly, for the prediction of ICU admission, the discriminative performance remained highly significant, demonstrating an Area Under the Curve of 0.615, with a p-value of less than 0.001 at an optimal cut-off threshold of 16.35%. Although the overall diagnostic accuracy for ICU triage was slightly lower than that observed for mortality, it remains highly meaningful. Because RDW is automatically calculated during a routine CBC without requiring additional financial costs or specialized equipment, it represents an ideal, cost-free tool for adjunctive risk stratification, particularly in resource-limited healthcare settings (28).

Limitations

Several limitations must be acknowledged in this study. First, the retrospective, single-center design restricts the ability to establish direct causal relationships and may limit the generalizability of the findings

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to broader geographic cohorts. Second, due to reliance on historical electronic registries, data regarding primary biochemical determinants of RDW, such as serum iron, ferritin, total iron-binding capacity, Vitamin B12, folate, and highly sensitive C-reactive protein, were unavailable. Third, RDW was evaluated only at a single static time point upon admission, preventing the analysis of RDW kinetics or longitudinal fluctuations during the disease course. Finally, while RDW was established as a highly significant independent predictor, its overall discriminative accuracy via analysis was moderate, indicating that it should be utilized as a supplement to, rather than a replacement for, established clinical risk scores.

Conclusion and Clinical Implications

In conclusion, this study successfully fulfills its primary aim by confirming that an elevated baseline Red Cell Distribution Width is independently and heavily linked to heart failure complications, ICU admission, and all-cause mortality in cardiovascular patients. From a practical standpoint, RDW represents an exceptionally attractive clinical biomarker. Because it is universally available via standard hematological profiles at zero extra cost, integrating specific RDW thresholds, particularly values exceeding 16.25%, into existing cardiovascular risk prediction models could substantially enhance prognostic accuracy, facilitating personalized, early, and aggressive therapeutic interventions for vulnerable patients.

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