



Research Article

Comparison of Red Cell Distribution Width and Serum Lactate as Predictors of Outcomes in Patients with Sepsis

Dr. Gangum Venkat Reddy ¹, Dr. Suresh Jana ²

¹ Associate Professor, Department of General Medicine, Prathima Institute of Medical Sciences, Naganoor, Karimnagar, Telangana.

² Associate Professor, Department of General Medicine, Prathima Institute of Medical Sciences, Naganoor, Karimnagar, Telangana.

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Corresponding Author:

Dr Suresh Jana

Associate Professor, Department of General Medicine, Prathima Institute of Medical Sciences, Naganoor, Karimnagar, Telangana.

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ABSTRACT

Background: Early risk stratification of sepsis is crucial for improving its management and enhancing outcomes. Serum lactate is an established prognostic biomarker; a growing body of evidence suggests that red cell distribution width (RDW), a routinely measured hematological value, can also be used as a predictor of mortality. The current study aimed to compare RDW with lactate as a predictor of outcome in patients with sepsis.

Methods: This prospective observational study was carried out in 40 adult sepsis cases admitted to the ICU. Baseline demographic, clinical, and laboratory values were collected. Measurement of RDW and lactate was performed at admission (T0), and clearance of lactate was done at 24 hours. SOFA scores were determined. The appropriate statistical tests were used to compare the outcomes of the survivors and non-survivors. Logistic regression and receiver operating characteristic (ROC) analysis were conducted to measure the predictive accuracy.

Results: The mean age of the cohort was 62.5 ± 1.48 years, with higher age, comorbidity burden, and SOFA scores in non-survivors ($p < 0.05$). Admission RDW and lactate were significantly elevated in non-survivors (17.5% vs. 14.9%, $p < 0.001$; 5.1 mmol/L vs. 2.3 mmol/L, $p < 0.001$). ROC analysis showed strong discriminatory ability for both RDW (AUC 0.86) and lactate (AUC 0.89), with the highest accuracy when combined (AUC 0.93). Logistic regression confirmed RDW $>16.5\%$ and lactate >3.8 mmol/L as independent predictors of mortality. Adding these biomarkers to SOFA scores improved risk reclassification (NRI 0.71, $p = 0.003$).

Conclusion: RDW and lactate are reliable and independent predictors of mortality in sepsis. Their combined assessment enhances prognostic accuracy beyond conventional clinical scoring systems. Given their simplicity and accessibility, RDW and lactate should be incorporated into routine sepsis risk stratification to facilitate early identification of high-risk patients and optimize management.

Keywords: Red Cell Distribution Width, Serum Lactate, Sepsis, Mortality.

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INTRODUCTION

Sepsis is a life-threatening dysfunction of organs caused by an inappropriate host response to an infection and remains one of the leading causes of morbidity and mortality across the global health landscape [1]. Despite antimicrobial therapy and critical care developments, sepsis causes nearly 11 million deaths each year, which is almost 20 percent of all worldwide deaths [2]. Early identification and prediction are essential to inform aggressive management, resource optimization, and outcomes. The use of biomarkers in the evaluation of sepsis has a long history, although clinical challenges arise in their attempt to find cost-effective and widely available predictors. Red Cell Distribution Width (RDW) is an integral part of the complete blood count (CBC), estimated routinely as a measure of variability in red blood cell size (anisocytosis). RDW

has recently become the focus of interest as a measure of systemic inflammation and worse outcomes in critically ill patients, traditionally employed in the differential diagnosis of anemia [3]. Some studies have shown that high RDW is linked with higher mortality in diseases like heart failure, acute coronary syndrome, and sepsis [4, 5]. The mechanism underlying this could be inflammation-induced bone marrow dysfunction, oxidative stress, and disrupted erythropoiesis resulting in anisocytosis [6]. As CBC is inexpensive and accessible worldwide, RDW can be a feasible risk stratification tool in sepsis. Serum lactate, on the other hand, is an established marker of tissue hypoperfusion and cell metabolic stress. Higher levels of lactate in sepsis indicate anaerobic metabolism (impaired oxygen delivery or oxygen consumption) and sustained high levels are strongly associated with increased mortality [7]. International guidelines recommend lactate-guided resuscitation strategies as they form a part of sepsis management [8]. Lactate measurements, however, are not a consistently feasible procedure in resource-constrained environments, and their interpretation can be confounded by factors like liver dysfunction, β -agonist therapy, and seizures [9]. Although the RDW and lactate have both been studied as independent prognostic variables, the literature has not definitively compared the predictive ability of the two variables in sepsis. Integrating both hematological and biochemical parameters could allow a better evaluation of the severity and the outcomes of the disease. According to some research, RDW may be an additional or a substitute measure of lactate, especially in resource-constrained environments where determination of lactate might not be easily available [10, 11]. Additionally, the benefit of RDW is that it is a component of regular investigations, making it free of both extra expense and infrastructure. Due to a high mortality rate of sepsis and the necessity of finding reliable and cost-effective prognostic measures, the comparison of the usefulness of RDW and lactate has considerable clinical importance. Their predictive accuracy might help clinicians to correctly risk-stratify early, initiate timely interventions, and enhance patient outcomes. The purpose of this study, thus, is to compare Red Cell Distribution Width and serum lactate as outcome predictors in patients with sepsis.

Materials and Methods

This prospective observational study was conducted in the Department of General Medicine and Medical ICU, Prathima Institute of Medical Sciences, Naganoor, Karimnagar, Telangana. Institutional Ethical approval was obtained for the study. Written consent was obtained from all the participants/ or their legally authorized representatives for the study. The study adhered to the Declaration of Helsinki and local regulations.

Inclusion criteria

1. Age ≥ 18 years.
2. Sepsis is defined by Sepsis-3: suspected/confirmed infection plus SOFA score increase ≥ 2 from baseline.
3. Enrollment within 6 hours of sepsis recognition.

Exclusion criteria

1. Proven hematologic malignancy or recent chemotherapy (<3 months).
2. Blood transfusion within 30 days, active major bleeding, or erythropoiesis-stimulating agents within 30 days.
3. Advanced chronic liver disease (Child–Pugh C) or end-stage renal disease on dialysis.
4. Pregnancy.
5. Refusal of consent.

Prospective observational cohort conducted in the adult emergency department and medical ICU of a tertiary-care teaching hospital over 12 months. A total of $n=40$ cases were included in the study based on the inclusion and exclusion criteria. The objective was to compare red cell distribution width (RDW) and serum lactate as predictors of adverse outcomes in sepsis.

At enrollment (T0), demographics, comorbidities (Charlson Comorbidity Index), infection source, vitals, and initial SOFA/qSOFA were recorded. Routine labs included CBC with RDW (RDW-CV, %) and serum lactate (mmol/L). Repeat lactate at 6 hours (T6) was obtained when clinically ordered; lactate clearance (%) = $(\text{lactate}_{T0} - \text{lactate}_{T6}) / \text{lactate}_{T0} \times 100$. RDW was taken from the first CBC within 2 hours of T0; if a historical CBC existed within 3–12 months, baseline RDW was abstracted for sensitivity analyses (Δ RDW).

Laboratory analysis: CBCs were analyzed with an automated hematology analyzer (three-part analyzer) ABX MIRCOS 60. RDW-CV (%) was used for primary analysis. Lactate was measured on a central laboratory blood gas/chemistry analyzer. All assays followed manufacturer calibration and internal quality control. Hemolysed samples were discarded per laboratory policy.

Primary outcome: in-hospital mortality (all-cause). **Secondary outcomes:** ICU admission within 24 h, vasopressor requirement within 24 h, invasive mechanical ventilation within 24 h, length of ICU stay, and composite adverse outcome (mortality and/or vasopressor use and/or ventilation).

Statistical analysis: All the available data were refined, uploaded to an MS Excel spreadsheet, and analyzed by SPSS version 26 in Windows format. Continuous variables were measured as Mean \pm SD or median (IQR), counts (%). Student's

t-test was used to compare the means of two groups, and the square test for variables between two groups. Receiver operating characteristic (ROC) curves with area under the curve (AUC) and 95% CIs for RDW and lactate. DeLong's test compared AUCs. Univariable logistic regression for each biomarker; multivariable models adjusted for age, sex, SOFA, mean arterial pressure, creatinine, and infection source. RDW and lactate were evaluated independently and combined

Results

The demographic profile of the cohort is given in Table 1. A critical analysis of the table shows that the mean age of the overall cohort was 62.5 years. The mean age of non-survivors was significantly older (70.8 vs. 58.9 years, $p = 0.02$). Male preponderance was seen in 55% of all cases without any survival-based differences. The existing comorbidities were found to be more severe in non-survivors. The Charlson Comorbidity Index (median 5 vs. 2, $p = 0.01$). The major source of infection in the cases was pulmonary (45%), followed by urinary infection (27.5%) and abdominal infection (20%). No significant variations between the groups. SOFA scores were higher in non-survivors (9 vs. 6, $p < 0.001$). The mean arterial pressure was lower (62 vs. 71 mmHg, $p = 0.03$), and serum creatinine was elevated (2.3 vs. 1.4 mg/dL, $p = 0.02$). These findings suggest that older age, higher comorbidity burden, and worse physiological derangement at admission were strongly associated with mortality.

Table 1: Baseline Characteristics of the Study Cohort (N=40)

Characteristic	Overall (N=40)	Survivors (n=28)	Non-Survivors (n=12)	P value
Age, years, mean (\pm SD)	62.5 (\pm 1.48)	58.9 (\pm 14.1)	70.8 (\pm 12.5)	0.02*
Male sex, (%)	22 (55.0)	14 (50.0)	8 (66.7)	0.49
Charlson Comorbidity Index, median [IQR]	3 [1 - 5]	2 [1 - 4]	5 [3 - 6]	0.01*
Infection, n (%)				
Pulmonary	18 (45.0)	12 (42.9)	6 (50.0)	0.72
Urinary	11 (27.5)	9(32.1)	2 (16.7)	
Abdominal	8 (20.0)	5(17.9)	3 (25.0)	
Other	3 (7.5)	2 (7.1)	1 (8.3)	
SOFA Score at T0, Median [IQR]	7 [5- 9]	6 [4- 8]	9 [8 - 11]	<0.001*
qSOFA Score \geq 2 n (%)	24 (60.0)	14 (50.0)	0.07	
Mean Arterial Pressure (mmHg), mean (\pm SD)	68 (\pm 12)	71 (\pm 11)	62 (\pm 10)	0.03*
Serum Creatinine (mg/dL), median [IQR]	1.6 [10 - 2.5]	1.4 [0.9 - 2.1]	2.3 [1.6- 3.8]	0.02*
Hemoglobin (g/dL), mean (\pm SD)	108 (\pm 2.2)	11.1 (\pm 2.0)	10.1 (\pm 2.5)	0.18
IQR: Interquartile Range SOFA: Sequential Organ Failure Assessment qSOFA P-values from t-test Mann-Whitney U test *Significant				

Table 2 shows the association of biomarker levels with clinical outcomes by survival status. The comparison between red cell distribution width (RDW), lactate levels, and clinical outcomes in survivors and non-survivors. A significant difference was found in median RDW between non-survivors (17.5% vs. 14.9%, $p < 0.001$), and 100% of non-survivors had RDW that exceeded 14.5%. Similarly, the lactate on admission was significantly high in non-survivors (5.1 mmol/L vs. 2.3 mmol/L, $p < 0.001$). High level of lactate (>4 mmol/L) was also closely associated with death (75% vs. 17.9%, $p < 0.001$). The lactate clearance $\geq 10\%$ was found in 72% of survivors and in 20% of non-survivors ($p = 0.006$). Secondary outcomes were the severity of the disease: vasopressor usage (75% vs. 32% and $p = 0.02$) and composite adverse events (91.7% vs. 50% and $p = 0.02$) were more frequent in non-survivors. These findings highlight the fact that elevated RDW and lactate consistently remain potent predictors of the adverse outcome.

Table 2: Comparison of Biomarker Levels and Clinical Outcomes by Survival Status				
Parameter	Overall (N=40)	Survivors (n =28)	Non-Survivors (n=12)	P value
RDW at T0 (%), median [IQR]	15.4 [14.2- 17.1]	149 [13.9- 16.0]	17.5 [16.2 - 190]	<0.001*
RDW >14.5%, n (%)	28 (70.0)	16 (57.1)	12 (100.0)	0.007*
Lactate at T0 (mmol/L), median [IQR]	2.8 [1.8 -45]	2.3 [1.6 - 3.4]	5.1 [3.8- 7.9]	<0.001*
Lactate at T0 > 2.0 mmol/L, n (%)	30 (75.0)	18 (64.3)	12 (100.0)	0.02*
Lactate at T0 > 4.0 mmol/L, n (%)	14 (35.0)	5 (17.9)	9 (75.0)	<0.001*
Lactate Clearance ≥10% (n=35), n (%)	20/35 (57.1)	18/25 (72.0)	2/10 (20.0)	0.006*
Secondary Outcomes, n (%)				
ICU Admission within 24h	32 (80.0)	21 (75.0)	11 (91.7)	0.39
Vasopressor Use within 24h	18 (45.0)	9 (32.1)	9 75.0)	0.02*
Mechanical Ventilation within 24h	15(37.5)	8(28.6)	7 (58.3)	0.09
# Composite Adverse Outcome	25 (62.5)	14 (50.0)	11 (91.7)	0.02*
# Composite outcome: Mortality and/or vasopressor use and/or mechanical ventilation *Significant				

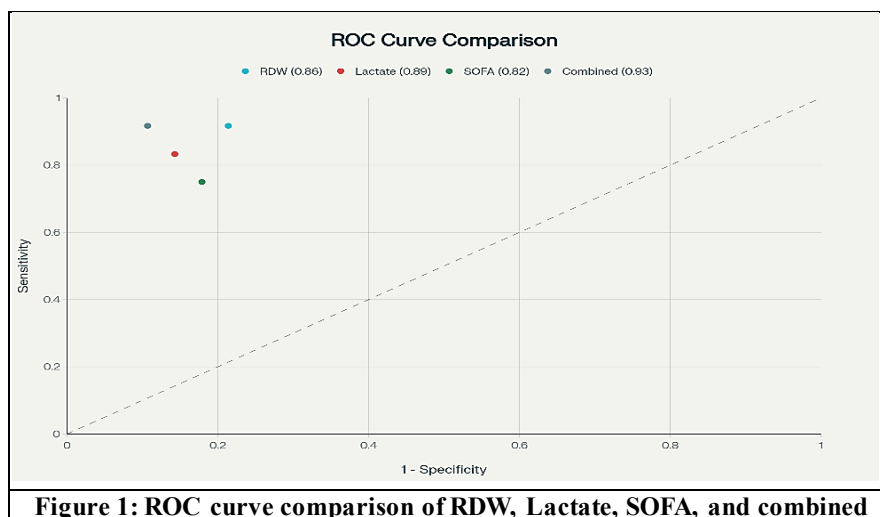


Figure 1: ROC curve comparison of RDW, Lactate, SOFA, and combined

Estimation of the discriminatory power of RDW and lactate in predicting mortality is given in Table 3. RDW showed good discrimination with a cut-off point of >16.5% with an AUC of 0.86 and a sensitivity of 91.7% and specificity of 78.6%. Lactate also did the same with an AUC of 0.89 with an optimal cut-off of >3.8 mmol/L (83.3% sensitivity and 85.7% specificity). The SOFA score by itself was less accurate in prediction (AUC 0.82). Notably, the combination of RDW and lactate delivered the highest performance (AUC 0.93), and a good trade-off of sensitivity (91.7%) and specificity (89.3%) (Figure 1). It implies that early risk stratification with RDW and lactate is better when the two are used together rather than alone in septic patients.

Table 3: Discriminatory Power of RDW and Lactate for Predicting In-Hospital Mortality					
Biomarker	AUC (95% CI)	Optimal Cut-off (Youden)	Sensitivity (%)	Specificity (%)	p-value
RDW at T0	0.86 (0.74 - 098)	>16.5%	91.7	78.6	0.42
Lactate at T0	0.89 (0.78- 099)	>3.8 mmol/L	83.3	85.7	Reference
SOFA Score	0.82 (0.68 - 096)	> 8 mmol/L	75.0	82.1	0.28
Combined (RDW + Lactate)	0.93 (0.85 - 1.00)	-	91.7	89.3	0.18
P value (DeLong's Test Vs Lactate)					

Table 4 presents the univariable and multivariable regression analysis for predictors of mortality. The table shows that univariable analysis for RDW per 1% increase had an Odds ratio of 2.15 with a p value of 0.001; the lactate levels per 1 mmol/L increase had an odds ratio of 1.65 and a p value of 0.001. These results were evaluated as strong predictors. Dichotomized values of RDW > 16.5% and lactate >3.8 mmol/L were associated with high odds of mortality (OR 38.75 and 18.0, respectively). Multivariable analysis confirmed that (aOR 1.89, $p = 0.02$) and lactate (aOR 1.48, $p = 0.03$) were independent predictors despite adjustment for age, SOFA, MAP, and infection score. SOFA scores remained significant with an adjusted odds ratio of 1.42 and p values of 0.04. However, age lost significance after adjustment, suggesting biomarker-driven risk estimation was superior.

Table 4: Univariable and Multivariable Logistic Regression for Predictors of In Hospital Mortality				
Predictor	Univariable Analysis		Multivariable Analysis'	
	OR (95% CI)	P-value	aOR (95% CI)	p-value
RDW (per 1% increase)	2.15 (1.38- 3.58)	0.001	1.89 (1.12- .442)	0.02
RDW >16.5%	38.75 (428 - 78.24)	0.002	25.10 (2.45- 512.6)	0.01
Lactate (per 1 mmol/L increase)	1.65 (1.22 - 2.38)	0.001	1.48 (1.08- 2.18)	0.03
Lactate > 3.8 mmol/L	18.0 (3.15- 127.5)	0.001	12.55 (1.89 - 98.7)	0.01
SOFA Score (per 1 point)	1.58 (1.18 - 2.25)	0.003	1.42 (1.03 - 2.08)	0.04
Age (per 5-year increase)	1.45 (1.08- 2.05)	0.02	1.21 (0.87 - 1.78)	0.25
<i>OR: Odds Ratio; aOR: Adjusted Odds Ratio; CI: Confidence Interval. Multivariable model adjusted for age, sex, SOFA score, mean Arterial Pressure, and infection source, RDW, and lactate were modelled separately to avoid collinearity</i>				

Table 5 shows that the incremental prognostic value for adding biomarkers to the base model (SOFA + age) was showing good predictive accuracy (AUC 0.85). Addition of lactate improved the discriminatory power (AUC 0.91) with significant reclassification and discrimination improvements (NRI 0.55, $p = 0.01$; IDI 0.08, and $p = 0.03$). RDW also enhanced the model (AUC 0.90, NRI 0.48, and $p = 0.02$). The greatest improvement was seen when both RDW and lactate were added together, achieving an AUC of 0.95, NRI 0.71 ($p = 0.003$), and IDI 0.12 ($p = 0.002$). These results confirm that combining RDW and lactate provides incremental prognostic power beyond conventional clinical parameters.

Table 5: Incremental Prognostic Value of Adding Biomarkers to a Clinical Model			
Model	AIK (95% CI)	NRI (95% CI), p-value	DI (95% CI), p-value
Base Clinical Model (SOFA + Age)	0.85 (0.72 – 0.97)	(Reference)	(Reference)
Base + Lactate	0.91 (0.81 - 1.00)	0.55 (0.12 -0.98), $p = 0.01$	0.08 (0.01 – 0.15), $p = 0.03$
Base + RDW	0.90 (0.80 – 0.99)	0.48 (0.08 - 0.88), $p = 0.02$	0.07 (0.01 – 0.13), $p = 0.04$
Base + Lactate + RDW	0.95 (0.88- 1.0)	0.71 (0.25 - 1.17), $p = 0.003$	0.12 (0.04- 0.20), $p = 0.002$
<i>NRI: Net Reclassification Improvement; IDI: Integrated Discrimination Improvement. The base clinical model includes the SOFA score and age. The NRI and IDI values indicate significant improvement in risk prediction when biomarkers are added</i>			

DISCUSSION

The current study was done in a tertiary care hospital with the inclusion of 40 cases of sepsis. We compared the prognostic values of serum lactate and red cell distribution width (RDW) for in-hospital mortality risk with established clinical parameters. The findings of this study showed that both hyperlactatemia and RDW were independently associated with the prediction of mortality, and the combination of both provided a strong discriminatory power. Our results underscore the utility of integrating these simple, readily available biomarkers with established scoring systems to increase early risk stratification in sepsis cases. In line with the previous studies in this field, the non-survivors were substantially older based on the mean age, and their score on the Charlson Comorbidity Index (CCI) was higher, which confirms the contribution of age and the burden of chronic diseases to the results of sepsis [12, 13]. Increased baseline SOFA scores and hemodynamic and renal changes (reduced MAP, increased creatinine) in non-survivors confirm once again that the severity of organ dysfunction is the key predictor of mortality [14, 15]. Notably, our research shows that RDW is a strong prognostic factor

among patients in sepsis. Baseline RDW was significantly higher in non-survivors, and all were above the threshold of 14.5% with RDW >16.5% confirmed as an independent mortality predictor in multivariate analysis. Such results can be attributed to recent reports demonstrating that anisocytosis is indicative of underlying systemic inflammation, oxidative stress, and bone marrow dysfunction, all processes that are closely associated with the pathophysiology of sepsis [16, 17]. Additionally, RDW is an inexpensive, regularly available alongside the complete blood counts, and does not involve any extra sampling and/or expenditure, which makes it very feasible in resource-limited health care settings [18]. In addition, lactate was also a strong prognostic indicator. High admission lactate levels (T0) and inadequate lactate clearance were closely linked with mortality, consistent with existing literature that hyperlactatemia is a reflection of a failure to deliver oxygen to tissues and microcirculatory dysfunction in sepsis [19]. Surviving Sepsis Campaign objectives focus on lactate measurements as a diagnosis and resuscitation goal [20]. Our data support this suggestion and indicate that the inability to eliminate lactate was highly predictive of adverse outcomes. Evaluation of discriminatory performance showed a close relationship with lactate (AUC 0.89), marginally over RDW (AUC 0.86), with a combination of the two having the highest predictive ability (AUC 0.93) (Figure 1). This indicates that RDW and lactate measure complementary variables of the hematologic stress of sepsis, in contrast to metabolic disruptions, and that their combination could sharpen mortality prediction. Our findings are supported by recent studies demonstrating the incremental prognostic utility of combining hematologic with metabolic or clinical scores [21, 22]. Biomarkers have a prognostic role, which is further supported by secondary outcomes that showed that non-survivors required increased vasopressor administration and composite adverse events, indicative of moving to refractory shock and multiorgan failure. Such associations propose that elevations of RDW and lactate could assist clinicians in predicting increasing demands of care. Our study has limitations. The sample size was small, which may limit its generalization and subgroup analysis (e.g., infection source-specific results). We also measured only RDW and lactate at admission; serial measurements would offer more information on their predictive variation throughout admission. Lastly, it was a single-center study and needs to be validated in other care centers. Overall, despite these limitations, our results confirm the growing evidence that RDW can act as a prognostic marker in sepsis, and it has synergistic values when combining it with the measurements of serum lactate levels.

CONCLUSION

Within the limitations of the current study, we conclude that both RDW and lactate levels are independent predictors of mortality in patients with sepsis. Their combined estimations have been shown to provide strong prognostic accuracy. Increased RDW levels show systemic inflammation and hematologic stress, and lactate indicates impaired tissue perfusion and metabolic dysfunction. Because these parameters are simple and low-cost, they can serve as valuable adjuncts to established clinical scoring systems such as SOFA for risk stratification. Incorporation of these parameters must be done for the timely identification of high-risk patients and their management.

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