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RESEARCH ARTICLE

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Use of renin versus lactic acid as tissue perfusion biomarkers for mortality prediction in hypotensive critically Ill patients

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ABSTRACT

Background: Exploring a biomarker with enhanced sensitivity and specificity for tissue perfusion may facilitate the timely identification of circulatory collapse, and enable more precise resuscitation efforts.

Objective: The objective of this study was to ascertain the correlation between whole blood lactate versus plasma renin concentration being a biomarker of tissue perfusion and predictor of mortality among hypotensive critically ill patients.

Methods: This prospective, observational cohort study enrolled 84 hypotensive critically ill patients. Plasma renin concentration and blood lactate were measured at enrollment, 24, 48, and 72 hours. The primary outcome is the correlation between the recorded renin, lactate concentrations and mortality rate during hospitalization.

Results: The mean plasma renin concentration at enrollment was 61.95 pg/ml in survivors, and 104.45 pg/ml in non-survivors ($p = <0.001$). The non-survivors exhibited a significant boost in plasma renin concentration after 48 and 72 hours, opposed to the survivors (112 vs 40.89, and 106.64 vs 28.85 pg/ml) respectively. There was a significant positive correlation between plasma renin, blood lactate concentrations and patient mortality ($r = 0.389$ & 0.601) respectively.

Conclusion: Plasma renin and whole blood lactate had positive correlation to mortality, yet plasma renin revealed superior diagnostic accuracy over blood lactate for mortality prediction in hypotensive critically-ill patients.

Trial registration: The protocol of this study can be obtained on ClinicalTrials.gov with the id NCT05810415.

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1. Introduction

Maintaining an optimal circulatory homeostasis is regulated by renin-angiotensin-aldosterone (RAAS), being capable offluid balance, and systemic vascular resistance coordination. The principal enzyme responsible for propelling this system along is renin. Hypoxic metabolism, sympathetic activity, and reduced tissue perfusion all trigger its secretion. Curiously, acute circulatory collapse has not provided a thorough characterization of renin [1].

Vasodilatory shock, in which peripheral vasodilation occurs alongside preserved or augmented cardiac output, is another prevalent type of shock. Vasodilatory shock is often caused by inflammatory syndromes or situations like sepsis or significant surgery. Although the exact mechanisms that cause vasodilatory shock remain unresolved, it is known that RAAS is one of several hormonal systems that coordinate patient response to shock [2]. As a physiological probable life-sustaining response, overexpression of RAAS occurs in the setting of vasodilatory shock. Additional physiologic consequences to infection include endothelial structural alterations, which may be enhanced by angiotensin II overexpression [3].

Moreover, disruption of angiotensin-converting enzyme results in an excessive amount of angiotensin I, which is subsequently broken down into angiotensin. These secondary products induce the widening of blood vessels and reduce the pressure of blood flow to the kidneys, so stimulating the juxtaglomerular cells to secrete renin. Renin enzymatically breaks down angiotensinogen, a protein produced by the liver, into angiotensin I. Due to reduced activity of ACE (angiotensin-converting enzyme), angiotensin I is further metabolized into other substances that promote blood vessel dilation. This causes persistent low blood pressure that is difficult to treat, and in turn, triggers an increase in renin levels [4].

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Undoubtedly, blood lactate and central venous oxygen saturation (ScvO2) serve as indicators of tissue-perfusion, although they possess several constraints. Lactate can rise during aerobic metabolism [5]. High levels of lactate indicate insufficient blood flow to the tissues, however this is only seen at a later stage [6]. Furthermore, the analysis of ScvO2 is intricate due to its U-shaped pattern in relation to the supply of oxygen to cells. Utilizing ScvO2 as a guidance for resuscitation did not result in improved outcomes for patients with sepsis [7].

Discovering a more sensitive biomarker for tissue-perfusion could enable the early detection of circulatory collapse and more precise resuscitation. A recent study conducted at a single site showed that plasma renin exhibited superior predictive value for intensive care unit (ICU) mortality compared to lactate [1]. Following that, a study conducted at a single center discovered no correlation between plasma renin levels and mortality. However, it did observe that plasma renin levels were significantly elevated in cases of increasing acute kidney injury (AKI) [8].

The objective of this prospective study was to ascertain the correlation between whole blood lactate versus plasma renin concentration, being biomarkers of tissue perfusion, for prediction of mortality among hypotensive critically ill patients.

2. Patient and methods

This prospective, observational cohort study was conducted at intensive care unit (ICU) of Al-Azhar university hospitals, between June 2022 till October 2023. A record of the trial can be obtained on ClinicalTrials.gov with the id NCT05810415.

3. Ethical considerations

Consistent with the Declaration of Helsinki, this research followed all relevant regulations. Departmental and institutional ethical committees granted approval for the research methodology. Each participant was individually provided with a comprehensive description of the study aims, process, and associated hazards, and their informed written consent was acquired.

4. Sample size estimation

The power analysis was performed utilizing version 20 of the MedCalc® Statistical Software. The effect size was estimated utilizing data provided by Jeyaraju et al., with a significance level set at 5% [9]. According to the calculations, in order to achieve a statistical power of 80% and detect the noteworthy discriminatory power of renin in predicting mortality with an area under the curve of 0.682, it was necessary to recruit 76 patients. In order to meet the specified minimum sample size requirement of 76 subjects and account for 10% for attrition, the research ultimately examined 84 patients in order to validate the hypothesis.

5. Inclusion criteria

This study included hypotensive critically-ill shocked patients, aged 21 to 60 years old, who were administered vasopressors for a minimum of 6 hours in order to keep MAP ≥65 mmHg. A minimum scheduled stay of twenty-four hours is mandatory.

6. Exclusion criteria

Families or patients who declined to engage were eliminated. Additionally, patients undergoing renal replacement therapy or beta blockers were precluded. Exclusion criteria also included conditions associated with raised serum lactate such as liver cell failure, malignancy, thiamine deficiency, seizures, compartment syndrome and drugs (metformin, epinephrine, propofol, theophylline, B2 agonists). Moreover, conditions associated with raised serum renin were excluded, such as Addison's disease, hyperthyroidism, pregnancy, liver cirrhosis, renal artery stenosis, pheochromocytoma, and drugs (diuretics, hydralazine, ACEIs, ARBs).

7. Primary outcome

The main objective was to examine the correlation involving the alteration in plasma renin and blood lactate levels and the mortality rate during hospitalization.

8. Secondary outcomes

(1) Defining renin and lactate kinetics, and correlation with the occurrence of mortality during the hospital stay.
(2) Sensitivity and specificity of plasma renin concentration and whole blood lactate for prediction of patient mortality.
(3) Comparison between survivors and non-survivors regarding vital data and laboratory parameters.

9. Procedure

Upon enrollment, the patients underwent a thorough evaluation and the data were meticulously collected by an independent investigator until either their death or the end of ICU stay. Upon completion of the trial, the primary and secondary endpoints were examined.

Quantification of blood lactate level was examined at registration and at 24, 48, and 72 hours. In addition, blood samples were collected from arterial catheters

while the subjects were supine to test the plasma renin levels. Patients prospectively had their full blood samples drawn in EDTA tubes at enrollment and then at 24, 48, and 72 hours. Centrifuged samples yielded 2 mL of EDTA plasma, which was subsequently frozen at −20°C. The samples are defrosted for batch analysis once they have been retrieved for the study. A kit for measuring active renin enzyme-linked immunosorbent assay levels in plasma was utilized.

10. Statistical analysis

The recorded outcomes were analyzed utilizing SPSS version 23.0, a statistical software designed for social sciences by SPSS Inc. in Chicago, Illinois, USA. The quantitative data was revealed employing the mean ±standard deviation, and ranges for variables that exhibited a parametric (normal) distribution. Variables that deviated from a normal distribution were identified using the median and inter-quartile range (IQR). Furthermore, qualitative features are expressed quantitatively by numerical values and percentages. The Independent-samples t-test is used to contrast the means of two parametric variables, whereas the Mann Whitney U test is appropriate for comparing non-parametric variables. The Chi-square test was employed to contrast groups with categorical data. The correlation involving plasma renin concentration and quantitative factors was assessed using Pearson correlation for variables that meet parametric assumptions, and Spearman rank correlation for those that do not meet parametric assumptions. The Kaplan-Meier method was employed to illustrate the hazard of patient mortality from the initiation of the trial till the day of death. A 95% confidence interval was established with a corresponding margin of error of 5%. P-value <0.05 deemed significant.

11. Results

This study prospectively enrolled 84 hypotensive, critically-ill patients. Twenty two patients died, while 62 patients survived. Figure 1 depicted the CONSORT schematic diagram, illustrating the progression of the study approach. There was no significant disparity in age, gender, and chronic health issues among the survivors and non-survivors groups, as indicated in Table 1.

The mean ICU length of stay for survivors was 7.48 days, as opposed to 13.41 days for non-survivors (p = <0.001). Furthermore, the mean hospital length of stay for survivors was 15.48 days, in contrast to 18.32 days for non-survivors (p = 0.004) (Table 1).

Vital variables

The opposition across the two groups illustrated a statistically significant distinction in heart rate (HR). The survivors group exhibited a lower HR, ranging from 92 to 118, with a mean ± SD of 104.98 ± 6.44. Comparatively, the non-survivors group had a higher HR, ranging from 99 to 134, with a mean ± SD of 115.36 ± 9.06 (p < 0.001). The survivors group exhibited a mean arterial pressure (MAP) of 74.87 mmHg, whereas the non-survivors group exhibited a mean arterial pressure of 67.91 mmHg (p < 0.001). Furthermore, the average temperature in survivors was 36.76, but it was 37.21 in non-survivors (p = <0.001). In addition, the non-survivors encountered a significantly enhanced mean respiratory rate opposed to the survivors (27.14 vs 24.77, p <0.001) (Table 2).

The average Glasgow Coma Scale score in the survivors was 13.95, yet it was 10.82 in the non-survivors (p= <0.001). Furthermore, the requirement for mechanical ventilation was noticed in 83.87% of the survivors and 77.27% of the non-survivors (p = 0.488) (Table 2).

12. Laboratory parameters

The current study indicated no statistically significant disparity between survivors and non-survivors, in terms of hematocrit (%) and WBC count (×103/µL), as indicated in Table 3. In addition, mean serum creatinine (mg/dL) in survivors was 1.67, while it was 1.59 in non-survivors, with no significant distinction (p = 0.305). There was no significant disparity in urine output (measured in milliliters per kilogram per hour) between survivors and non-survivors. In addition, the average blood urea level (measured in mg/dL) was 79.66 in survivors, opposed to 87.91 in non-survivors (p= 0.047) (Table 3).

The bilirubin levels (measured in mg/dL) in survivors varied from 0.7 to 1.4, with a mean of 1 ± 0.17. In non-survivors, bilirubin levels ranged from 0.8 to 1.6, with a mean of 1.2 ± 0.25. There was a statistically significant disparity (p= 0.002) across the two groups. Furthermore, the random blood glucose levels in survivors exhibited no significant variation, opposed to non-survivors (Table 3).

13. Measurement of renin and lactate levels

The mean plasma renin concentration at enrollment was 61.95 pg/ml in survivors, and 104.45 pg/ml in non-survivors (p= <0.001). The mean plasma renin concentration at 24 hours was significantly reduced in the survivors, opposed to the non-survivors (43.61 vs 98.73 pg/ml, p= <0.001). In addition, the non-survivors exhibited a significant boost in plasma renin concentration after 48 and 72 hours, opposed to the survivors (112 vs 40.89, and 106.64 vs 28.85 pg/ml) respectively (Table 4).

Furthermore, the average blood lactate concentration at registration was 1.79 mmol/L for survivors, whereas it was 2.65 mmol/L for non-survivors

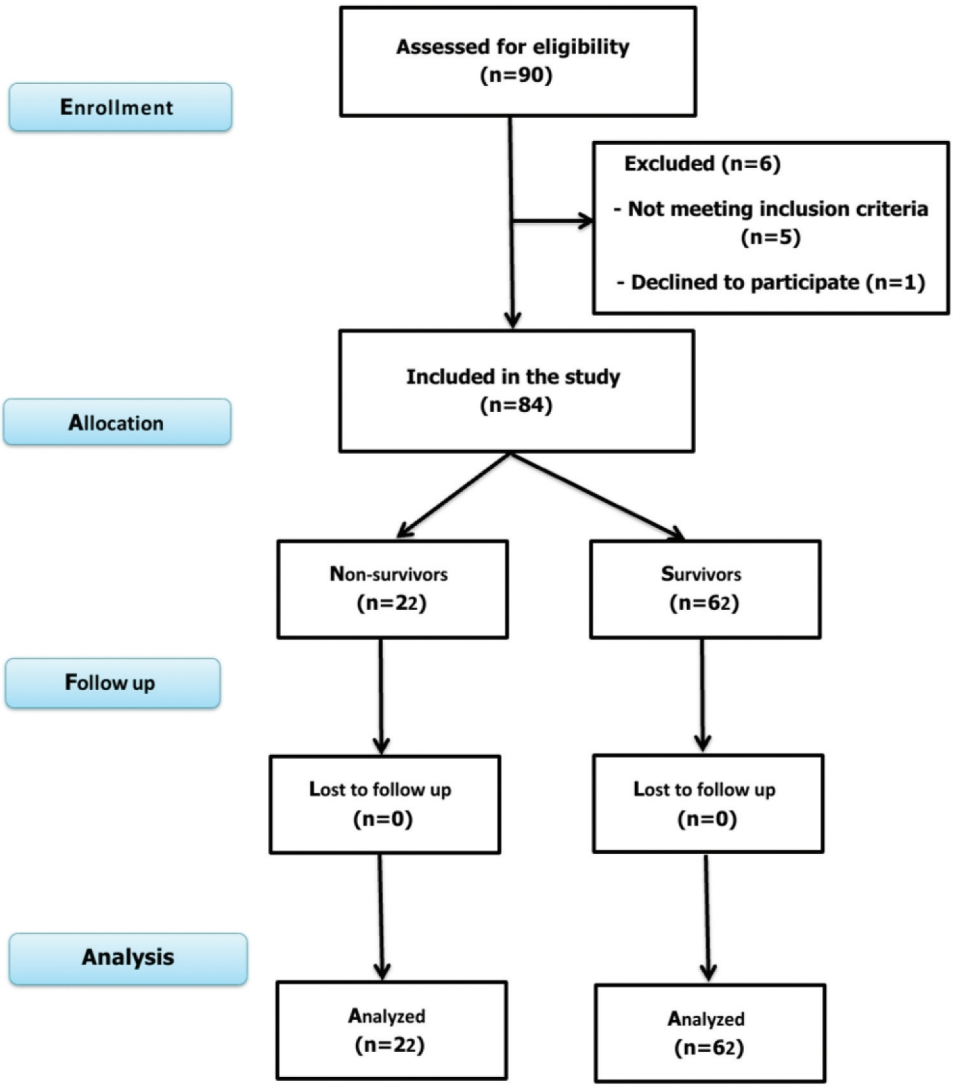

Figure 1. CONSORT flow diagram of the study process.

(p = <0.001). After 24 hours, the mean blood lactate concentration (measured in mmol/L) did not indicate a significant disparity in the two groups. Furthermore, the blood lactate levels at 48 hours were reduced in survivors, contrasted with non-survivors. Nevertheless, the distinction was not statistically significant (p = 0.342). After 72 hours, survivors exhibited a significant decline in blood lactate levels, as opposed to non-survivors (1.86 vs 2.15, p = 0.023), (Table 4).

14. Correlation between plasma renin, blood lactate and other variables

The Pearson correlation coefficient (r) across patient mortality and plasma renin levels was 0.389, indicating a significant positive correlation among the two variables. Furthermore, there was a positive correlation between blood lactate concentrations and patient mortality, as indicated by a Pearson's correlation value (r) of 0.601 (Table 5).

15. Diagnostic performance of renin and lactate

Table 6 includes the cut-off value, sensitivity, and specificity of each predictor. The Plasma renin concentration has an Area Under the Curve (AUC) of 0.744. The Cutoff value was 67.5. The Sensitivity was 90.9% and the Specificity was 54.8%. With respect to blood lactate concentrations, AUC was 0.871, cutoff value was 2.285, with 72.7% sensitivity, and 95.2% specificity.

16. Discussion

This study highlighted that Pearson correlation coefficient (r) between mortality rate and plasma renin was 0.389, implying a robust positive correlation between the two covariates. In addition, there was a significant positive correlation (r = 0.601) amongst lactate and patient mortality. Furthermore, the findings of our study implied that area under the curve (AUC) of plasma renin was 0.744, with a Cutoff value of 67.5, a sensitivity of 90.9%, and a specificity of 54.8%. Concerning blood

Table 1. Demographic data and length of stay among the study population.

	Survivors group (*n* = 62)	Non-survivors group (*n* = 22)	Test of Sig.	p
Sex			X2 = 0.031	0.861
Male	41 (66.13%)	15 (68.18%)		
Female	21 (33.87%)	7 (31.82%)		
Age (years)			*t* = 1.153	0.255
Mean ± SD.	42.35 ± 8.27	40.23 ± 7.12		
Median (IQR)	41 (37 – 49)	37.5 (36 – 44.75)		
Range (Min-Max)	33 (26 – 59)	25 (28 – 53)		
Comorbidities n (%)				
HTN	40 (64.52%)	10 (45.45%)	X2 = 2.449	0.118
Chronic kidney disease	11 (17.74%)	3 (13.64%)	X2 = 0.197	0.657
Diabetes Mellitus	14 (22.58%)	12 (54.46%)	X2 = 2.285	0.161
ICU LOS (days)			t = −7.672	<0.001*
Mean ± SD.	7.48 ± 2.01	13.41 ± 3.42		
Median (IQR)	7.5 (6 – 9)	13 (10 – 15)		
Range (Min-Max)	9 (2 – 10)	13 (8 – 21)		
Hospital LOS (days)			t = −3.087	0.004*
Mean ± SD.	15.48 ± 3.37	18.32 ± 3.81		
Median (IQR)	16 (14 – 17.75)	19 (16.25 – 20.75)		
Range (Min-Max)	15 (7 – 22)	14 (11 – 26)		
Reasons of ICU admission			X2 = 17.038	<0.001*
–Hypovolemia	15 (24.19%)	0 (0.00%)		
–Distributive shock	25 (40.32%)	20 (90.91%)		
–Cardiogenic shock	22 (35.48%)	2 (9.09%)		

χ2: Chi- Square test.
SD: standard deviation.
IQR: interquartile range.
t: Independent T test
p: *p* value for comparing between the studied groups
*P-value < 0.05: Significant.

Table 2. Vital data among the study population.

	Survivors group (*n* = 62)	Non-survivors group (*n* = 22)	Test of Sig.	P
Heart rate			t = −4.947	<0.001*
Mean ± SD.	104.98 ± 6.44	115.36 ± 9.06		
Median (IQR)	104 (100.25 – 110.75)	114.5 (109.25 – 123)		
Range (Min-Max)	26 (92 – 118)	35 (99 – 134)		
MAP (mmHg)			t = −6.469	<0.001*
Mean ± SD.	74.87 ± 7.58	67.91 ± 8.08		
Median (IQR)	71 (68 – 82)	58 (54.25 – 73.25)		
Range (Min-Max)	19 (66 – 85)	68 (52 – 75)		
Temperature			t = −5.835	<0.001*
Mean ± SD.	36.76 ± 0.21	37.21 ± 0.34		
Median (IQR)	36.8 (36.6 – 37)	37.3 (36.85 – 37.5)		
Range (Min-Max)	0.8 (36.3 – 37.1)	1 (36.6 – 37.6)		
Respiratory rate			t = −4.331	<0.001*
Mean ± SD.	24.77 ± 1.67	27.14 ± 2.36		
Median (IQR)	25 (23.25 – 26)	27.5 (26 – 28)		
Range (Min-Max)	7 (21 – 28)	9 (22 – 31)		
Glasgow Coma Scale score			*t* = 11.986	<0.001*
Mean ± SD.	13.95 ± 0.56	10.82 ± 1.18		
Median (IQR)	14 (12 – 14)	11 (13 – 11)		
Range (Min-Max)	2 (12 – 15)	4 (9 – 12)		
MV need	52 (83.87%)	17 (77.27%)	X2 = 0.482	0.488

t: Independent T test
SD: standard deviation
IQR: interquartile range.
p: p value for comparing between the studied groups
MAP: mean arterial pressure.
*P-value < 0.05: Significant.
MV: Mechanical ventilation.

lactate, the AUC was 0.871, with cutoff value of 2.285, a sensitivity of 72.7%, and specificity of 95.2%.

The outcome of our study aligned with GLEESON et al., who noticed that renin exhibited a sensitivity of 100% (with a 95% confidence interval of 61–100%) and a negative predictive value of 100% (with a 95% confidence interval of 65–100%) for ICU mortality [1]. The maximum renin level showed a significant variance in distinguishing between survivors and non-survivors (AUC, 0.80; $p = 0.04$), but the maximum lactate level did not show a significant disparity (AUC 0.70; $p = 0.17$).

Moreover, our findings were consistent with Jeyaraju et al., who noticed that the likelihood of mortality escalated by enhanced renin levels exceeding 40 pg/mL, yet not lactate levels beyond

Table 3. Laboratory parameters among the study population.

	Survivors group (n = 62)	Non-survivors group (n = 22)	Test of Sig.	p
Hematocrit (%)			t = 0.577	0.565
Mean ± SD.	32.9 ± 2.41	32.68 ± 1.09		
Median (IQR)	33 (31 – 34)	33 (32 – 33)		
Range (Min-Max)	11 (28 – 39)	4 (31 – 35)		
WBC count (×103/μL)			t = −1.944	0.061
Mean ± SD.	14.77 ± 4.72	17.36 ± 5.58		
Median (IQR)	15 (11 – 18)	17 (13.25 – 22)		
Range (Min-Max)	20 (3 – 23)	20 (6 – 26)		
Serum creatinine (mg/dL)			t = 1.037	0.305
Mean ± SD.	1.67 ± 0.38	1.59 ± 0.28		
Median (IQR)	1.7 (1.4 – 2)	1.6 (1.42 – 1.78)		
Range (Min-Max)	1.6 (0.9 – 2.5)	1 (1.1–2.1)		
Urine output (ml/kg/h)			t = 0.184	0.855
Mean ± SD.	0.92 ± 0.35	0.91 ± 0.27		
Median (IQR)	0.9 (0.7 – 1.27)	0.9 (0.7 – 1.1)		
Range (Min-Max)	1.4 (0.2 – 1.6)	1 (0.5 – 1.5)		
Blood urea (mg/dL)			t = −2.076	0.047*
Mean ± SD.	79.66 ± 12.07	87.91 ± 17.2		
Median (IQR)	80 (71 – 88)	85.5 (73.5 – 104.75)		
Range (Min-Max)	50 (56 – 106)	53 (62 – 115)		

t: Independent T test
SD: standard deviation
IQR: interquartile range.
p: p value for comparing between the studied groups
WBC: White blood cells.
*P-value < 0.05: Significant.

Table 4. Serum renin concentrations among the study population.

	Survivors group (n = 62)	Non-survivors group (n = 22)	P value
At enrollment			
Serum renin (pg/ml)	61.95 ± 45.36	104.45 ± 43.35	<0.001*
Blood lactate (mmol/L)	1.79 ± 0.47	2.65 ± 0.6	
At 24 hours			
Serum renin (pg/ml)	43.61 ± 35.68	98.73 ± 47.64	<0.001*
Blood lactate (mmol/L)	2.14 ± 0.54	2.33 ± 0.4	0.077
At 48 hours			
Serum renin (pg/ml)	40.89 ± 33.33	112 ± 46.89	<0.001*
Blood lactate (mmol/L)	2.07 ± 0.51	2.19 ± 0.5	0.342
At 72 hours			
Serum renin (pg/ml)	28.85 ± 24.86	106.64 ± 49.71	<0.001*
Blood lactate (mmol/L)	1.86 ± 0.46	2.15 ± 0.49	0.023*

Data presented as Mean ± SD.
t: Independent T test.
*P-value < 0.05: Significant.

Table 5. Pearson's correlation coefficients (r) between plasma Renin concentration, whole blood lactate and other variables.

		Pearson's correlation coefficients (r)	P
Hematocrit (%)	Lactate	0.025	0.825
	Renin	0.292	0.007*
WBC count (×103/μL)	Lactate	−0.025	0.824
	Renin	0.086	0.437
Blood urea (mg/dL)	Lactate	−0.008	0.940
	Renin	0.102	0.358
Bilirubin (mg/dL)	Lactate	0.151	0.170
	Renin	0.254	0.020*
Patient mortality	Lactate	0.601	<0.001*
	Renin	0.389	<0.001*

WBC: White blood cell
* P-value < 0.05: Significant.

2 mmol/L, were correlated to mortality occurring during the patient's hospital stay. The results demonstrated that the assessment of plasma renin kinetics may be more effective than lactate kinetics for estimating death of hypotensive, critically ill patients.[9]

In addition, MA et al. designed a study encompassing 1,393 critically ill individuals, who were divided into two groups: a group that survived for 30 days and a group that died within 30 days [10]. The analysis established that elevated serum lactate levels were a distinct risk factor for the mortality rate of critically ill patients.

Table 6. Receiver operating characteristic (ROC) curve analysis with cut-off value, sensitivity and specificity of plasma Renin concentration and blood lactate concentrations to predict patient mortality.

	Diagnostic parameters			
	AUC	Cutoff value	Sensitivity	Specificity
Plasma Renin concentration	0.744	67.5	90.9%	54.8%
Blood lactate concentrations	0.871	2.285	72.7%	95.2%

Indeed, this study identified that, the average plasma renin at the beginning of the trial was significantly reduced in the survivors, as opposed to the non-survivors (p = <0.001). In addition, the non-survivors exhibited a significant enhancement in plasma renin after 24, 48 and 72 hours, as opposed to survivors. After 24 hours, the mean blood lactate did not indicate a significant disparity between survivors and non-survivors. Furthermore, survivors exhibited a lower blood lactate level at 48 hours, opposed to non-survivors, however the distinction was not significant

The findings of our study were consistent with those of GLEESON et al., who spotted that the rate fluctuation of lactate over time did not distinguish significantly among survivors and non-survivors (p = 0.07). Additionally, they observed that having a lactate value above the limit of normality of normal (>2 mmol/L) was not a significant indicator of mortality in ICU (p = 0.30) [1].

Supportingly, LEŚNIK et al., showed enhanced plasma renin concentration in non-survivors, contrasted with survivors, with significant outcomes on the first and third day. Moreover, the authors reported that renin was a robust predictor of patient mortality. Unlike our study, the cut-off value of renin was 87 pg/mL, compared with 67.5 pg/mL in our study. This discrepancy may be postulated by the different enrollment criteria, as they enrolled sepsis and septic shock cases, and our study included hypotensive critically-ill patients [11].

This study emphasized a significant boosted HR in non-survivors, contrasted with survivors. Furthermore, the survivors had a significant higher level of MAP, whereas the non-survivors encountered hypotension.

In line with this demonstration, LEŚNIK et al., indicated a significantly declined MAP in non-survivors, contrasted with survivors. There was a constant and substantial correlation between renin, lactate and MAP on the first and fifth days [11].

Furthermore, our findings were consistent with ULINA et al., who achieved a correlation research examining the relationship between lactate and fatality among patients with vasodilatory shock [12]. The researchers encountered that MAP was significantly lower in non-survivors, contrasted with survivors (p = 0.015).

Moreover, our findings were consistent with those of GLEESON et al., who designed a prospective research examining renin as a biomarker in tissue perfusion [1]. A significant association was noticed between renin and MAP (r = −0.35; p < 0.001). Non-survivors significantly encountered more declined MAP, compared with survivors.

This study revealed no statistically significant disparity between survivors and non-survivors in terms of hematocrit (%), WBC count (×103/µL), creatinine (mg/dL), and urine output (ml/kg/h). Furthermore, the average bilirubin level among survivors was 1, while it was 1.2 among non-survivors (p = 0.002).

Our findings aligned with JEYARAJU et al., who discovered that, the measurement of lactate concentration in whole blood is frequently utilized in cases of shock to evaluate perfusion [9].

The average ICU length of stay for survivors was 7.48 days, whereas it was 13.41 days for non-survivors (p = <0.001). Furthermore, the average hospital length of stay for survivors was 15.48 days, whilst it was 18.32 days for non-survivors (p = 0.004).

These outcomes are accordant with the study accomplished by Küllmar et al., who illustrated a statistically significant disparity in the length of ICU and hospital stay between survivors and non-survivors (p < 0.002) [13].

17. Conclusion

The assessment of plasma renin kinetics offer a more accurate prediction of mortality in hypotensive, critically ill patients, compared to blood lactate. Renin and lactate had positive correlation to mortality, yet plasma renin revealed superior diagnostic accuracy over blood lactate for mortality prediction in hypotensive critically-ill patients.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Author contribution

Authors contributed equally in the research.

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