Renal Replacement Therapy

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Alactic base excess predicts the use of renal replacement therapy in patients with septic shock

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Abstract

Background Alactic base excess (ABE) is a novel biomarker that estimates the renal capability of handling acid–base alterations during the sepsis. Hence, the aim of this study was to evaluate the use of ABE to predict the renal replacement therapy (RRT) in patients with septic shock.

Methods A total of 164 patients admitted to the intensive care units with a diagnosis of septic shock according to the third international consensus on sepsis and septic shock (Sepsis-3) were included. This study was retrospective, single center, and conducted between January 1, 2016, and December 31, 2020. The individuals were stratified in patients who did [$n=68$] or did not [$n=96$] receive the RRT. The diagnostic performed of the variables for the classification into patients who required RRT was evaluated by receiver operating characteristic (ROC) analysis and area under curve (AUC) was calculated. Univariate and multivariate logistic regression models were used to identify risk factors for RRT.

Results The median age of the patients was 59 years and female sex (51.8%) predominated. ABE (odds ratio [OR] 1.2270, [95% confidence interval [CI] 1.0453–1.4403], $p=0.0124$) and urea (OR 1.0114, [95% CI 1.0053–1.0176], $p=0.0002$) were associated with risk of RRT. HCO_3- (OR 0.6967, [95% CI 0.5771–0.8410], $p=0.0002$) was a protective factor of RRT. ABE (AUC = 0.649, $p<0.0008$), HCO_3- (AUC = 0.729, $p<0.0001$), and urea (AUC = 0.76, $p<0.0001$) had a cutoff point of ≤ − 5.7 mmol/L, ≤ 19.36 mmol/L and > 75 mg/dL, respectively.

Conclusion Although HCO_3- is associated with low risk, ABE and urea are independent risk factors for RRT in the patients with septic shock.

Keywords Alactic base excess, Renal replacement therapy, Septic shock, Acute kidney injury

Introduction

Nearly half of the patients admitted to the intensive care unit (ICU) present acute kidney injury (AKI) [1], with sepsis accounting for the majority of cases at just over 50% [2]. About 10% of the patients will require renal replacement therapy (RRT) [3], although will increase up to 50% when the AKI is related to sepsis [4]. The death risk increases in parallel to the severity of AKI with an OR 2.19 (stage 1), OR 3.88 (stage 2), OR 7.18 (stage 3), and up to 80% of patients with RRT die. Seventy-five

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percent of patients receive continuous RRT and 25% receive intermittent RRT [1, 5]. Of patients presenting AKI and receiving RRT, up to 15% will be dialysis dependent, even after discharge from the hospital [6].

The hemodynamic status of the patient could influence the RRT type we use; therefore, continuous renal replacement therapy (CRRT) could be the best option in the presence of instability. Therefore, the KDIGO (Kidney Disease Improving Global Outcomes) guidelines recommend CRRT for patients with hemodynamic instability (Level of Evidence 2B) [7]. AKI requiring RRT is a life-threatening problem, so time to intervention could influence outcomes. On the other hand, hydroelectrolytic disturbances, acid–base imbalance, azo level, and amount of uresis may be useful in deciding the start of RRT. Thus, the initiation of RRT is based on the time of AKI evolution and the hydrometabolic needs of the patient [8]. The timing of RRT initiation is controversial; in fact, there is no univocal protocol among countries, hospitals, or even among intensivists and nephrologists [9].

Recently, Gattinoni et al. [10] introduced the concept of "alactic base excess," which is related to renal function. This is understood using Stewart's model for acid–base balance disturbances. As lactate increases, the apparent strong ion difference (SIDa) will decrease, resulting in metabolic acidemia (pH decrease). Renal compensatory mechanisms are responsible for correcting acidemia; if the above does not occur, impaired renal function is reflected with creatinine > 2 mg/dl and negative alactic base excess, indicating an accumulation of fixed or nonvolatile acids (unmeasured anions) other than lactate as the main cause of metabolic acidemia. An alactic base excess of zero or close to zero with creatinine ~ 2 mg/dl suggests that the kidney still can remove fixed or nonvolatile acids, but cannot fully "compensate" for metabolic acidemia, with lactate as its main cause. On the other hand, positive alactic base excess (usually with creatinine < 2 mg/dl) suggests that either renal mechanisms fully compensate for metabolic acidemia or that other mechanisms contribute to metabolic alkalemia (diuretics, decreased intravascular volume). Thus, negative alactic base excess in septic shock patients helps the physician identify that renal function is impaired. Likewise, negative alactic base excess may be useful in determining the use of RRT in this patient population.

Methods

Study design

A cohort, retrospective, observational, cross-sectional study conducted in the ICU of the Centro Médico Nacional "Adolfo Ruiz Cortines," Hospital de Especialidades No.14 of the Instituto Mexicano del Seguro Social (IMSS) Veracruz, México, from January 1, 2016, to December 31, 2020. The research protocol was approved by the local Ethics and Research Committee R-2022-3001-112 and the Federal Commission for the Protection from Sanitary Risks (COFEPRIS) 17 CI 30 193 067. The study consisted of reviewing the medical records of patients who met the inclusion criteria. This was a non-intervention study, so the informed consent present in the medical records was that of admission to the ICU. The research was carried out based on the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) methodology for observational studies [11].

Participant selection and objective

Convenience sampling was performed, which included the records of patients admitted to the ICU with a diagnosis of septic shock according to the third international consensus on sepsis and septic shock (Sepsis-3) [12]. The inclusion criteria were: age > 18 years and diagnosis of septic shock of any etiology. Patients with a history of previous RRT (peritoneal dialysis, intermittent hemodialysis, continuous renal replacement therapy), patients with renal transplantation, restrictions for RRT, do not resuscitate order, maximum therapeutic range (medical practice based on the application of extraordinary and disproportionate methods of life support in terminally ill or irrecoverable patients) and patients with pregnancy or puerperium were excluded. Patients with incomplete variables in the record were eliminated. RRT was provided at the discretion of the intensivist: TIMING OF INITIATION: the clinical judgment guides CRRT initiation, CRRT MODALITY: continuous venovenous hemodiafiltration (CVVHDF) modality was used in all patients, CRRT DOSE: effluent flow rate of 25 ml/kg/h, BLOOD FLOW RATE: typical blood flow rates (150–250 ml/min) do not affect hemodynamics, PATIENT FLUID REMOVAL: avoid net ultrafiltration rates > 2 ml/kg/h, ANTICOAGULATION: we used systemic anticoagulation with unfractionated heparin, CRRT SOLUTIONS: bicarbonate buffered solutions are available, WHEN TO STOP: the decision to discontinue CRRT is based on clinical judgment [13]. The PRSIMAFLEX® system was used. The main objective was to evaluate the association between alactic base excess and the use of RRT in patients with septic shock.

Study variables

The variables obtained were classified as general: sex, age; comorbidities: diabetes mellitus (DM), systemic arterial hypertension (SAH), heart disease, ICU stay, mechanical ventilation (MV), etiology of shock: pulmonary, abdominal, urinary, skin and soft tissue, other; norepinephrine, inotropic, Simplified Acute Physiology Score II (SAPS II) and mortality. Gasometric variables and serum renal

biomarkers were: hydrogen potential (pH), partial pressure arterial oxygen/fraction of inspired oxygen (PaO_2/FiO_2), arterial pressure of carbon dioxide ($PaCO_2$), bicarbonate (HCO_3-), standard base excess (SBE), alactic base excess (ABE), lactate (Lac), anion gap (AG), sodium-chloride difference (Na–Cl), serum creatinine (SCr) and serum urea (urea). The variables were obtained from the ICU patient admission record. The formula used to calculate the SBE was: [14]

$$\text{SBE (mmol/L)} = (HCO_{3^-} - 24.8) + 16.2 * (\text{pH} - 7.4)$$

The formula used to calculate the standard ABE was: [10]

$$\text{ABE (mmol/L)} = \text{SBE} + \text{lactate}$$

Statistical analysis

No statistical power calculation was performed before the study because the sample size is indirectly conditioned to the administrative information (clinical records) available which is finite. The distribution of quantitative variables was determined using the Shapiro–Wilk test. Normally distributed variables are expressed as mean (± standard deviation, SD) and non-normally distributed variables are presented as the median (interquartile range, IQR). Student's *t* test or Mann–Whitney *U* test was used for comparison between patients groups. Qualitative variables were presented as number and percentage (%) and their association was evaluated with the X^2 test or Fisher's exact test. Receiver operating characteristics (ROC) curves were created to evaluate the sensitivity and specificity according to the variables associated with RRT [15]. Both univariate and multivariate analyses were carried out evaluate the associations of potential risk factors with the risk for RRT. The Odds Ratio (OR) was estimated with 95% confidence intervals (CI). A p-value < 0.05 was defined as a statistical significance. Data were analyzed using the R Studio software (version 1.0.153) and IBM SPSS v.25.

Results

During this period, 180 patients were recruited, of whom 164 were included in the study cohort (Fig. 1). The individuals included in the study were stratified in patients who did [$n=68$] or did not [$n=96$] receive the renal replacement therapy. The median age was 59 years, female sex (51.8%) predominated, SAH (50%) was the most frequent comorbidity, while pulmonary etiology (41.5%) was the main cause of septic shock, the median ICU stay was 5 days and 4 days of MV, the median norepinephrine was 0.32 mcg/kg/min, 12.8% received inotropic, the median SAPS II was 71 points and mortality 50.6% (Table 1). Table 2 shows the gasometric variables and serum renal biomarkers. In the RRT group the median ABE was − 6.0 mmol/L, SCr 3.0 mg/dL, urea 116.4 mg/dL compared to the patients who did not receive the RRT where ABE was − 3.0 mmol/L, SCr 1.55 mg/dL, urea 61.5 mg/dL with statistically significant difference ($p<0.05$). Except for pH and PaO_2/FiO_2, the other variables showed significant differences. Patients who received the RRT had the highest lactate and AG, as well as, lower HCO_3-, SBE, and Na–Cl differences.

To determine the variables associated with RRT an univariate binary logistic regression analysis was performed (Table 3). The variables with highest odds ratio (OR, 95% CI) values were lactate (OR 1.3152, 95% CI 1.1302–1.5305, $p=0.0004$), AG (OR 1.0715, 95% CI 1.0074–1.1396, $p=0.0283$), SCr (OR 1.1980, 95% CI 1.0597–1.3543, $p=0.0039$), and urea (OR 1.0137, 95% CI 1.0076–1.0199, $p<0.0001$). Subsequently, variables with statistical significance ($p<0.05$) in the univariate analysis were included in the multivariate regression model with stepwise to determine the predictors of RRT (Table 3). ABE (OR 1.2270, 95% CI 1.0453–1.4403, $p=0.0124$) and urea (OR 1.0114, 95% CI 1.0053–1.0176, $p=0.0002$) were independent risk factors for RRT in the patients. In contrast, HCO_3- (OR 0.6967, 95% CI 0.5771–0.8410, $p=0.0002$) was a protective factor (Table 3).

To assess the diagnostic value of the hematologic parameters ROC curves were generated (Fig. 2a–c). Figure 2a, d shows an ABE with a cut-off point ≤ − 5.7 mmol/L, AUC 0.649 (95% CI 0.57–0.721, $p<0.0008$), sensitivity 55.88%, and specificity 71.87%. Figure 2b, d shows HCO_3- with a cut-off point of ≤ 19.36 mmol/L, AUC = 0.729 (95% CI 0.654–0.795, $p<0.0001$), sensitivity 79.41%, and specificity 56.25%. Urea had an AUC = 0.76 (95% CI 0.688–0.824, $p<0.0001$), cutoff point > 75 mg/dL with sensitivity 80.88% and specificity 67.71% (Fig. 2b, d). The comparative analysis of the baseline characteristics according to the cutoff point of ABE reveled that the gender, diabetes mellitus, etiology, pH, PaO_2/FiO_2, SBE, HCO_3-, lactate, AG, Na–Cl, SCr, and urea were statistically significant (Table 4).

Discussion

Maintaining acid–base balance depends on 3 systems: (1) intra- and extracellular buffers, (2) respiratory compensation (carbon dioxide elimination) and (3) renal compensation (elimination of non-volatile acids and renal bicarbonate synthesis). These systems are interrelated, starting to work within minutes, where renal compensation is the most effective, but takes the longest time (days) to be completed. Therefore, the deterioration of renal

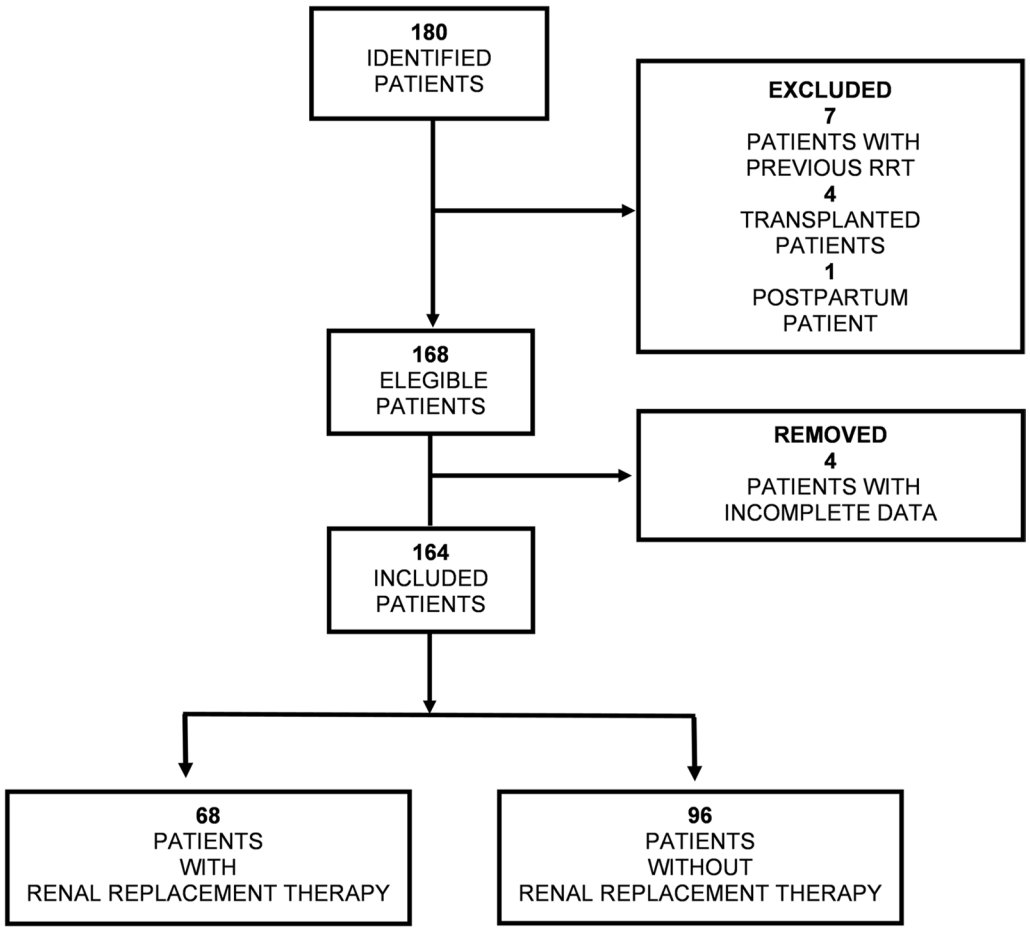


Fig. 1 Flowchart of patients included in the cohort

function leads to the accumulation or lack of elimination of non-volatile acids and, consequently, metabolic acidemia [16]. Critically ill patients with metabolic acidemia have a mortality rate of 45%, while those without metabolic acidemia have a mortality rate of 25%. The origin of metabolic acidemia is important, as lactic acidosis is the most common and the one with the highest mortality; in fact, most of the time, severe metabolic acidemia is an indication to initiate RRT [17]. In this sense, alactic base excess allows rapid differentiation of metabolic acidemia secondary to lactate accumulation from that caused by increased fixed acids, which cannot be eliminated by the lungs. Finally, the resulting acidemia indicates impaired renal function [10].

Gattinoni et al. [10] documented that metabolic acidemia in septic patients is concomitant with impaired renal function, where negative ABE is related to increased creatinine, decreased diuresis, and the use of RRT. In this study, the patients who received RRT had a median of ABE − 6.0 mmol/L (IQR − 9.95 to − 2.55), while in the individuals who did not receive RRT, the median ABE was − 3.0 mmol/L (IQR − 5.05 to − 0.60), where SCr and urea levels were higher, the median lactate was ≥ 4 mmol/L with a significant difference. Smuszkiewicz et al. [18] in 143 patients in shock state demonstrated a relationship between negative ABE and renal function deterioration, as well as increased mortality with ABE cutoff point < − 3.63 mmol/L (HR 3.19, 95% CI 1.62–6.27). In our work, mortality was higher in the patients who received RRT (58.8%) compared to the individuals who did not receive RRT (44.8%), but without significant difference. This is a recurrent trend in other studies, due to the delay in starting RRT or simply because they were more severe patients. Medina et al. [19] reported, in 414 patients, a higher 90-day mortality (58.5%) in patients who received RRT. The results are comparable to those reported by Prasad et al. [20] (64%), Kao et al. [21] (66.5%), and Gonzalez et al. [22] (68.4%).

Table 1 Baseline characteristics of the patients included in the study

Variable	Renal replacement therapy		
	Total *n* = 164	Yes *n* = 68	No *n* = 96
Age, years old (IQR)	59 (46–69)	59 (48–68)	60 (45–74)
Gender			
Female *n* (%)	85 (51.8)	39 (57.4)	46 (47.9)
Male *n* (%)	79 (48.2)	29 (42.6)	50 (52.1)
Comorbidities n (%)			
DM, Yes	73 (44.5)	34 (50.0)	39 (40.6)
SAH, Yes	82 (50.0)	40 (58.8)	42 (43.8)
Heart disease, Yes	23 (14.0)	8 (11.8)	15 (15.6)
Etiology, *n* (%)			
Lung, Yes	68 (41.5)	20 (29.4)	48 (50.0)
Abdominal, Yes	64 (39.0)	34 (50.0)	30 (31.3)
Urinary, Yes	24 (14.6)	12 (17.6)	12 (12.5)
Soft tissues, Yes	3 (1.8)	1 (1.5)	2 (2.1)
Other	5 (3.0)	1 (1.5)	4 (4.2)
Norepinefrine mcg/kg/min (IQR)	0.32 (0.21–0.48)	0.33 (0.22–0.52)	0.30 (0.21–0.45)
Inotropic, Yes *n* (%)	21 (12.8)	8 (11.8)	13 (13.5)
MV days (IQR)	4 (3–8)	5 (3–8)	4 (2–8)
ICU stay days (IQR)	5 (3–9)	5 (3–9)	6 (3–8)
SAPS II points (IQR)	71 (64–80)	72 (65–80)	70 (63–80)
Mortality, YES *n* (%)	83 (50.6)	40 (58.8)	43 (44.8)

IQR, interquartile range; DM, diabetes mellitus; SAH, systemic arterial hypertension; mcg/kg/min, microgram/kilogram/minute; MV, mechanical ventilation; ICU, intensive care unit; SAPS II, Simplified Acute Physiology Score II

An elevated urea level is almost universal during acute kidney injury [23], although in critically ill patients it can be due to multiple causes [24]. Elevated urea level could be an indication to start RRT although, there is not specific cutoff point [8]. Evidence suggests that starting RRT with a high urea level may increase mortality [25, 26]. Not only is the urea level at the start of RRT important, also a reduction of at least 25% during RRT, a value associated with mortality [27]. Our study demonstrated that ABE (≤ − 5.7 mmol/L) and urea (> 75 mg/dL) are independent risk factors for RRT.

Studies on the timing of RRT have included severe metabolic acidosis as one of the absolute indications; in fact, the variables considered were pH < 7.15 and HCO_3- < 18 mEq/L [28, 29]. The main aim of STARRT-AKI trial was to assess whether an accelerated strategy to start RRT would improve outcomes compared with a delayed initiation with absolute indications. The absolute indications for RRT included severe metabolic acidemia defined as pH ≤ 7.2 or HCO_3- < 12 mmol/L [30]. In our cohort, we found that a HCO_3- with a cutoff point of ≤ 19.36 mmol/L was a protective factor. The "optimal time" to initiate RRT is controversial and the "best variable" will remain controversial, but it is certain that medical judgment [31] should be taken into account when making this decision.

Some limitations of our study were the sample size ($n = 164$) and the fact that it was carried out in a single center; we did not have the variable of urine output; the

Table 2 Gasometric variables and serum renal biomarkers

Variable	Renal replacement therapy		
	Total *n* = 164	Yes *n* = 68	No *n* = 96
pH	7.3 (7.2–7.4)	7.3 (7.2–7.3)	7.3 (7.2–7.4)
PaO_2/FiO_2, mmHg (IQR)	96.5 (75.0–137.5)	100 (80–147.5)	93.5 (71–125)
$PaCO_2$, mmHg (IQR)	37.2 (35.6–38.7)	33.5 (32–36)	37 (36–41)*
SBE, mmol/L (SD)	− 8.38 (± 6.44)	− 10.79 (± 6.03) *	− 6.67 (± 6.19) *
HCO_3-, mmol/L (SD)	18.07 (± 5.02)	15.83 (± 4.07) *	19.66 (± 5.04) *
Lactate mmol/L (IQR)	3.2 (1.95–4.5)	4.0 (2.8–5.9) *	2.6 (1.7–3.8) *
ABE, mmol/L (IQR)	− 4.2 (− 8.25 to − 1.35)	− 6.0 (− 9.95 to − 2.55)*	− 3.0 (− 5.05 to − 0.60)*
AG, mEq/L (IQR)	14.5 (11.7–17.5)	15.3 (12.4–18.65)*	13.85 (10.25–16.75)*
Na–Cl, mEq/L (IQR)	32 (30–35)	31 (28.5–34.5)*	33 (31–36)*
SCr, mg/dL (IQR)	2.05 (1.15–3.30)	3.0 (2.10–5.35)*	1.55 (0.90–2.65)*
Urea, mg/dL (IQR)	80 (50–126)	116.4 (80–165)*	61.5 (39–94)*

pH, hydrogenion potential; PaO_2/FiO_2, partial pressure arterial oxygen and fraction of inspired oxygen; mmHg, millimeters of mercury; IQR, interquartile range; SD, standard deviation; $PaCO_2$, arterial carbon dioxide pressure; SBE, standard base excess; HCO_3-, bicarbonate; ABE, alactic base excess; AG, anion gap; Na–Cl, sodium-chloride difference; SCr, serum creatinine

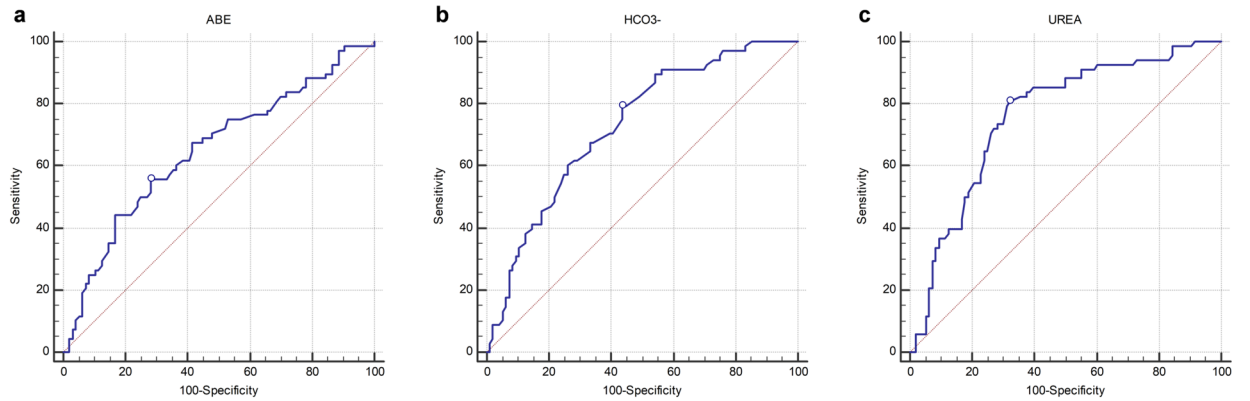
*Significant difference between groups ($p < 0.05$)

Table 3 Univariate and multivariate analysis to evaluate the importance of the variables in the patients who received the renal replacement therapy

Variable	Univariate analysis			Multivariate analysis		
	Odds ratio	95% CI	*p* value	Odds ratio	95% CI	*p* value
pH	0.0407	0.0021–0.8027	0.035*	13.212	0.002–106211.039	0.574
PaO_2/FiO_2	1.0017	0.9961–1.0074	0.547	–	–	–
$PaCO_2$	0.952	0.9196–0.9868	0.007*	1.0300	0.9440–1.1238	0.505
SBE	0.8946	0.8452–0.9468	0.0001*	0.9207	0.7367–1.1507	0.468
HCO_3-	0.8338	0.7703–0.9026	< 0.0001*	0.6967	0.5771–0.8410	0.0002*
Lactate	1.3152	1.1302–1.5305	0.0004*	1.4338	0.985–2.482	1
ABE	0.9158	0.8601–0.9751	0.0060*	1.2270	1.0453–1.4403	0.0124*
AG	1.0715	1.0074–1.1396	0.0283*	0.940	0.866–1.020	0.138
Na–Cl	0.9228	0.8576–0.9930	0.0316*	0.6956	0.2416–1.5829	1
SCr	1.1980	1.0597–1.3543	0.0039*	1.033	0.892–1.198	0.662
Urea	1.0137	1.0076–1.0199	< 0.0001*	1.0114	1.0053–1.0176	0.0002*

pH, hydrogenion potential; PaO_2/FiO_2, partial pressure arterial oxygen and fraction of inspired oxygen; mmHg, millimeters of mercury; IQR, interquartile range; SD, standard deviation; $PaCO_2$, arterial carbon dioxide pressure; SBE, standard base excess; HCO_3-, bicarbonate; ABE, alactic base excess; AG, anion gap; Na–Cl, sodium-chloride difference; SCr, serum creatinine

*Significant difference between groups ($p < 0.05$)



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Variable	AUC	95% CI	P value	Optimal cut-off point	Sensitivity (%)	Specificity (%)
Alactic base excess (ABE)	0.649	0.570 to 0.721	0.0008	≤-5.7	55.88	71.87
Bicarbonate (HCO3-)	0.729	0.654 to 0.795	<0.0001	≤19.3	79.41	56.25
UREA	0.760	0.688 to 0.824	<0.0001	>75	80.88	67.71

Fig. 2 ROC curves of predicting variables for renal replacement therapy

decision to initiate RRT is not protocolized in our ICU and depends on the intensivist in charge of the patient. The results could differ in settings where the decision corresponds to the nephrologist. The strengths are that it is a homogeneous population. Finally, the gasometric resource is possible in most hospitals.

Conclusion

Our study demonstrated that ABE and urea are independent risk factors for RRT in the patients with septic shock. Additionally, HCO_3- is associated with low risk of RRT. These results may be useful for predicting and monitoring patients with septic shock at high risk of developing RRT.

Table 4 Baseline characteristics, gasometric variables and serum renal biomarkers according to the ABE cutoff point

Variable	ABE ≤ − 5.7 *n* = 65	ABE > − 5.7 *n* = 99	*p* value
Renal replacement therapy, Yes *n* (%)	38 (58.5)	30 (30.3)	< 0.0005*
Age, years old (IQR)	62 (17.5)	57 (26)	0.351
Gender			0.019*
Female, *n* (%)	41 (63.1)	44 (44.4)	
Male, *n* (%)	24 (36.9)	55 (55.6)	
Comorbidities n (%)			
DM, Yes	37 (56.9)	36 (36.4)	0.010*
SAH, Yes	34 (52.3)	48 (48.5)	0.632
Heart disease, Yes	12 (18.5)	11 (11.1)	0.185
Etiology, *n* (%)			0.001*
Lung, Yes	15 (23.1)	53 (53.5)	
Abdominal, Yes	30 (46.2)	34 (34.3)	
Urinary, Yes	14 (21.5)	10 (10.1)	
Soft tissues, Yes	2 (3.1)	1 (1)	
Other	4 (6.2)	1 (1)	
Norepinefrine mcg/kg/min (IQR)	0.32 (0.32)	0.28 (0.29)	0.077
Inotropic, Yes *n* (%)	6 (9.2)	15 (15.2)	0.267
MV, days (IQR)	4 (5)	4 (5)	0.170
ICU STAY, days (IQR)	5 (5)	6 (6)	0.062
SAPS II, points (IQR)	70 (13.5)	71 (18)	0.926
Mortality, Yes *n* (%)	34 (52.3)	49 (49.5)	0.725
pH	7.25 (0.11)	7.34 (0.11)	< 0.001*
PaO_2/FiO_2, mmHg (IQR)	105 (65)	90 (58)	0.003*
$PaCO_2$, mmHg (IQR)	33 (11)	38 (11)	< 0.001*
SBE, mmol/L (SD)	− 13.6 (5.45)	− 4.5 (4.9)	< 0.001*
HCO_3-, mmol/L (SD)	13.6 (4.35)	20.4 (5.2)	< 0.001*
Lactate, mmol/L (IQR)	3.8 (2.4)	2.8 (2.2)	0.004*
AG, mEq/L (IQR)	17 (6.5)	13 (5.7)	< 0.001*
Na–Cl, mEq/L (IQR)	31 (4.5)	33 (6)	< 0.001*
SCr, mg/dL (IQR)	2.9 (2.75)	1.8 (1.8)	< 0.001*
Urea, mg/dL (IQR)	100 (63.75)	64 (70.3)	< 0.001*

IQR, interquartile range; DM, diabetes mellitus; SAH, systemic arterial hypertension; mcg/kg/min, microgram/kilogram/minute; MV, mechanical ventilation; ICU, intensive care unit; SAPS II, Simplified Acute Physiology Score II; pH, hydrogenion potential; PaO_2/FiO_2, partial pressure arterial oxygen and fraction of inspired oxygen; mmHg, millimeters of mercury; IQR, interquartile range; SD, standard deviation; $PaCO_2$, arterial carbon dioxide pressure; SBE, standard base excess; HCO_3-, bicarbonate; ABE, alactic base excess; AG, anion gap; Na–Cl, sodium-chloride difference; SCr, serum creatinine

*Significative difference between groups ($p < 0.05$)

Abbreviations

ABE	Alactic base excess
RRT	Renal replacement therapy
ICU	Intensive care unit
COFEPRIS (acronym in Spanish)	Federal Commission for the Protection from Sanitary Risks
IMSS (acronym in Spanish)	Instituto Mexicano del Seguro Social
AKI	Acute kidney injury
OR	Odds ratio
CRRT	Continuous renal replacement therapy
KDIGO	Kidney disease improving global outcomes
SIDa	Apparent strong ion difference
STROBE	Strengthening the reporting of observational studies in epidemiology
CVVHDF	Continuous venovenous hemodiafiltration
PRISMAFLEX	Trademark
DM	Diabetes mellitus
SAH	Systemic arterial hypertension
MV	Mechanical ventilation
SAPS II	Simplified Acute Physiology Score II
SBE	Standard base excess
AG	Anion gap
ROC	Receiver operating characteristics

Acknowledgements
Not applicable.

Author contributions
JSSD performed concepts, design, literature search, data analysis, manuscript preparations, manuscript editing and review. KGPM performed design, literature search, data analysis, manuscript preparations, manuscript editing. FBG performed literature search, data analysis, manuscript preparations. JMRR performed data analysis, statistical analysis, manuscript preparations. ORPN performed literature search, manuscript preparations, MVCS performed manuscript preparations, manuscript editing.

Funding
This work did not receive funding.

Availability of data and materials
All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
The research protocol was approved by The Local Ethics and Research Committee R-2022-3001-112 and The Federal Commission for the Protection from Sanitary Risks (COFEPRIS) 17 CI 30 193 067.

Consent for publication
This was a non-intervention study, so the informed consent present in the medical records was that of admission to the ICU. Human Subjects Research: According to the WMA Declaration of Helsinki—Ethical Principles for Medical Research involving human subjects in paragraph number 32: Medical research using human identifiable data. The informed consent document is available from the family member responsible for the patient at the time of admission to the intensive care unit, which includes protection of personal data.

Competing interests
The authors declare that they have no competing interests.

Received: 24 February 2023 Accepted: 28 July 2023
Published online: 09 August 2023

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