ORIGINAL ARTICLE

Mortality Rate and Risk Factors Associated Mortality Outcome in Medical Critically Ill Patients with Severe Lactic Acidosis

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Background: Blood lactic acid is converted to lactate, a biomarker of cellular hypoxia, which correlates with patient mortality. However, there is limited data available regarding the mechanisms of lactic acidosis and the factors associated with mortality in severe cases.

Objective: The present study aims to investigate the mortality rate among critically ill patients with severe lactic acidosis (more than 10 millimole/liter (mmol/L)) and associated factors.

Materials and Methods: A retrospective descriptive study involving 285 critically ill patients aged over 18 years, who were admitted to Srinagarind Hospital. The present study period extended from June 2018 to December 2021.

Results: A total of 285 critically ill patients with severe lactic acidosis were included, comprising 57.19% males, with a median age of 62 years (interquartile range [IQR] 52 to 72). The median baseline and maximum lactic acid levels were 12.40 (10.90 to 15.40) and 17 (12.20 to 23.30) mmol/L, respectively. The median baseline acidity [pH] was 7.20 (7.09 to 7.26). Septic shock was identified as the leading cause of severe lactic acidosis in 65.96% of cases. There were 216 in-hospital deaths, resulting in a mortality rate of 76.14%. Independent factors associated with mortality were cardiogenic shock (adjusted odds ratio [AOR]=5.53, p=0.03, 95% CI 1.15 to 26.63) and acute hepatitis (AOR=2.66, p=0.03, 95% CI 1.11 to 6.41)

Conclusion: Severe lactic acidosis has a very high in-hospital mortality rate, with cardiogenic shock and acute hepatitis identified as independent factors associated with mortality.

Keywords: Severe lactic acidosis; Mortality; Critically ill patients; Related factors

Received 2 December 2024 | Revised 21 April 2025 | Accepted 28 April 2025

J Med Assoc Thai 2025;108(Suppl.2):S7-14

Website: http://www.jmatonline.com

Under normal conditions, the body produces energy through aerobic glycolysis, converting glucose to pyruvate and then to acetyl coenzyme A for the Krebs cycle. Without oxygen, lactate production occurs instead of Pyruvate[1,2]. The body produces about 1,500 mmol of lactate daily, mainly eliminated by the liver (60%) and kidneys (30%)[1]. Normal blood lactate is 1 mEq/L[3]. Elevated lactate levels result from increased production or decreased elimination, as seen in oxygen deprivation, infections, respiratory distress, and shock[4-6].

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How to cite this article:

Anutraungkool T, Deawtrakulchai P. Mortality Rate and Risk Factors Associated Mortality Outcome in Medical Critically Ill Patients with Severe Lactic Acidosis. J Med Assoc Thai 2025;108(Suppl.2):S7-14.

DOI: 10.35755/jmedassocthai.2025.S02.S7-S14

From 1983[7] onwards, there have been studies about the use of lactate to be a biological indicator of the state of oxygen deprivation in patients with crisis conditions. From past studies, the higher concentration of lactates in the blood and even a slight rate increase of lactates in the blood are correlated to higher patient mortality rates in patients in crises[8-18], accident patients[19], and post-surgery patients[20-22].

From the literature review, there are studies of patient mortality rates in critically ill patients with lactate acidosis and relevant factors. These are mostly studies in the group of patients with mid-high lactates in their blood[7,8]. Only 2 of the studies studied critically ill patients with severe lactic acidosis, with a concentration of lactates in the blood of more than 10 (mEq/L)[12,23]. The ICU mortality rates in the group of patients were as high as 78%[12] and 65%[23]. The factors related to mortality rates included lactate clearance, sepsis, liver failure, intravenous vasopressor, and renal replacement therapy (RRT) requirement. However, there is no data available regarding the mechanisms of lactic acidosis and its correlation with the mortality rate. And the

data about factors associated with mortality in severe cases is still limited. Therefore, the present study aims to investigate the mortality rate among critically ill patients with severe lactic acidosis and associated factors, including subtypes of lactic acidosis based on mechanisms.

Materials and Methods

Study design

This is a retrospective descriptive study of medical critically ill patients with severe lactic acidosis. All patients admitted to the internal medicine ICU, Srinagarind Hospital, Khon Kaen, Thailand, from June 2018 to December 2021 were eligible for th study. The inclusion criteria consisted of 1) Patients aged 18 or over, both males and females, 2) initially detected lactate level ≥10 mmol/L at ICU admission documented in venous or arterial blood samples, and 3) Admitted to medical ICU 1 or 2 in Srinagarind Hospital, Khon Kaen, Thailand. The patients who 1) had incomplete admission data, 2) were admitted with the surgical condition, and 3) Do-Not-Resuscitate (DNR) order signed since first detected severe lactic acidosis were excluded from this study. The primary objective of this study is to find the in-hospital mortality rate and determine the factors associated with mortality outcomes in hospitalized critically ill patients with severe lactic acidosis.

Operational definitions

Severe lactic acidosis and lactate type

The authors classified patients with serum lactate levels ≥10 mmol/L into categories based on the mechanism of lactic acidosis: 1) Stagnant (systemic hypoperfusion or shock state), 2) Anemic, 3) Hypoxemia, 4) Drug-induced (type B lactic acidosis), by reviewing data from medical records. If it was inconclusive, a discussion with an intensivist was held to categorize patients into the proper subgroups. If the patients could be classified in more than one category, the result would show in combination of those subtypes.

Acute hepatitis

The authors categorized patients with elevated liver function indices, comprising alanine aminotransferase (ALT) or aspartate aminotransferase (AST), due to any cause within less than six months if they have prior liver function data, or newly detected abnormal indices if there is no previous recorded data, as “acute hepatitis”.

Metformin-associated lactic acidosis

Metformin-associated lactic acidosis (MALA) is defined as a pH <7.35 and lactate levels ≥10 mmol/L in the setting of metformin use.

Ethnic consideration and procedure

The present study was approved by the Ethics Committee of the Center for Ethics in Human Research, Khon Kaen University (HE631065, 09/20/2022), by the Helsinki Declaration. The information obtained from the study, including the patient’s history, was kept confidential and took into account the patient’s rights. The results are represented as an overview, not individual data. The present study is retrospective. However, there was no contact with patients and relatives for the patients’ blood for further information. If the information is incomplete and needs more information besides the database of outpatient records, inpatient files, and laboratory information, it must be approved by the director or supervisor of each institution before accessing the information.

The authors collected data by reviewing medical records from the hospital information systems database (Health Object system) and inpatient log files. Patient demographic data, vital signs, laboratory results including lactate levels, severity scoring systems, diagnosis, treatment, and supportive modalities were collected. Shock types and lactic acidosis subtypes were then classified by the authors, case by case, afterward.

Sample size calculations

The infinite population proportion was used for estimating the sample size. Based on a previous study, the mortality rate was 78%[12]. The estimated sample size was calculated with a Proportion (p) of 0.78, Error (d) of 0.05, Alpha (α) of 0.05, and Z (0.975) of 1.96. At least 264 patients were required to reach statistical significance for the primary outcome. A total of 285 patients were included in the present study.

Statistical analysis

The authors used the SPSS program version 26 (Khon Kaen University, Khon Kaen, Thailand) for statistical analysis. Based on descriptive statistics, categorical data were presented as numbers and percentages, and descriptive numerical data were presented as mean ± standard deviation (SD) for normally distributed parameters or median ± interquartile range (IQR) for non-normally distributed parameters. Fisher’s exact Chi-square test, Student’s t-test, and Mann-Whitney U-test were used for dichotomous variations. Multivariate analysis of the association between independent factors related to mortality and mortality outcome was determined by binary logistic regression. Variables p-value <0.15 will be selected into the model. Adjusted odds ratio (AOR) was calculated with a 95% confidence interval (95% CI). Statistical significance was obtained using a p-value <0.05.

Results

From the data collected between June 2018 and

December 2021, 320 potentially relevant patients were identified. After being excluded by exclusion criteria, a total of 285 patients were included in the present study (as Figure 1). There were 217 in-hospital deaths, with an in-hospital mortality rate of 76.14%. The patients' characteristics are shown in the table1 in both groups. 57.19% (163/285) of patients were male. The median age was 62 years. The median APACHE II score was 29 (IQR 24 to 35) in the non-survivor group and 20.50 (IQR 15.75 to 26) in the survivor group. The median SOFA score was 12 (IQR 10 to 14) in the non-survivor group and 7 (IQR 4 to 9.25) in the survivor group. The principal diagnoses in the non-survivor group were 37.33% (81/217) pneumonia, 12.44% (27/217) bacteremia, and 11.98% (26/217) intraabdominal infection, respectively. Meanwhile, in the survivor group were 25% (17/68) GI bleeding, 16.17% (11/68) Metformin-associated lactic acidosis [MALA], 11.76% (8/68) pneumonia, and 11.76% (8/68) bacteremia. As can be seen in the Table 1, 31.23% of patients (89/285) were diagnosed with pneumonia as a principal diagnosis, and hospital mortality in this group was 91.01% (81/89, p-value <0.001)

Their underlying diseases and co-morbidities are demonstrated in Table 1. Their underlying diseases included: 36.49% (104/285) diabetes mellitus (DM) and 48.07%(137/285) hypertension. 80.35% (229/285) of patients had acute kidney injury (AKI), and the hospital mortality rate was 81.7% (187/229, p-value <0.001) in this group. 46.32% (132/285) of patients had acute hepatitis with a mortality rate of 89.39% (118/132, p-value <0.001). Laboratory results, including liver function tests and creatinine, are given in the Table 1. The median baseline acidity was 7.2. The median maximum lactic acid level was 18.10 mmol/L in the non-survivor group and 13 mmol/L in the survivor group.

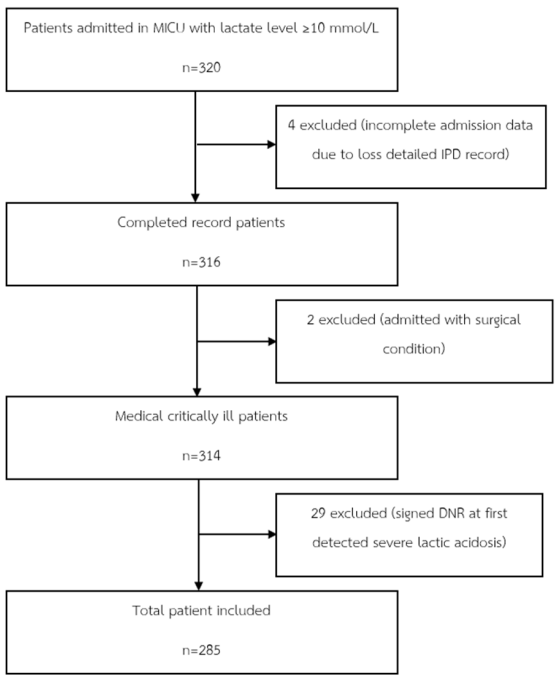

Figure 1. Recruitment flow chart.

In 285 patients, the leading cause of lactic acidosis was stagnant, which was 62.11% (177/285) of all cases. In this group, the hospital mortality was 87.01% (154/177, p-value <0.001). Hospital mortality in other subtypes tends to be lower: anemic 16.7% (1/7, p-value <0.001), hypoxemic 25% (4/16, p-value <0.001), drugs 0% (0/3, p-value = 0.002). For other combination subtypes, data are presented in Table 2. The leading shock type in both groups was septic shock, representing 65.96% (188/285) of cases with a mortality rate of 87.77% (165/188, p-value <0.001). The second most common shock type was cardiogenic shock, with an incidence of 14.74% (42/265), and the mortality rate in this group was 92.86% (39/42). Other detailed percentage distributions are shown in Table 2.

The main treatments received included: invasive mechanical ventilation (IMV) (205/285, 90.18%), intravenous vasopressors (256/285, 89.82%), bolus intravenous sodium bicarbonate (196/285, 68.77%), and RRT (157, 55.09%).

The distribution of detailed treatment in each group is included in Table 3. In 257 patients who needed IMV, the hospital mortality was 79.76% (205/257, p-value <0.001). In 256 patients who needed intravenous nor-adrenaline, the hospital mortality was 82.03% (210/256, p-value <0.001). In 196 patients who received bolus intravenous sodium bicarbonate, the hospital mortality was 83.67% (164/196, p-value <0.001), with the median bolus dose 267.60 meq (IQR 178.40 to 401.40) in the non-survivor group. The hospital mortality for 157 patients who had RRT was 87.90% (138/157, p-value <0.001).

In 285 patients with severe lactic acidosis, hospital mortality was 76.14% with a median hospital LOS of 6 days (IQR 2 to 17), ICU mortality was 73.68% with a median ICU LOS of 4 days (IQR 2 to 9), and 28-day mortality was 72.98%, as shown in Table 4.

The association between independent factors related to mortality and mortality outcome evaluated by multivariate analysis by Binary Logistic regression is demonstrated in Table 5. From this analysis, cardiogenic shock (AOR=5.53, p-value = 0.03 [95% CI 1.15 to 26.63]) and acute hepatitis (AOR=2.66, p-value = 0.03 [95% CI 1.11 to 6.41]) were significant factors associated with mortality outcome.

Discussion

Several studies have revealed that hyperlactatemia is

Table 1. Patient characteristics

Factor	All (n=285)	Death (n=217)	Alive (n=68)	p-value
Basic characteristic				
Sex, male (n, %)	163 (57.19)	123 (56.68)	40 (58.82)	0.76
Age, median (IQR)	62 (52 to 72)	62 (49 to 73)	61.50 (54.50 to 71)	0.97
Ideal body weight, median (IQR)	58 (51 to 65)	59 (52 to 64)	57.50 (48.75 to 65)	0.44
APACHE II score, median (IQR)	27(21 to 33)	29 (24 to 35)	20.50 (15.75 to 26)	<0.001
SOFA score, median (IQR)	11 (8 to 14)	12 (10 to 14)	7 (4 to 9.25)	<0.001
Body temperature, median (IQR)	37 (36.20 to 38.70)	37 (36 to 39)	37 (36.50 to 38.03)	0.74
Pulse rate, median (IQR)	119 (100 to 130)	120 (100 to 130)	110 (94.50 to 120.50)	0.05
Respiratory rate, median (IQR)	26 (22 to 30)	26 (22 to 30)	24 (20 to 30)	0.07
Mean arterial pressure, median (IQR)	58 (51 to 65)	56 (50 to 62)	67 (62.75 to 83)	<0.001
Oxygen saturation [SpO_2], median (IQR)	97 (96 to 99)	97 (96 to 99)	98 (95 to 99)	0.88
Principle diagnosis				
Pneumonia (n, %)	89 (31.23)	81 (37.33)	8 (11.76)	<0.001
Urinary tract infection (n, %)	21 (7.37)	15 (6.91)	6 (8.82)	0.60
Intra-abdominal infection (n, %)	30 (10.53)	26 (11.98)	4 (5.88)	0.15
Bacteremia (n, %)	35 (12.28)	27 (12.44)	8 (11.76)	0.88
GI bleeding (n, %)	31 (10.88)	14 (6.45)	17 (25)	<0.001
MALA (n, %)	22 (7.72)	11 (5.07)	11 (16.17)	0.003
Congestive heart failure (n, %)	3 (1.05)	1 (4.61)	2 (2.94)	0.55
Co-morbidity				
Diabetes mellites (n, %)	104 (36.49)	71 (32.72)	33 (48.53)	0.02
Hypertension (n, %)	137 (48.07)	102 (47)	35 (51.47)	0.52
COPD (n, %)	13 (4.56)	8 (3.69)	5 (7.35)	0.21
AKI (n, %)	229 (80.35)	187 (86.18)	42 (61.76)	<0.001
CKD (n, %)	84 (29.47)	64 (29.46)	20 (29.41)	0.99
Cirrhosis child A (n, %)	16 (5.61)	7 (3.23)	9 (13.24)	0.002
Cirrhosis child B and C (n, %)	23 (8.07)	20 (9.23)	3 (4.41)	0.20
Acute hepatitis (n, %)	132 (46.32)	118 (54.38)	14 (20.59)	<0.001
Solid tumor (n, %)	30 (10.53)	18 (8.29)	12 (17.65)	0.03
Lymphoma (n, %)	19 (6.67)	18 (8.28)	1 (1.47)	0.05
Leukemia (n, %)	8 (2.81)	6 (2.76)	2 (2.94)	0.94
Initial laboratory				
Hemoglobin; gm, median (IQR)	8.90 (7.30 to 11)	8.70 (7.30 to 10.60)	9.65 (7.95 to 12.13)	0.05
AST; U/dL, median (IQR)	116 (45 to 319)	128 (53 to 426)	60.50 (33 to 154.25)	<0.001
ALP; U/dL, median (IQR)	108 (70 to 185)	114 (74 to 206)	85.50 (65.75 to 136)	0.004
Total bilirubin; mg/dL, median (IQR)	1.30 (0.60 to 3.60)	1.60 (0.70 to 4.10)	0.60 (0.30 to 1.65)	<0.001
Creatinine; mg/dL, median (IQR)	2.30 (1.40 to 3.90)	2.50 (1.70 to 4)	1.65 (1 to 3.53)	0.005
pH, median (IQR)	7.20 (7.09 to 7.27)	7.20 (7.09 to 7.26)	7.21 (7.07 to 7.28)	0.51
Initial lactate; mmol/L, median (IQR)	12.40 (10.90 to 15.40)	12.50 (10.90 to 15.40)	12.10 (10.60 to 15.48)	0.27
Maximum lactate; mmol/L, median (IQR)	17 (12.20 to 23.30)	18.10 (13.60 to 25.30)	13 (11.10 to 18.18)	<0.001

associated with poor outcomes[8-23]. Our study demonstrated that patients with severe lactic acidosis had a very high mortality rate, with a nearly 80% hospital mortality rate consistent with previous studies[12,23]. Cardiogenic shock and acute hepatitis were the independent factors related to mortality significantly according to multivariate analysis.

Low cardiac output leads to impaired tissue oxygenation, resulting in lactic acidosis type A. Furthermore, some causes of cardiogenic shock, such as myocardial ischemia, can cause hyperlactatemia by regional hypoperfusion itself without cardiac function decline[24-28]. On the other hand, hyperlactatemia is considered a risk factor for reduced cardiac contractility[21,28,29]. There is still controversy about cardiogenic shock and mortality rates from previous studies[12,21,23-25,28,29]. However, a recent large meta-analysis showed decreased cardiac function related to decreased

Table 2. Lactate and shock type

Factor	All (n=285)	Death (n=217)	Alive (n=68)	p-value
Lactate type				
Anemic (n, %)	7 (2.46)	1 (0.46)	6 (8.82)	<0.001
Stagnant (n, %)	177 (62.11)	154 (70.97)	23 (33.82)	<0.001
Hypoxemic (n, %)	16 (5.61)	4 (1.84)	12 (17.65)	<0.001
Type B (drug) (n, %)	3 (1.05)	0	3 (4.41)	0.002
Anemic + stagnant (n, %)	40 (14.04)	30 (13.82)	10 (14.71)	0.86
Anemic + hypoxemic (n, %)	1 (0.35)	0	1 (1.47)	0.07
Anemic + stagnant + hypoxemic (n, %)	1 (0.35)	1 (0.46)	0	0.58
Anemic + stagnant + type B (n, %)	1 (0.35)	1 (0.46)	0	0.58
Stagnant + hypoxemic (n, %)	20 (7.02)	14 (6.45)	6 (8.82)	0.50
Stagnant + type B (n, %)	19 (6.67)	11 (5.07)	8 (11.76)	0.05
Shock type				
Septic (n, %)	188 (65.96)	165 (76.04)	23 (33.82)	<0.001
Cardiogenic (n, %)	42 (14.74)	39 (17.97)	3 (4.41)	0.006
Hemorrhagic (n, %)	27 (9.47)	17 (7.83)	10 (14.71)	0.044
Obstructive (n, %)	3 (1.05)	3 (1.38)	0	0.33

Table 3. Treatment of each group

Factor	All (n=285)	Death (n=217)	Alive (n=68)	p-value
Supportive treatment				
IMV (n, %)	257 (90.18)	205 (94.47)	52 (76.47)	<0.001
Intravenous nor-adrenaline (n, %)	256 (89.82)	210 (96.77)	46 (67.65)	<0.001
Intravenous adrenaline (n, %)	168 (58.95)	148 (68.20)	20 (29.41)	<0.001
Intravenous dopamine (n, %)	10 (3.51)	9 (4.15)	1 (1.47)	0.30
Intravenous dobutamine (n, %)	24 (8.42)	22 (10.14)	2 (2.94)	0.06
IABP (n, %)	7 (2.46)	7 (3.23)	0	0.13
ECMO (n, %)	3 (1.05)	3 (1.38)	0	0.33
Bicarbonate intravenous				
Bolus (n, %)	196 (68.77)	164 (75.58)	32 (47.06)	<0.001
Bolus dose (meq), median (IQR)	223 (178.40 to 356.80)	267.60 (178.40 to 401.40)	156.10 (89.20 to 178.40)	<0.001
Continuous (n, %)	66 (23.16)	52 (23.96)	14 (20.59)	0.57
Continuous dose (meq), median (IQR)	133.80 (133.80 to 175.31)	133.80 (133.80 to 217.43)	133.80 (133.80 to 150.53)	0.14
Renal replacement therapy				
Total (n, %)	157 (55.09)	138 (63.59)	19 (27.94)	<0.001
IHD (n, %)	14 (4.91)	7 (3.23)	7 (10.29)	0.02
CRRT				
CVVH (n, %)	63 (22.11)	57 (26.27)	6 (8.82)	0.002
CVVHD (n, %)	15 (5.26)	15 (6.91)	0	0.026
CVVHDF (n, %)	73 (25.61)	64 (29.49)	9 (13.24)	0.07
CRRT dose; ml/kg/hr, mean (SD)	30.87±3.46	30±0	30.95±3.62	0.32

lactate clearance and mortality outcomes[30].

Our study also showed that nearly 90% of patients with acute hepatitis were in the non-survivor group. Consistent with previous studies on lactic acidosis in critically ill patients[23], liver failure was a key factor associated with higher mortality rates, as the liver plays a crucial role in lactate clearance[1-6]. Hyperlactatemia in critically ill patients with liver dysfunction, especially acute liver failure, was linked to high mortality compared to patients with stable cirrhosis[23,31-34]. However, the mortality rate in our study was higher than in previous studies, likely due to a higher proportion of acute liver dysfunction patients, different causes of liver dysfunction, and less availability of liver transplantation in our setting.

Many factors, such as septic shock, RRT requirement, and intravenous vasopressors, were significantly relevant

Table 4. Mortality outcome

Factor	n=285
Hospital mortality (n, %)	217 (76.14)
Hospital LOS, median (IQR)	6 (2 to 17)
ICU mortality (n, %)	210 (73.68)
ICU LOS, median (IQR)	4 (2 to 9)
28-day mortality (n, %)	208 (72.98)

Table 5. Multivariate analysis for independent mortality factor by Binary logistic regression

Factor	AOR	p-value	95% CI
Adrenaline use	1.97	0.15	0.78 to 4.95
Acute kidney injury	2.06	0.13	0.80 to 5.28
Acute hepatitis	2.66	0.03	1.11 to 6.41
Cardiogenic shock	5.53	0.03	1.15 to 26.63
Coronary artery disease	5.45	0.08	0.81 to 36.85
Invasive mechanical ventilator use	2.68	0.10	0.82 to 8.78
Lymphoma	5.36	0.15	0.55 to 51.91
Renal replacement therapy	3.18	0.12	0.75 to 13.53
Septic shock	2.60	0.08	0.89 to 7.63

to poor outcomes in the previous studies[12,23]. In our study, these factors appeared to be associated with poorer outcomes but were not statistically significant after multivariate analysis. This might be due to an inadequate sample size, as we did not consider sample size calculation for secondary outcomes in the first place.

The authors observed that patients with lactic acidosis subtypes such as anemic, hypoxemic, and drug-induced (Type-B lactic acidosis) tended to have a better prognosis compared to the stagnant group. Data showed that more than 50% (17/31) of patients with GI bleeding as the primary diagnosis survived. Thirteen out of these 17 patients had no hemorrhagic shock, highlighting the better prognosis of the anemic subtype of hyperlactatemia. This could be beneficial as a prognosis indicator, and further study on subtype correlation is suggested.

Our study is the first in Thailand to evaluate the mortality rate and related factors in medically critical patients with severe lactic acidosis. It is the first study to examine the correlation between lactic acidosis subtypes and mortality outcomes. The obtained data emphasize the poor outcomes of patients with severe lactic acidosis and will benefit intensivists by improving the management of critically ill patients with severe lactic acidosis in terms of patient care direction, prognosis, and end-of-life decisions. We suggest stopping treatment and proceeding to palliative care in patients with high lactate and high central venous oxygen saturation ($ScVO_2$ >90%)[35], which means cytotoxic hypoxia: cells cannot already extract oxygen from blood[36].

Limitations

Due to the retrospective nature of the present study, not all patients underwent the same uniform evaluations, such as echocardiograms or advanced hemodynamic monitoring. This might affect subgroup categorization, especially regarding shock type. Additionally, the lack of a protocol for the time interval of lactate sampling resulted in missing follow-up lactate data and different time intervals between patients, leaving information regarding lactate clearance incomplete.

Secondly, the sample size may not have been sufficient to conclude secondary outcomes, especially the subtype of lactic acidosis and mortality outcomes, as previously mentioned. Additionally, because this was a single-center study, the results might not represent the majority of the population in Thailand.

Conclusion

Severe lactic acidosis has a very high in-hospital mortality rate, with cardiogenic shock and acute hepatitis identified as independent factors associated with mortality.

What is already known on this topic?

Previous international studies in severe lactic acidosis patients show very high mortality, around 80%, with a lack of data to demonstrate the mortality rate and associated risk factors in medical critically ill Thai patients.

What this study adds?

The present study shows the same result in high mortality rate in severe lactic acidosis as a previous study worldwide; additionally founded newly independent factors associated with mortality were cardiogenic shock and acute hepatitis conditions.

Acknowledgements

The authors could not have completed this project without colleagues in the Division of Internal Medicine at Khon Kaen University, as their collaboration provided us access to study this research through the electronic medical record system. Additionally, we would like to acknowledge the MICU registered nurses who assisted us with collecting MICU admission data.

Conflict of interest

The authors declare no conflict of interest.

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