

Using Blood Lactate to Predict Prognosis in Emergency Shock Patients

Wei-Kung Chen, Yi-Chang Cheng, Jheng-Jing Hung, Soon-Boek Lin, Kim-Choy Ng,
Chiung-Tsung Lin¹, Chin-Min Chuang

Department of Emergency Medicine, ¹Department of Clinical Laboratory, China Medical College

Hospital, Taichung, Taiwan, R.O.C.

Background. Lactic acidosis is frequently caused by systemic hypoperfusion, tissue hypoxia and the severity of shock. The purpose of this study was to investigate whether blood lactate concentration is a useful prognostic parameter in patients with shock in the emergency department (ED).

Methods. The criteria for patient selection were 1) arrival at ED with shock syndrome, 2) blood lactate (BLC1) > 19.6 mg/dL prior to resuscitation. All patients were monitored of blood lactate (BLC2) after resuscitation over a period of four hours prior to being sent to the operation room, intensive care unit or ordinary ward.

Results. Seventy-five shock patients had abnormal blood lactate levels (BLC1 > 19.6 mg/dL) before resuscitation. Patients who died (mortality group) had significantly higher BLC2 levels (74.4 ± 72.1 vs 27.0 ± 11.1 mg/dL, $p < 0.0001$) than patients who survived (survival group). In addition, there was a significantly higher mortality rate in patients with blood lactate concentrations (BLC1) > 100 mg/dL than patients with blood lactate concentrations (BLC2) \leq 100 mg/dL (87% vs 43%, $p < 0.005$). Patients with blood lactate concentrations (BLC2) > 50 mg/dL had a higher mortality rate than patients with blood lactate concentrations (BLC2) \leq 50 mg/dL (100% vs 36%, $p < 0.0001$). Patients with changes in blood lactate levels (BLC2-BLC1) > 20 mg/dL had a higher rate of mortality than those with a change of \leq 20 mg/dL ($n = 66$) (100% vs 47%, $p < 0.005$).

Conclusions. Blood lactate and its clearance may help emergency physicians predict the prognosis of shock patients in the ED. The detection of the changes in blood lactate concentrations after resuscitation is more valuable than before resuscitation. (Mid Taiwan J Med 2001;6:133-8)

Key words

emergency department, lactate, shock

INTRODUCTION

Assessment and treatment of shock pose a great challenge to emergency physicians due to atypical clinical presentations and difficulty of accurate evaluation during the course of shock. In the emergency department (ED), evaluation and therapy of shock is commonly

guided by physiological variables, such as mean arterial pressure, central venous pressure, heart rate and urine output. However, several clinical studies have shown that normalization of these hemodynamic variables does not improve the mortality rate in shock patients [1,2].

Lactic acidosis most frequently results from systemic hypoperfusion and tissue hypoxia. Abnormal lactate metabolism is frequently encountered among shock patients. Blood lactate levels which are supposed to

Received : April 10, 2001.

Revised : July 30, 2001.

Accepted : August 10, 2001.

Address reprint requests to : Kim-Choy Ng, Department of Emergency Medicine, China Medical College Hospital, No 2, Yuh-Der Road, Taichung 404, Taiwan, R.O.C

directly reflect the magnitude of anaerobic metabolism related to cellular hypoxia, have been correlated to survival from various forms of acute circulatory failure. The severity of lactic acidosis in critically ill patients correlates with patients' survival [3,4]. In this respect, the duration of lactic acidosis is more important than the initial lactate value. Therefore, in this study we investigated whether lactate, or the clearance of lactate, helped to predict the prognosis of patients with shock in the ED.

METHODS AND MATERIALS

This was a prospective study. The patients' criteria for participation in this study were 1) arrival at ED with shock syndrome, such as tachycardia, hypotension, tachypnea, fever or conscious disturbance, requiring management and treatment in the resuscitation room; 2) blood lactate (BLC1) > 19.6 mg/dL prior to resuscitation. Lactate was measured with an enzymatic assay (Beckman, America). Blood lactate concentrations (BLC2) were measured again 4 hours after resuscitation, prior to being sent to the operation room, ordinary ward or intensive care unit (ICU). Shock patients who had terminal cancer, were dead on arrival, or who died within an hour after resuscitation in the ED were excluded from this study.

The clinical management was under the sole direction of ED attending physicians who had received training in advanced cardiac life support (ACLS), advanced trauma life support (ATLS) or had taken an emergency trauma training course (ETTC). Patients were resuscitated according to ACLS and ATLS guidelines. Patients' vital signs, including blood pressure, heart beat and pulse oximetry were monitored in the resuscitation room.

Shock type was classified as hypovolemic shock, cardiogenic shock, distributive shock or obstructive shock, according to the diagnosis in the ED.

All quantitative data are expressed as mean \pm SD. The differences between the patient groups were analyzed by an unpaired *t* test. The Chi-square test with Yates' correction or the Fisher's exact test was used for non-parametric data. The difference between BLC1 and BLC2 was analyzed by paired *t* test. A *p* value of <0.05 was considered statistically significant.

RESULTS

Differences Between the Survival and Mortality Groups

From September 1998 to February 2000, we collected data from 75 patients admitted

Table 1. Patients characteristics and lactate levels in the mortality group and survival group

Basic data	Survival group (%)	Mortality group (%)	<i>p</i> value
Patient number	36	39	
Sex			NS
male	20 (44.4%)	25 (54.6%)	NS
female	16 (53.3%)	14 (46.7%)	NS
Age (years)	60.5 \pm 20.0	61.8 \pm 17.9	
Shock type			NS
hypovolemic	6 (40%)	9 (60%)	NS
cardiogenic	6 (55%)	5 (45%)	NS
distributive	24 (49%)	25 (51%)	NS
HR* at arrived (/min)	94 \pm 25	101 \pm 34	NS
SBP at arrived (mm/Hg)	98 \pm 42	99 \pm 45	NS
Shock index	0.96 \pm 0.18	1.02 \pm 0.23	NS
BLC1 (mg/dL)	48.7 \pm 33.3	80.2 \pm 59.1	<0.005
BLC2 (mg/dL)	27.0 \pm 11.1	74.4 \pm 72.1	<0.0001
Days in hospital	18.2 \pm 15.8	12.4 \pm 15.8 [†]	NS

*HR = heart rate; [†]Excluding the patients who died in the emergency department. SBP = systolic blood pressure; BLC1 = blood lactate concentration on arrival; BLC2 = blood lactate concentration after resuscitation; NS = not significant.

Table 2. The relationship between blood lactate and prognosis

	BLC1 (mg/dL)	BLC2 (mg/dL)	<i>p</i> value
Survival group	48.7 ± 33.3	27.0 ± 11.1	< 0.0005
Mortality group	80.2 ± 59.1	74.4 ± 72.1	NS

BLC1= blood lactate concentration prior to resuscitation, BLC2 = blood lactate concentration after resuscitation or prior to admission.

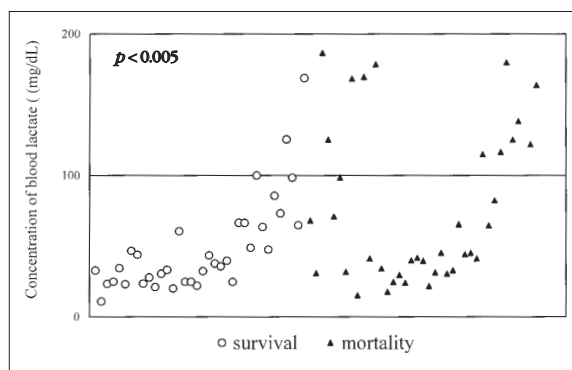


Fig. 1 The prognosis related to blood lactate concentrations prior to resuscitation (BLC1). Patients with blood lactate concentrations > 100 mg/dL had a higher mortality rate than patients with blood lactate concentrations ≤ 100 mg/dL (87% vs 43%, $p < 0.005$).

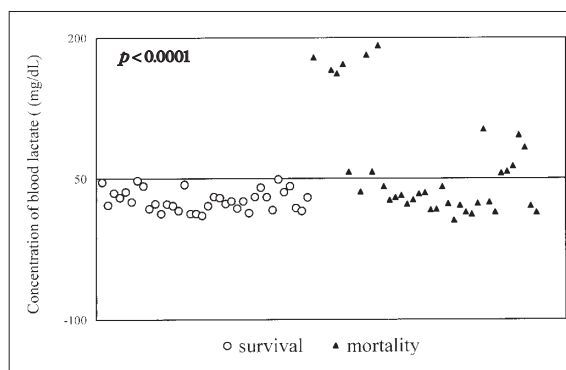


Fig. 2 The prognosis related to blood lactate concentrations after resuscitation (BLC2). Patients with blood lactate concentrations > 50 mg/dL had a higher mortality rate than patients with blood lactate concentrations ≤ 50 mg/dL (100% vs 36%, $p < 0.0001$).

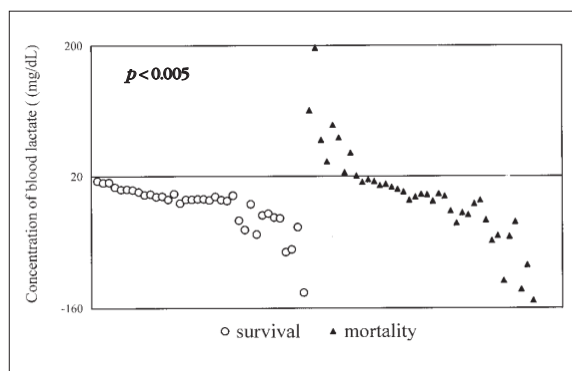


Fig. 3 The prognosis related to blood lactate concentration changes. Patients whose blood lactate levels increased to (BLC2-BLC1) > 20 mg/dL had a higher mortality rate than those ≤ 20 mg/dL (100% vs 47%, $p < 0.005$).

to our ED with shock and abnormal blood lactate prior to resuscitation. Thirty-six (45%) patients survived and 39 (55%) died after resuscitation or admission.

The basic patient characteristics and blood lactate levels of the survival and mortality groups are shown in Table 1. There were no significant differences between the two groups with regard to age, sex, vital signs on arrival, shock index, days in hospital or

types of shock. However, the blood lactate concentrations prior to resuscitation (BLC1) and after resuscitation were higher in the mortality group than in the survival group. The relationship between blood lactate clearance and prognosis before and after resuscitation is shown in Table 2. After resuscitation, blood lactate concentrations were significantly lower in the survival group ($p < 0.0005$). However, in the mortality group, there was no significant difference between blood lactate concentrations before and after resuscitation.

Lactate Level and Mortality in Shock Patients

The correlations between blood lactate concentrations before resuscitation and their prognoses are shown in Fig. 1. For the patients with BLC1 ≤ 100 mg/dL ($n = 60$), 34 (57%) survived and 26 (43%) died. There was a significantly higher mortality rate for patients with BLC1 > 100 mg/dL ($n = 15$): only 2 (13%) patients survived and 13 (87%) died ($p < 0.005$). Patients with BLC2 < 50 mg/dL ($n = 58$), 36 (64%) survived and 22 (36%) died (Fig. 2). In

contrast, there was 100% mortality ($n = 17$) with $BLC2 > 50$ mg/dL, which was significantly higher than patients with $BLC2 \leq 50$ mg/dL ($p < 0.0001$). The relationship between BLC and their prognoses is shown in Fig. 3. There was a significant difference between blood lactate levels before resuscitation (BLC1) and after resuscitation (BLC2). All patients ($n = 9$) whose BLC had increased by more than 20 mg/dL after resuscitation died ($BLC2 - BLC1 > 20$ mg/dL). However, of patients ($n = 66$) with blood lactate levels which increased by less than 20 mg/dL after resuscitation ($BLC2 - BLC1 \leq 20$ mg/dL) ($p < 0.005$), only 30 patients (47%) died.

DISCUSSION

Emergency physicians usually use the patient's vital signs and clinical presentations to determine whether or not the patient is in shock before resuscitation. However, our results revealed that there was no correlation between vital signs and prognosis, which suggests that the severity of shock can not be determined by the patients' vital signs alone. Our results are consistent with Radys' report in that the shock index was superior to vital signs, such as heart rate or blood pressure, as a parameter for estimating patients' shock status [5]. The symptoms of shock are manifold and may affect the patient's vital signs in different ways. Also, the time of onset of shock varies, so it is often difficult for the physician to respond immediately. Because the determination of shock may be affected by the assessment skills of the physician, the use of the shock index has its limitations.

Blood lactate concentrations, as a specific quantitative indicator of the perfusion deficit, can be provided quickly by most hospital laboratories. Some papers have challenged the concept that increased blood lactate levels reflect tissue hypoperfusion [6,7]. James et al postulated that increased blood lactate levels often reflect increased aerobic glycolysis in skeletal muscle secondary to epinephrine-stimulated Na^+ , K^+ -ATPase activity and not

anaerobic glycolysis due to hypoperfusion [8]. Increased blood lactate levels related to either increased pyruvate or epinephrine indicate that there are increased demands of energy and perfusion in tissue. In our study, blood lactate levels before and after resuscitation were highly correlated and BLC was found to be a good prognostic tool for shock. This finding was consistent with other studies which showed that lactate levels correlated strongly with severity of shock insult and mortality [9-11]. In contrast, Parker et al reported that the initial blood lactate concentration did not differ between survivors and non-survivors in patients with septic shock [12]. In our study, the concentrations of blood lactate after resuscitation was more accurate in predicting a patient's outcome than blood lactate levels before resuscitation. This difference suggests that the sensitivity of initial blood lactate sampling in predicting the prognosis of shock still has some limitations. The initial lactate concentration might be more weakly correlated with prognoses when tissue hypoperfusion has occurred or deteriorated during observation or treatment in the emergency room.

Falk et al monitored sequential lactate concentrations over a period of time in 24 patients with circulatory shock [13]. A delayed lactate clearance rate was observed in the non-survival group. Patients who survived the study period had progressive clearing of lactate. In our study, the clearance percentage was 45% in the survival group compared to 7% in the mortality group. Our results showed that when the clearance amount ($BLC2 - BLC1$) was less than 20 mg/dL, no patients survived. This may have been due to either irreversible shock or inadequate resuscitation. Jeng et al reported that serum lactate may be one of the parameters of inadequate resuscitation of patients [14]. A greater reduction of lactate during therapy was noted in the survivors relative to non-survivors [12]. Similarly, Vincent et al noted that patients in circulatory shock who experienced a reduction in lactate $> 5\%$ of the initial value during the first hour of

treatment had a better prognosis than patients who did not [15]. When patients are resuscitated to the clinician's satisfaction, serial lactate concentrations should be monitored over time. If lactate levels are high, more aggressive resuscitation may be required, which may include mechanical ventilation support, fluid supplement or medication to improve cardiac output. If resuscitation is delayed or inadequate, patient mortality rates may be higher. Serial blood lactate measurements every 8 hours have been used to predict patients' prognoses [16,17]. Patients in these studies were in intensive care units, not in the emergency department. For emergency physicians, the efficiency of resuscitation is more important than the prognosis after admission. Emergency physicians need more information to decide on a strategy during resuscitation and can not depend solely on the patients' vital signs. Lactate is a good indicator of the efficiency of resuscitation but the optimum time for rechecking the blood lactate during the period of resuscitation requires further study.

In conclusion, blood lactate concentration is a good parameter for predicting patient outcome in the emergency room. Changes in lactate levels after resuscitation can also provide a useful reference for prognoses.

ACKNOWLEDGEMENT

This study was supported by a grant (DMR-88-037) from the China Medical College Hospital.

REFERENCES

1. Shoemaker WC, Appel PL, Bland R, et al. Clinical trial of an algorithm for outcome prediction in acute circulatory failure. *Crit Care Med* 1982;10:390-7.
2. Bland RD, Shoemaker WC, Abraham E, et al. Hemodynamic and oxygen transport patterns in surviving and nonsurviving postoperative patients. *Crit Care Med* 1985;13:85-90.
3. Bakker J, Coffernils M, Leon M, et al. Blood lactate levels are superior to oxygen-derived variables in predicting outcome in human septic shock. *Chest* 1991;99:956-62.
4. Rashkin MC, Bosken C, Baughman RP. Oxygen delivery in critically ill patients. Relationship to blood lactate and survival. *Chest* 1985;87:580-4.
5. Rady MY, Rivers EP, Nowak RM. Resuscitation of the critically ill in the ED: response of blood pressure, heart rate, shock index, central venous oxygen saturation, and lactate. *Am J Emerg Med* 1996;14: 218-25.
6. Gore DC, Jahoor F, Hibbert JM, et al. Lactic acidosis during sepsis is related to increased pyruvate production, not deficits in tissue oxygen availability. *Ann Surg* 1996;224:97-102.
7. Astiz ME, Rackow EC. Septic shock. [Review] *Lancet* 1998;351:1501-5.
8. James JH, Luchette FA, McCarter FD, et al. Lactate is an unreliable indicator of tissue hypoxia in injury or sepsis. [Review] *Lancet* 1999;354:505-8.
9. Bernardin G, Pradier C, Tiger F, et al. Blood pressure and arterial lactate level are early indicators of short-term survival in human septic shock. *Intensive Care Med* 1996;22:17-25.
10. Dunham CM, Siegel JH, Weireter L, et al. Oxygen debt and metabolic acidemia as quantitative predictors of mortality and severity of the ischemic insult in hemorrhagic shock. *Crit Care Med* 1991;19:231-43.
11. Abramson D, Scalea TM, Hitchcock R, et al. Lactate clearance and survival following injury. *J Trauma* 1993;35:584-9.
12. Parker MM, Shelhamer JH, Natanson C, et al. Serial cardiovascular variables in survivors and nonsurvivors of human septic shock: heart rate as an early predictor of prognosis. *Crit Care Med* 1987;15:923-9.
13. Falk JL, Rackow EC, Leavy J, et al. Delayed lactate clearance in patients surviving circulatory shock. *Acute Care* 1985;11:212-5.
14. Jeng JC, Lee K, Jablonski K, et al. Serum lactate and base deficit suggest inadequate resuscitation of patients with burn injuries: application of a point-of-care laboratory instrument. *J Burn Care Rehabil* 1997;18:402-5.
15. Vincent JL, Dufaye P, Berre J, et al. Serial lactate determinations during circulatory shock. *Crit Care Med* 1983;11:449-51.
16. Abrarnson D, Scalea TM, Hitchcock R, et al. Lactate clearance and survival following injury. *J Trauma* 1993;35:584-9.
17. Bakker J, Gris P, Coffernils M, et al. Serial blood lactate levels can predict the development of multiple organ failure following septic shock. *Am J Surg* 1996;171:221-6.

測定血中乳酸濃度對預測急診病患預後之探討

陳維恭 鄭宜昌 黃正金 林順木 黃金財 林炯聰¹ 莊錦銘

中國醫藥學院附設醫院 急診部 醫學檢驗部¹

背景 乳酸通常會由於組織灌流不足、缺氧或重度疾病而產生，本研究的目的是探究在急診室偵測休克病患血液乳酸濃度可否做為預測其預後的工具。

方法 進入本研究的病患條件是 1) 進入急診時伴隨有休克症候群，2) 急救前血液乳酸濃度出現異常值 (>19.6 mg/dL) 的病患。這些病患於接受急救後 4 小時或者是在要送入開刀房、加護病房或一般病房前，均再次接受血液乳酸濃度的測定。

結果 本研究共收集 75 位病患於急救前有不正常的血液乳酸濃度 (>19.6 mg/dL)。死亡的病患急救後的血液乳酸濃度高於存活的病患 (74.4 ± 72.1 vs 27.0 ± 11.1 mg/dL, $p < 0.0001$)。除此外，當急救前的血液乳酸濃度大於 100 mg/dL 時其死亡率也比小於 100 mg/dL 為高 (87% vs 43%, $p < 0.005$)，急救後的血液乳酸濃度若大於 50 mg/dL 時其死亡率也比小於 50 mg/dL 為高 (100% vs 36%, $p < 0.0001$)，而急救前及急救後的血液乳酸濃度的清除量若大於 20 mg/dL，其死亡率也比小於 20 mg/dL 為高 (100% vs 47%, $p < 0.005$)。

結論 測定乳酸濃度及其變化可已協助急診醫師在急診室預測病患的預後。急救後乳酸濃度的測定在預測病患的預後上比急救前有價值。(中台灣醫誌 2001;6:133-8)

關鍵詞

急診，乳酸，休克

聯絡作者：黃金財

地址：404 台中市北區育德路 2 號

中國醫藥學院附設醫院 急診部

收文日期：4/10/2001

修改日期：7/30/2001

接受日期：8/10/2001