

Relevance of Blood Lactate Measurements as a Marker of Resuscitation in Patients with Severe Burns

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ABSTRACT

In major burns the traditional clinical parameters (urine output, and mean arterial pressure) provide insufficient information to serve as a guide for resuscitation. Other semi-invasive or invasive techniques have some potential hazards, methodological problems and need expensive equipment. Blood lactate level has been suggested as a resuscitative parameter. It helps to identify patients who are not adequately resuscitated and in a further need for fluids after conventional parameters have been normalized. It also has a predictive value. In this study, the diagnostic impact of initial and serial blood lactate determinations in assessing adequacy of fluid resuscitation in severe burns and predicting burn severity and outcome were examined.

Data from 40 adult patients admitted to the Burn Unit ICU of Ain Shams University Hospitals were analyzed. Resuscitation was guided by the Parkland formula. Blood lactate level was measured upon admission and every 6 hrs during the initial 48 hrs after the burn injury.

Initial and 48hrs lactate levels were elevated in all patients. With resuscitation, urine output and blood pressure were normalized and blood lactate decreased relative to the degree of success of resuscitative measures. This was more evident in patients with burns covering less than 35% of BSA, and in survivors rather than non survivors, indicating lactate level sensitivity to adequacy of resuscitation.

Patients with inhalation injury had higher percentage of BSAB and demonstrated significantly higher initial and serial blood lactate levels and worse outcome. The changes in blood lactate correlated well with burn severity and had an accurate predictive value.

Data obtained from this study demonstrated that measurement of blood lactate is strongly related to burn severity, outcome and proved to be a reliable marker for adequacy of fluid resuscitation.

INTRODUCTION

Fluid resuscitation after major burn injuries is the most important corner stone in burn management and is directly related to patient outcome. Different formulas were designed to establish a standard regimen for burn resuscitation by crystalloid infusion at a rate of 2-4mL/kg/% BSAB in the first 24 hrs following the thermal injury.

These treatment standards, however, have been increasingly objected during the last years as a rising number of publications reported on suboptimal resuscitation and hypo-perfusion when any of these formulas is used alone [1,2].

Standard criteria for adequate resuscitation have included monitoring of urine output (UOP) and blood pressure with production of 0.5-1.0mL urine/kg/h in adults and normalization of BP as hemodynamic targets. However, multiple studies [1,3-5], concluded that these traditional clinical parameters provide insufficient information to serve as a guide for major burn resuscitation. This highlights the shortcomings of these commonly measured clinical variables in the critically ill burned patients.

Global oxygenation indices such as oxygen delivery (DO₂) and oxygen consumption (VO₂) and intra-gastric PH (PHi) have been proposed as possible markers of resuscitation, but it was found that, they have more predictive value rather than being end-points of resuscitation [6].

As a consequence, more invasive hemodynamic monitoring as CVP and intra-thoracic blood volume (ITBV) have been used more frequently to guide volume therapy during the resuscitation phase of major burns. It was associated with significantly higher fluid administration than predicted by the empirical formulas [7,8]. Again, this raised a debate regarding the adequacy of the current resuscitation formulas and the need for more practical and reliable parameters of monitoring [7]. Although invasive monitoring will identify all perfusion deficits, but unfortunately, this is labor intensive, may have potential hazards in burn victims, and associated with serious methodological problems in addition to the expensive equipments and catheters needed [9].

Most deaths in the critically traumatized patients and severely burned patients in ICU are secondary to multiple organ failure (MOF) which is an end-stage of the systemic inflammatory response syndrome (SIRS) and is most often a result of repeated or continuing hypo-perfusion [10]. So, organ perfusion is the ultimate goal for a successful resuscitation.

In patients with severe trauma or burns, failure to establish an adequate organ and cellular perfusion results in a cellular hypoxic state and shift to anaerobic metabolism on the cellular level. This increases the production of lactic acid denoting inadequate resuscitation. Lactic acidosis is frequently seen in critically ill patients. Despite large number of potential etiologies, tissue hypoperfusion is by far its most common etiology among the critically ill. Aggressive cardio-respiratory resuscitation designed to restore tissue perfusion is the fundamental approach to these patients.

Accordingly, lactate and base deficit have been suggested as resuscitative endpoint parameters. They help to identify patients who are not adequately resuscitated and in a further need for fluids after conventional parameters have been normalized.

Both parameters also add information for assessment of burn severity and help to improve therapeutic strategies [10]. They are also highly reliable outcome predictors and correlate with organ failure and survival. But in a cohort of burned patients, initial and serial lactate measurement was proved to be a better predictor of mortality than initial and serial base deficit [5].

The purpose of this study is to examine the diagnostic impact of initial and serial lactate levels determination in predicting burn severity, adequacy of fluid resuscitation and patient outcome in severe burns.

MATERIAL AND METHODS

Forty adult burned patients with 20% to 85% BSAB (mean 40.85%), admitted within six hours of the injury to Ain-Shams University Burn Unit ICU were enrolled in this prospective study. Twenty six patients were males and fourteen were females. Patients' age ranged from twenty to fifty six years (mean 35.05 years).

Immediately after admission, all patients had an initial blood lactate measured. The patients were then resuscitated according to the Parkland formula using lactated Ringer's solution. Fluid administra-

tion rates were adapted in order to maintain urine output of 0.5-1.0mL/kg/h and a mean arterial blood pressure at or greater than 65mmHg.

Blood lactate level was measured and recorded every 6 hours during the initial resuscitation period (first 48hrs) by Accutrend Lactate®-Roche, using lactate test strips. A normal lactate level was defined as ≤ 2 mmol/L [11]. Demographics, clinical, and laboratory data were recorded during the same period, as well as the final patient outcome. All the data were statistically analyzed by Chi-Square and ANOVA tests.

RESULTS

The study included forty adult burned patients. The mean age was 35.05 years (range: 20-56 years) and the mean BSAB was 40.85% (range: 20-85%). Fifty percent of the patients (20 patients) had sustained inhalation injury that was bronchoscopically verified. No patient died directly due to burn shock. All deaths (16 patients) occurred after the initial post burn resuscitation phase and were caused by inhalation injury, sepsis or MOF.

There was a significant difference between survivors (S) and non-survivors (NS) regarding burn size (28.5% BSAB in S versus 46.8% in NS), but no significant difference in age was found (30.4 years in survivors versus 37.9 years in non survivors) (Table 1).

The median time between burn and admission was 2.1hrs (range: 45 min. – 6 hrs) and there was no significant difference between survivors and non-survivors concerning this time interval ($p=0.328$).

Patients with inhalation injury had higher percentage of BSAB and demonstrated significantly high initial and serial lactate level and had the worst final out come.

To reach the targeted resuscitation parameters, adequate UOP and BP, the patients received more fluids than calculated by Parkland formula. The mean UOP was 52.6mL/h and the mean blood pressure was 64mmHg throughout the study period.

The initial blood lactate was high in all patients. It correlated with the extent of BSAB being highly significant ($p=0.000$). In burns more than 35%, it was always >4 mMol/L (mean 5.1) and between 2-4 mMol/L (mean: 2.65) in burns less than 35% BSAB.

The initial blood lactate level correlated also with the final outcome. It was relatively high in

survivors (mean: 3.65) in comparison to extremely high levels reaching up to 9.9 mMol/L in non-survivors (mean: 5.26).

The subsequent samples showed steep reduction in blood lactate level at all time points throughout the study period. This reduction was steady and approached near normal values in patients with

burns less than 35% BSAB in comparison to those with more extensive burns (Fig. 1).

In survivors, the serial blood lactate level decreased progressively. On the other hand, in non-survivors, the mean level remained high near its initial values with non significant changes (Fig. 2).

Table (1): Details of the patients.

Outcome	Number	Age in years	BSAB %	No inhalation injury	Inhalation injury
Total patients	40	20-56 ys (Mean: 35.05)	20-85% (Mean: 40.58)	20	20
Survivors	24	20-44 ys (Mean: 30.4)	20-46% (Mean: 28.5)	18	6
Non-survivors	16	23-56 ys (Mean: 37.9)	36-85% (Mean: 46.8)	2	14

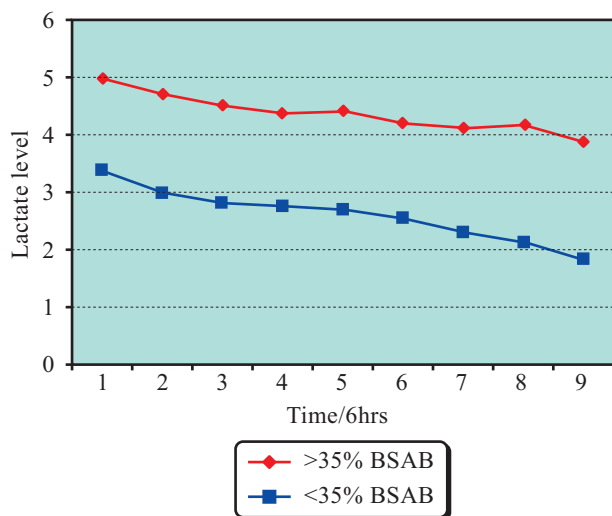


Fig. (1): Mean lactate level in relation to % BSAB.

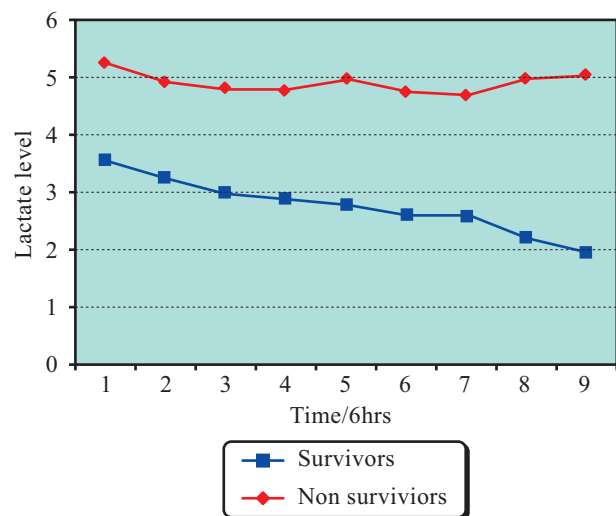


Fig. (2): Mean lactate level in relation to outcome.

DISCUSSION

Baxter in 1974 [12], suggested that complete resuscitation from burn shock is achieved with the return to normality of blood pressure, heart rate and urine output. These traditional vital signs are beneficial but proved to be invalid as outcome related resuscitation goals and were too insensitive to ensure appropriate fluid replacement. Stopping the volume resuscitation in critically injured patients after normalization of these parameters may leave these patients in a state of compensated shock and promote hypo-perfusion. Repeated or continuous hypo-perfusion will ultimately results in multiple organ failure (MOF) [13], which is an end-stage of the systemic inflammatory response syndrome (SIRS) [14].

A later definition of successful resuscitation would be that resuscitation is complete when the oxygen debt has been repaid; tissue acidosis is eliminated and aerobic metabolism restored [15].

Data derived from invasive hemodynamic monitoring of burned patients were informative and delivered evidence that burn resuscitation as practiced with existing formulas produces inadequate circulatory responses, and that additional crystalloid administration is necessary to enhance hemodynamic functions and to optimize oxygen delivery and consumption [8,16]. These invasive monitoring parameters and techniques, yet accurate, were too complicated, expensive and need well trained personnel.

An optimal marker of adequate resuscitation would possess a number of desirable qualities: accurate, easy, fast and sensitive to the changes in clinical conditions or in response to resuscitation. Blood lactate as an indirect marker of global perfusion is an optimal parameter and fulfilled most of these criteria.

Lactate is a byproduct of anaerobic cellular metabolism, elevated in hypo perfusion states due to insufficient cellular oxygen supply. In anaerobic conditions, pyruvate, which is an intermediate product of glycolysis, cannot proceed to the Krebs cycle with subsequent mitochondrial oxidative phosphorylation. As a result, the pyruvate is converted to lactate through the enzyme lactate dehydrogenase. So, lactate production exceeds its rate of metabolism in the liver and kidneys, resulting in an elevation in its blood level. Furthermore, decreased liver perfusion caused by the presence of a generalized capillary leak may result in slowing of the rate of lactate clearance [11].

Elevated blood lactate has been correlated strongly with mortality in critical patients. The rapidity at which lactate is cleared from the blood through vigorous resuscitation strongly correlates with ultimate outcome, including organ failure and mortality [17].

In this prospective study, determination of the validity of the blood lactate level is tested in patients with severe burns. Lactate levels were high in all patients at all time points over the study period.

Initial and serial lactate levels were higher in those with extensive burns and the values were proportionate to burn size, denoting strong correlation between initial lactate level and burn severity represented mainly by the extent of burn.

Unexpectedly, delay in admission did not have much impact on the initial lactate level or even final outcome. This goes with other reports [18], but though the mean delay time in the current study was much longer (2.1hrs), it still showed non significant difference.

On the other hand inhalation injury did have an effect. Lactate levels were higher in patients with inhalation injury and the percentage of non-survivors was higher than in patients without inhalation injury, (Table 1). These results are in accordance to other reports [19-21].

Serial lactate measurements showed progressively decreased levels with resuscitation. The

changes were marked in all groups in relation to the admission values.

In patients with major burns (>35% BSAB), though the decrease in lactate level with resuscitation did not reach the normal by the end of the study period, the levels were progressively declining in a response parallel to that seen in patients with less extensive burns (<35%) with difference only in the numerical value (Fig. 1).

This reflects the sensitivity of blood lactate level to the adequacy of fluid resuscitation; and that the resuscitation resulted in better perfusion and improved the cellular hypoxic stage in response to the fluids given. The fact that it did not reach the normal levels is that the used perfusion protocol was short of providing the actual patients needs.

On contrary, in non survivors, the initial high levels did not respond to resuscitation, though the other clinical parameters relatively did, indicating the predictive value of blood lactate estimation and the failure of resuscitative measures to restore perfusion or even improve patient condition. This was translated to persistently high blood lactate levels (Fig. 2).

In conclusion, the value and pattern of changes in the initial and serial lactate levels correlates with burn severity, response to resuscitation and predict final patient outcome.

Blood lactate can be used as a good marker for defining endpoint of burn shock resuscitation and indicates when supportive therapies are restoring perfusion. This information is important for enhancement of treatment strategy or selection of different therapeutic options. Tailoring fluid needs according to blood lactate level is tempting, but it needs extensive study on a bigger patient population.

The availability of blood lactate estimation as a bedside and simple monitoring parameter is helpful in limited resource burn facilities to screen patients who are in need for more care. It should become one of the standards in the treatment of life threatening burns and to be added as an important parameter in burn scores.

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