



## PROGNOSTIC VALUE OF SERIAL SERUM LACTATE LEVELS IN POLY TRAUMA PATIENTS

### Community Medicine

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### ABSTRACT

**Background:** Accurate triage tools such as blood pressure, heart rate, pulse rate, and injury severity score lack the sensitivity to identify at risk trauma patients in Emergency Department. Marker of hypo perfusion such as lactate and base-deficit reflect perfusion defect and being shown to be helpful in the identification of high risk trauma patients. Our aim was to find whether serial blood lactate levels could be predictors of outcome in polytrauma patients.

**Methods:** This is a prospective analytical study involving all trauma patients admitted to Aarupadai Veedu Medical College & hospital, puducherry, between December 2008 to July 2010, were included. Injury severity score and Glasgow coma scale score were recorded at admission. Blood Pressure, heart rate arterial blood oxygenation, were obtained and serum lactate levels were measured at admission, 6, 12 and 24 hrs intervals. Patient with lactate levels greater than 2.5 mmol/l underwent invasive monitoring and vigorous resuscitation to correct their lactic acidosis. **Results:** The Nonsurvivors (n=8) had similar blood lactate levels initially ( $17.2 \pm 2.9$  mmol/l versus  $2 \pm 1$  mmol/l)  $P=0.002$  but had higher levels after 6 hours ( $4.3 \pm 0.6$  mmol/l versus  $2.3 \pm 0.8$  mmol/l)  $P=0.07.1$ (NS) after 12hours ( $5.5 \pm 1.3$  mmol/l versus  $6.3 \pm 1.5$  mmol/l),  $P=0.13$ (NS) and after 24 hours ( $10.3 \pm 1.9$  mmol/l versus  $4.5 \pm 3.3$  mmol/l),  $P=0.27$  as compared with survivors (n=70). There were no significant differences in blood pressure, heart rate, and arterial blood oxygenation, between survivors and Non survivors. Duration of stay categorized into three groups, upto 7 days, 8-14 days, 15- 30 days. Among this groups serum lactate levels were compared which was taken initially at admission and 6<sup>th</sup>, 12<sup>th</sup>, 24<sup>th</sup> hours after admission). Among 78 patients studied, 76 patients showed elevated serum lactate levels at admission and all were subjected to aggressive fluid resuscitation. In spite of that, there were 8 casualty, 10 cases of Multisystem organ failure and 14 had Respiratory complications within 24 hours of admission.

**Conclusion:** The study showed that the serum lactate levels even though altered initially is not a good prognostic marker for survival of trauma patients.

### KEYWORDS

serum lactate level, polytrauma etc.

### INTRODUCTION

Lactate is the final product of the anaerobic glycolysis and serve as indicator for the oxygen status in the cellular tissue. A relationship between increased blood lactate levels and the presence of oxygen dept (tissue hypoxia) in patients with circulatory shock was suggested as early as 1927(1). When first described by Gaglio in 1886, measurements of lactate levels required the collection of 100 – 200 ml blood and took several days to complete. The labor - intensive nature of early lactate measurements techniques limited their clinical use, become result were not available until long after their therapeutic decisions had to be made. In 1964 Broder and weil (2) were the first to use a photo spectrometric method to measure lactate levels in whole blood decreasing turnaround times greatly. Current handheld device and mobile blood gas analyzers have decreased turnaround time to less than 2 min using minimal amount of blood (3) Acidosis arises from an increased production of acids loss of alkali, or decreased renal excretion of acids. The underlying etiology of metabolic acidosis is classically categorized into those that cause an elevated anion gap (AG) and those that do not. Lactic acidosis, identified by a state of acidosis and an elevated plasma lactate concentration is one type of anion gap metabolic acidosis and may result from numerous conditions. The normal blood lactate concentration in unstressed patients is 0.5-1 mmol/L. Patients with critical illness can be considered to have normal lactate concentrations of less than 2 mmol/L. Hyperlactatemia is defined as a mild-to-moderate persistent increase in blood lactate concentration (2-5 mmol/L) without metabolic acidosis, whereas lactic acidosis is characterized by persistently increased blood lactate levels (usually >4-5 mmol/L) in association with metabolic acidosis.

### Mortality/morbidity

Patients exhibiting a disorder of lactate metabolism are typically significantly ill and are at risk for developing multiple organ failure. Patients suffer a hospital mortality rate that increases nearly linearly

with the concentration of serum lactate. Several studies have shown that vigilant correction of hyperlactatemia is associated with decreased morbidity and mortality. The mortality rate of patients with a serum lactate level greater than 2 mmol/L persisting after 24 hours with an associated acidemia approaches 70%.

### Systemic effects of acidosis

#### Respiratory

Dyspnea, tachypnea, Kussmaul respirations: Increased minute ventilation  
Decreased diaphragm contractility

#### Cardiac

Decreased catecholamine responsiveness: Decreased fibrillation threshold  
Decreased contractility at pH <7.1: Increased heart rate and contractility at pH >7.2

#### Neurologic

Increased cerebral blood flow: Decreased cerebral metabolism  
Altered mental status: Increase sympathetic catecholamine discharge

#### Others

Decreased renal and hepatic perfusion  
Increased metabolic rate  
Increased protein catabolism

### Classification of lactic acidosis

The most frequent cause of lactic acidosis is poor tissue perfusion, which is induced by various shock state causing tissue hypoxia. In ischemic tissues of the skeletal muscle (and less significantly intestine, erythrocytes, and brain), production of lactate is accelerated with a concomitant fall in lactate consumption by the liver, kidney, and myocardium. The accumulation of a normally balanced level of serum lactate overwhelms the body's buffering capacity and results in

acidosis. Lactic acidosis occurring from associated underlying diseases, known as type B1, has been identified with diabetes mellitus, bowel ischemia, liver ,severe iron deficiency anemia. Disease, alcoholic ketoacidosis,pancreatitis, malignancy (leukemia, lymphoma, lung cancer), infection, renal failure, seizures, heat stroke, pheochromocytoma, short gut syndrome, thiamine deficiency and other carbohydrate malabsorption syndromes. Medicinal and toxic causes of lactic acidosis, known as type B2, are numerous, including acetaminophen, alcohols and glycols (ethanol, ethylene glycol, methanol, propylene glycol), antiretroviral nucleoside analogs (zidovudine, didanosine, lamivudine), beta-adrenergic agents (epinephrine, ritodrine, terbutaline), biguanides (phenformin, metformin), cocaine, cyanogenic compounds (cyanide, aliphatic nitriles, nitroprusside), diethyl ether, 5-fluorouracil, halothane, iron, isoniazid, propofol,sugars and sugar alcohols (fructose, sorbitol, and xylitol),salicylates, strychnine, sulfasalazine, and valproic acid. Type B3 lactic acidosis may result in those with in born errors of the metabolism. Lactic acid exists in 2 forms, the L-lactate and D-lactate. L-lactate is the most commonly measured level, as it is the only form produced in human metabolism. Its excess represents increased anaerobic metabolism due to tissue hypoperfusion. D-lactate is a byproduct of bacterial metabolism and may accumulate in patients with short-gut syndrome or in those with a history of gastric bypass or small-bowel resection. Elevation of blood lactate are common in the patients admitted to the intensive care unit and have been associated with adverse outcomes (4 – 7).In basic terms, lactic acid is the normal endpoint of the anaerobic breakdown of glucose in the tissues. The lactate exits the cells and is transported to the liver where it is oxidized back to glucose. In the setting of decreased tissue oxygenation, lactic acid is produced as the anaerobic cycle is utilized for energy production. With a persistent oxygen debt and overwhelming of the body's buffering abilities (whether from chronic dysfunction or excessive production), lactic acidosis ensues.

**OBJECTIVES**

1. To assess the lactate level at admission in poly trauma patients.
2. To assess the possibility of prediction.
  - a. Hospital Mortality
  - b. ICU Mortality
  - c. Hospital stay based on admission and lactate levels.

**METHODOLOGY:**

This is a prospective analytical study involving all trauma patients admitted to Aarupadai vedu medical college & hospital, puducherry, and Ganga Hospital, Coimbatore. All patients coming to emergency department(trauma patients) from December 2008 to July 2010 were included in our study. When patients were admitted to the emergency department during admission the serum lactate level was measured using manual method. They also screened for Glasgow coma scale based on the vital signs and Injury severity score using standard scales.

Based on the admission lactate level the patients are divided in to two groups that is normal and abnormal serum lactate levels and they followed for Serial serum lactate level measurement was done with the interval of 6<sup>th</sup> hour, 12<sup>th</sup> hour, 28<sup>th</sup> hour and 48<sup>th</sup> hour subsequently. Patient's conditions and the serum lactate level were compared and noted. Duration of the hospital stay as well as outcome of the patient's results was also noted.

Simple statistical method like mean median chi square test , P value were calculated using SPSS statistical software method.

**RESULTS:**

From the above table we observed that the maximum number of age group patients are belong to between 20 and 29. Male patients were more when compare to the female patients.

**TABLE NO: 1 - MECHANISUM OF INJURY**

S.NO.	TYPE OF INJURY	NO.	%
1	ROAD TRAFFIC ACCIDENT	66	84.6
2	FALL FROM HEIGHT	6	7.7
3	STAB INJURY BY KNIFE	1	1.3
4	FALL OF ASBETOS SHEETS	1	1.3
5	FALL OF PILLAR	4	5

Road traffic accidents was the major mechanism of injury among the all type of mechanism of injury.

**TABLE NO: 2-INJURY BASED ON GCS SCORE**

S.NO.	SCORE RANGE	NO.	%
1	1-3	10	12.8
2	4-6	8	10.3
3	7-9	20	26
4	10-12	35	44.5
5	13-15	5	6.4

Based on the Injury GCS score the maximum number of patients belongs to the category of between 10 and 22.

**TABLE NO: 3 - INJURY BASED ON ISS SCORE**

S.NO.	SCORE RANGE	NO.	%
1	10-20	18	23
2	21-30	34	43.6
3	31-40	3	3.8
4	41-50	6	7.8
5	51-60	0	
6	61-70	0	
7	71-78	17	21.8

Injury based on ISS score the maximum number of patients were belongs to the category of between 21 and 30.

**TABLE NO: 4 - NO.OF PATIENTS DURING ADMISSION BASED ON THE SERUM LACTATE LEVELS**

S.NO.	SERUM LACTATE LEVEL	NO.	%
1	0-2	4	5.1
2	2.1-4	1	1.8
3	4.1-6	24	30.6
4	6.1-8	25	32
5	8.1-10	11	14.1
6	10.1-12	6	7.6
7	12.1<	7	8.8

Maximum number of patients during admission based on the serum lactate level was between 4.1 and 6

**TABLE NO: 5-MEAN VALUE OF SURVIVORS VS NON-SURVIVORS BASED ON THE SERUM LACTATE LEVELS ON HOURLY BASIS**

S.no.		Mean Value Of Serum Lactate Level Based On				P.value
		During Admission	After 6 Horus	After 12 Hours	After 48 Horus	
1	Non- survivors N=8	9.5	7.2	6.3	4.2	2.01
2	Survivors N=70	7.3	3.4	3.1	2.2	

Since P value is more than 0.05. It show that there is no significant of serum lactate level, between the survivors and non-survivors.

**TABLE NO: 6-MEAN VALUE OF SERUM LACTATE LEVELS BASED ON THE DURATION OF STAY**

Duration Of Stay	Mean Value Of Serum Lactate On Hourly Basis				P. Value
	During Admission	After 6 Horus	After 12 Hours	After 48 Horus	
No.of Patients Upto 1 Week = 35	8.4	5.3	4.7	1.8	1.61
No.of Patients Upto 15 Days = 22	8.6	6.2	5.1	2.8	
No.of Patients Morethan 35 Days = 13	8.7	6.3	5.8	4.2	

THE PATIENTS BASED ON THE DURATION OF STAY AT HOSPITAL IS CLASSIFIED IN 3 CATEGORIES:

- 1) UPTO 1 WEEK
- 2) UPTO 15 DAYS
- 3) MORE THAN 15 DAYS

THE MEAN VALUE ON HOURLY BASIS WAS COMPOUND HERE ALSO THERE IS NO SIGNIFICANT.

**DISCUSSION:**

In our study population the maximum number of persons belongs to the age group of 20 to 29. This age group of populations is very active.

So naturally the number of patients is more in this group of age when compare to the other age group. Similarly the male patients were more when compare to the female among the total population in our study group. Male have more exposure then the female. Road traffic accident was the major mechanism of injury in our study population. Since our hospital was located on high ways, it gave access to the more number of road traffic accidents patients to come in to the hospital. 30.6% of the patient's level of serum lactate during admission was between 4.1 to 6 which scores maximum in our study population. Only 5% of them had normal level of lactate during admission. Our study result consistent with the study result of David W.Callaway et al. They also found that 39.6% of the total population belongs to this category. In our study result shows only 10% of the total population was died due to the elevated lactate level. More recently both the presence of hyperlactatemia on admission and subsequent development of hyperlactatemia have been associated with increased survival probability (8,9). Knudson MM et al and also Tornetta et al have found that 20% of the total population have died among their elevated serum lactate level. Similarly David W.Callaway et al also found that among the elevated lactate level patients 40% of the total population have died among their study population. So our study result not consistent with the earlier study results. The mean value of serum lactate level was compared with patients duration of stay at hospitals is classified in to four categories. That is during admission, up to one week , up to fifteen days and more than fifteen days viceversa. The results indicate that though there was significant different we could observe in the mean value of the serum lactate level among the different hourly basis, P value does not shows any significant results among these value. Many studies like Houman Khosravani et al, Terry L.Shirey, Farah A.Husain et al, Jan Bakker et al, Andre Meregalli et al have already proved that there was a correlation between the serum lactate level and the patients conditions. In our study we could not get because the sample number may be one of the reason. So It is suggested that the sample number may be increased in the future study.

## REFERENCES

1. David W.Callaway, MD, Nathan I. Shapiro, MD, MPH, Michael W. Donnino, MD, Christopher Baker, MD, and Carlo L. Rosen, MD "Serum Lactate and base deficit as predictors of mortality normotensive elderly blunt trauma patients" The journal of Trauma injury, infection, and critical care.2009; 1040 – 1044.
2. Kundson MM, Lieberman J Morris JA, Cushing BM, Stubbs HA, "Mortality factors in geriatric blunt trauma patients". Arch surg.1994; 129:448 – 453.
3. Tornetta P, Mostavi H, Riina J, et al. Morbidity and mortality in elderly trauma patients. J Trauma.1999;46:702 – 706.
4. Houman Khosravani, Reza shahpori, H Thomas Stelfox, Andrew W Kirkpatrick and Kevin B Laupland " Occurrence and adverse effect on outcome of hyperlactemia in the critically ill" Critical care.2009
5. Farah A. Husain, MD, Matthew J Martin, MD., Philip S. Mullenix, MD., Scott R. Steele, MD., David C. Elliott, MD." Serum lactate and base deficit as predictors of mortality and morbidity" The American journal of Surgery 185(2003)485 – 491.
6. Jan Bakker and Alex Pinto de Lima " Increased blood lactate levels: an important warning signal in surgical practice. Critical care.2004;8(2):96 – 98.
7. Andre Meregalli, Roselaine P Oliveora and Gilnerto friendman "Occult hypoperfusion is associated with increased mortality in hemodynamically stable, high – risk, surgical patients" Critically care.2004;8(2)60 – 64.
8. Xavier M Lerverve and Iqbal Mustafa. "Lactate: a key metabolite in the intercellular metabolic interplay" Critical care.2002;6(4).284 - 285.
9. Lorenzo Paladino, Richard sinert, david Wallace, Todd Anderson, Kabir Yadav, Shahriar Zehtabchi. " The utility of base deficit and arterial lactate in differentiating major from minor injury in trauma patients with normal vital signs". Resuscitation(2008);77.363-368.