



International Journal of Surgery Science

E-ISSN: 2616-3470
P-ISSN: 2616-3462
© Surgery Science
www.surgeryscience.com
2019; 3(3): 34-37
Received: 27-05-2019
Accepted: 30-06-2019

Dr. Muffazzal Rassiwal
Consultant Pediatric Surgeon,
M.Ch., Department of General
Surgery, Shalby Hospital, Indore,
Madhya Pradesh, India

Dr. Ishant Kumar Chaurasia
Assistant Professor, Department of
General Surgery, Index Medical
College Hospital & Research
Centre, Indore, Madhya Pradesh,
India

To determine the prognosis on the serum lactate and base deficit values in Nonsurvivors of sepsis

Dr. Muffazzal Rassiwal and Dr. Ishant Kumar Chaurasia

DOI: <https://doi.org/10.33545/surgery.2019.v3.i3a.145>

Abstract

Background: The charts of 50 consecutive surgical admissions intensive care unit (ICU) All patients were admitted to the ICU by the Causality department for the identified purpose of resuscitation from shock due to trauma, Burns, or as a consequence of a major abdominal catastrophe. Vascular and cardiothoracic patients were excluded. All patients had both serial lactate and base deficit measurements determined simultaneously.

Conclusion: An analysis of observation revealed higher mean lactate levels in non survivor as compared to survivors. Also mean values in serum lactate levels in nonsurvivors did not attain normal levels by 18 hrs in trauma patients and normal levels in sepsis patients. Both lactate and base deficit values that increase serially or persist at high levels indicate ongoing worsening of patient condition, which may be unrecognized otherwise, and the need for more aggressive treatment. They also hint at the need for greater resources of the hospital and the patient.

Keywords: Prognosis, initial lactate & deficit

Introduction

Serum lactate concentration increases when its production by ischemic tissues overwhelms its elimination by the liver and kidneys. Elevated lactate levels suggest ongoing anaerobic metabolism and therefore an imbalance between oxygen delivery and tissue oxygen demands. A relationship between increasing serum lactate and mortality in shock has been demonstrated in many studies. In an experimental model of hemorrhage in dogs, *Dunham et al.*^[1] found lactic acidosis to be more predictive of mortality than conventional hemodynamic parameters such as blood pressure or cardiac output. Both absolute lactate value and time to normalization of lactate have been shown to predict outcome in human critically ill patients^[1]. *Abramson, et al.* reported on trauma patients resuscitated to supranormal values of oxygen transport^[1]. They found that the time to normalization of lactate levels was prognostic for survival. All patients who had normalized lactate levels at 24 hours survived; those patients who normalized their lactate levels between 24 and 48 hours had 25% mortality and those that did not normalize by 48 hours had an 86% mortality rate. Normalization of lactate appears to be a useful endpoint for resuscitation of compensated shock^[2, 3].

Material and Method

The charts of 50 consecutive surgical admissions to the Index Medical College Hospital & Research Centre, Indore, M.P. intensive care unit (ICU) from March 2018 to April 2019 were reviewed retrospectively. All patients were admitted to the ICU by the Causality department for the identified purpose of resuscitation from shock due to trauma, Burns, or as a consequence of a major abdominal catastrophe. Vascular and cardiothoracic patients were excluded. All patients had both serial lactate and base deficit measurements determined simultaneously. Clinical management decisions were by senior surgical residents in concert with a general surgery attending staff member and ICU attending staff in a collaborative ICU. All patients had an initial arterial lactate with calculated base deficit measured at the time of admission to the ICU. Our routine protocol is to check lactate levels and base deficit every 6 hours during the initial resuscitation, but the timing of repeat measurements was at the discretion of the managing surgical team. A normal lactate level was defined as ≤ 2 mmol/L or 9-20 mg/dl. "Normal base excess/deficit was defined as that between 2 and -2 mmol/L.

Correspondence

Dr. Ishant Kumar Chaurasia
Assistant Professor, Department of
General Surgery, Index Medical
College Hospital & Research
Centre, Indore, Madhya Pradesh,
India

Demographic, hemodynamic, laboratory and outcome data were recorded.

Initial and 18th-hour lactate and base deficit levels, as well as time to lactate clearance were compared among survivors and nonsurvivors for the entire group. Patients were also stratified into four groups based on lactate clearance time. Group 1 never achieved normal lactate levels. Group 2 normalized lactate within 12 hours. Group 3 achieved a normal lactate level between 13 and 18 hours, and group 4 took longer than 18 hours to normalize. These groups were then compared with respect to mortality, morbidity, and functional status at time of discharge. Parametric data were analyzed by Chi-square test.

Statistical significance was set at a *P* value less than 0.05.

Statistical analyses were performed using Microsoft Excel.

1. Selection of patient

- Patients presenting with Hypovolemic shock of trauma,

Burns or Gastrointestinal catastrophe.

- Patients of age above 12 years.
- Patients of suspected or overt sepsis
- A total of 50 patients were included from August 2003 to August 2005.

2. Exclusive criteria

- Patients below 12 years (i.e. Pediatric group)
- Patients with serious medical problem like hypertension, diabetes mellitus, CHD, malignancy, liver disorder etc.
- Patient of head injuries and psychiatric disorder with history of alcohol ingestion
- Known Inborn Error of Lactate metabolism.

Results

Table 1: Serum Lactate and Base Deficit in Nonsurvivors of Sepsis

S. No.	Lac	BD
1. Adm	59	-3
6 th hr	52	-5
12 th hr	46	-2
18 th hr	41	-5
5. Adm	68	-13
6 th hr	49	-12
12 th hr	35	-12
18 th hr	30	-13
12. Adm	82.1	-4.4
6 th hr	78.5	-3.3
12 th hr	72.1	-4
18 th hr	61	-6.9
17. Adm	43.4	-7.8
6 th hr	30.2	-5
12 th hr	32.2	-6
18 th hr	33.5	-7.5
24. Adm	43	-5.2
6 th hr	32.5	-4.5
12 th hr	30.9	-7.1
18 th hr	30	-6.3
37. Adm	65	-10.2
6 th hr	58	-7.6
12 th hr	49	-5.6
18 th hr	37.2	-0.5
38. Adm	65.3	-1.4
6 th hr	53.2	-2.3
12 th hr	54.2	-1.4
18 th hr	35.4	-0.3
48. Adm	65.5	-2.5
6 th hr	58.5	-0.6
12 th hr	42.2	-0.9
18 th hr	46.3	-4.1

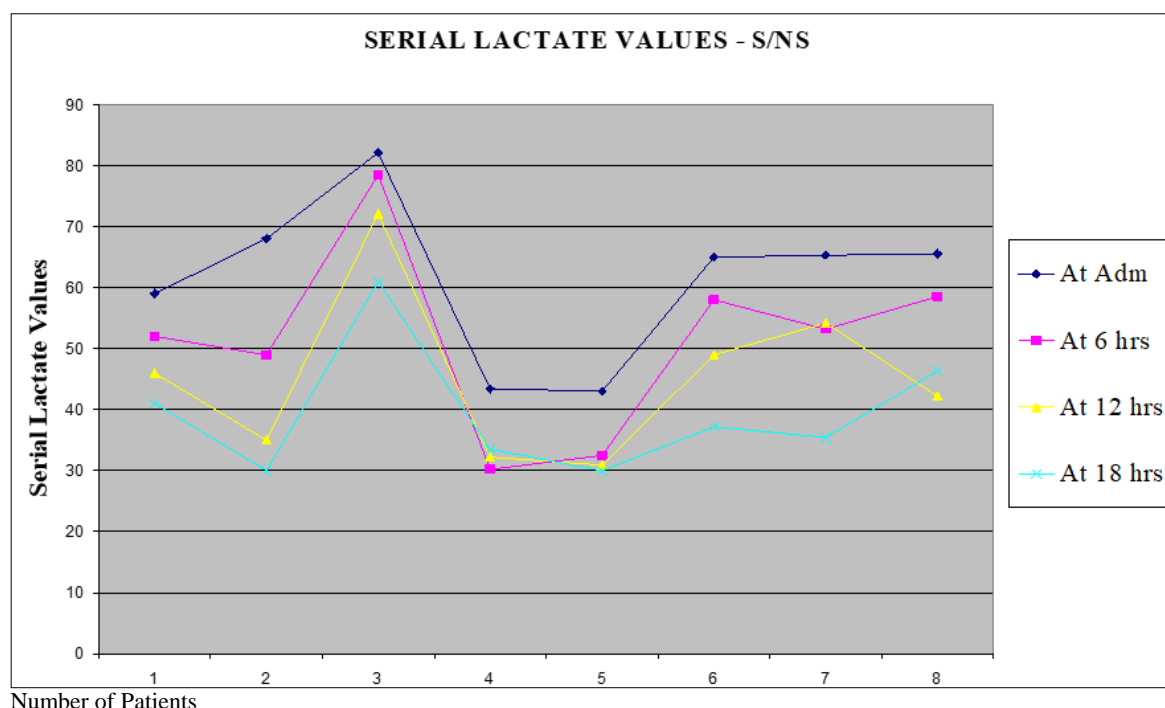


Fig 1: Serial Lactate Values in Nonsurvivors of Sepsis

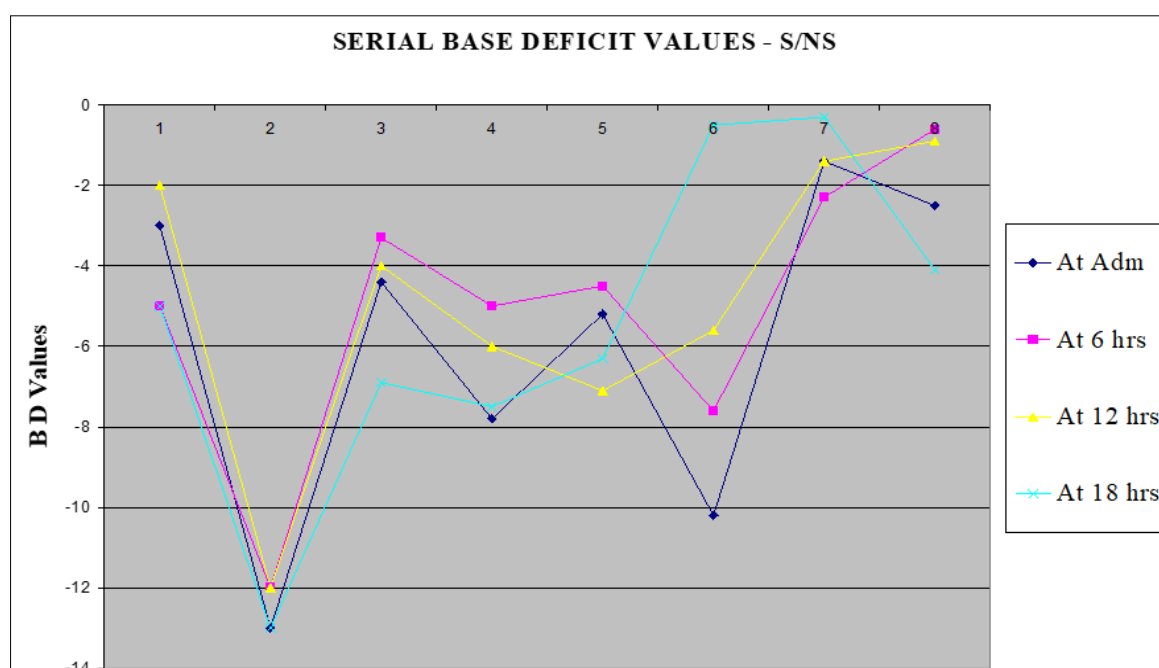


Fig 2: Serial Base Deficit Values in Nonsurvivors of Sepsis

Discussion

The base deficit is readily and rapidly obtainable and as a nearly stoichiometric relationship to serum lactate. This relationship was confirmed when *James et al.* and *Canizaro et al.* noted a decrease in base deficit associated with normalization of serum lactate levels after resuscitation [4, 5].

Our report indicates that initial serum lactate and base deficit values at admission is a reliable indicator of the relative magnitude of volume deficit. The volume of fluid required for resuscitation was greater in the groups with the more severe base deficit [6].

The results of this study indicate that lactate level is superior to base deficit as a marker for shock and resuscitation. Lactate levels on admission and after 18 hours in hospital separated survivors from non-survivors. This relationship held true for

trauma patients, nontrauma surgical ICU patients, and all patients combined, making it a useful marker for potential mortality in all types of shock encountered [7].

Conclusion

An analysis of observation revealed higher mean lactate levels in non-survivor as compared to survivors. Also mean values in serum lactate levels in non-survivors did not attain normal levels by 18 hrs in trauma patients and normal levels in sepsis patients. Both lactate and base deficit values that increase serially or persist at high levels indicate ongoing worsening of patient condition, which may be unrecognized otherwise, and the need for more aggressive treatment. They also hint at the need for greater resources of the hospital and the patient.

References

1. Dunham CM, *et al.* Critical Care Medicine. 1991; 19:231.
2. Abramson D, *et al.* J Trauma. 1993; 35:584.
3. Bikovski RN, *et al.* Current Opinion in Critical Care. 2004; 10:529.
4. James JH, Fang CH, Schrantz SJ, Hasselgren PO, Paul RJ, Fischer JE. Linkage of aerobic glycolysis to sodium-potassium transport in rat skeletal muscle. Implications for increased muscle lactate production in sepsis. J Clin Invest. 1996; 98(10):2388-97.
5. Canizaro PC, Prager MD, Shires GT. The Infusion of Ringer's Lactate Solution during Shock (Changes in Lactate, Excess Lactate, and pH). Am J Surg. 1971; 122:494-500.
6. Davis JW, Parks SN, Kaups KL, Gladen HE, *et al.* Admission Base Deficit Predicts Transfusion Requirements and Risk of Complications. J Trauma. 1996; 41:769-774.
7. Jeng JC, Lee K, Jablonski K, Jordan MH. 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.