THE SERIAL ESTIMATION OF SERUM LACTATE, PYRUVATE, AND BASE DEFICIT IN TRAUMA PATIENTS WITH HYPOVOLAEMIC SHOCK: INDICATORS OF ADEQUATE RESUSCITATION AND MANAGEMENT

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SUMMARY

The raised serum lactate and base deficit (BD) are indicators of tissue hypoxia associated with hypoperfusion that follows hypovolemia. In our study we estimated base deficit, serum lactate and pyruvate ratio of 50 trauma patients with systolic blood pressure less than 90 mm of Hg. The indices were estimated immediately at admission (i.e. 0 hours) and 6, 12, 24, 48 hours later. These patients were divided into three groups depending on the BD values at admission (group I - BD 3 to 5.9, group II - 6 to 14.9, group III-15 or less). Serum lactate and L/P ratios were 0.193±0.04 mmol.L⁻¹, 2.726±0.253 respectively in 25 patients (control group), who were not in shock and posted for routine elective surgery. The values were raised significantly in all trauma patients on admission. Among them, they were significantly higher in group II (serum lactate 0.516±0.36 mmol.L⁻¹, L/P ratio 7.72±2.49) and group III (0.66±0.134 mmol.L⁻¹, 10.99±2.16) compared to group I (0.362±0.049 mmol.L⁻¹, 5.63±1.77). There were seven deaths in the study group. On admission serum lactate levels of survivors (0.535±0.182) were comparable to those of non survivors (0.557±0.126). The values decreased to 0.340±0.119 by 24 hours of treatment in survivors, but in the non survivors the values remained unchanged (0.529±0.232). The serum lactate levels were more in patients with higher base deficit. The serial estimation of serum lactate and decline in values are useful in judging the adequacy of resuscitation and outcome of the trauma patients in hypovolaemic shock.

Keywords: Hypovolaemic shock, Tissue hypoperfusion, Resuscitation, Base deficit, Lactate clearance.

Introduction

Fluid resuscitation of the trauma patients in a state of hypovolaemic shock, has traditionally been assessed by the usual clinical parameters, such as heart rate, blood pressure, central venous pressure and urine output. However these endpoints of volume replacement could be misleading in patients with compensated shock and the tissue hypoperfusion following vasoconstriction can not be ruled out. The oxygen debt following tissue hypoperfusion leads to anaerobic metabolism, accumulation of serum lactate and metabolic acidosis. Davis JW¹ suggested that base deficit (BD) can be used as an indicator of depressed oxygen delivery for those in a state of shock and would be useful in the clinical diagnosis of compensated shock. The improvement in the BD can also assess efficacy of fluid resuscitation. Weiskopf² also postulated that lactate is nearly stochiometric to BD and an increase in BD is a valuable metabolic indicator of shock and volume deficit. Hence during resuscitation of 50 trauma patients, in hypovolaemic shock, we measured serum lactate, pyruvate and base deficit levels. We also determined the usefulness of serial estimation of these indices in the management.

Material and methods

After obtaining institutional ethical committee clearance and consent the study was conducted on 50 adult trauma patients admitted to the intensive trauma care unit (ITCU) in hypovolaemic shock (systolic blood pressure less than 90 mmHg) and 25 adult healthy patients posted for elective surgery (control group). The patients with associated medical diseases were excluded from the study. After admission to the ITCU the trauma victims were examined clinically and their injuries were evaluated. They were resuscitated with intravenous fluids (including blood and blood products) and oxygen supplementation. The surgery was performed to arrest the source of bleeding if required, fractures were stabilized and open wounds dressed.
Assessment of blood loss was based on clinical parameters, nature of injury and measured drain output during surgery and postoperatively. The rate of fluid administered was titrated to achieve adequate resuscitation. The adequacy was judged by the heart rate less than 100 min⁻¹, systolic blood pressure more than 100 mmHg, CVP 5-10 cm of H₂O and urine output more than 0.5 mlkg⁻¹hr⁻¹. Type and volume of fluids infused during the first 24 hours were recorded. The arterial blood was collected for base deficit, serum lactate and pyruvate estimation on admission to the ITCU (0 hour) and at 6, 12, 24, and 48 hours later. Blood of control group was collected one day prior to surgery.

Normal range of BD was taken as +3 to −3 mmolL⁻¹. The trauma patients were grouped according to initial BD on admission²; Group I between 3 to 5.9, group II between 6 to 14.9 and group III 15 and less. The patients were followed during their stay in the hospital to detect and treat any complications; morbidity and mortality were recorded.

The data was subjected to statistical analysis by using the student ‘t’ test.

**Results**

The age and weight were comparable in both study and control group. There were 13, 20 and 17 trauma patients in groups I, II and III respectively. We had aimed at adequate resuscitation clinically with intravenous fluid and surgery; hence in most of the patients the systolic blood pressure was maintained above 100 mmHg and the pulse rate settled to below 100 min⁻¹ within 6 hours of admission. Since the patients were divided according to admission base deficit, group I (n=13) had lowest BD of 4.06±0.60 compared to −10.11±2.40 in group II (n=20) and −16.08±0.98 for group III (n=17). Following resuscitation the BD had decreased significantly (p< 0.01) at 6 hours and subsequently in all groups (table-1). Serum lactate levels were significantly raised in all trauma patients (group I - 0.362±0.049, group II - 0.516±0.360, group III - 0.660±0.134 mmolL⁻¹) compared to control group - 0.193±0.041 mmolL⁻¹. These values were significantly (p< 0.001) higher in group II and III than group I. However serum pyruvate levels were statistically comparable between trauma and non-trauma patients. (Pyruvate levels in control 0.070±0.017 mmolL⁻¹). Hence in the trauma patients L/P ratio were also raised (table-2). The ISS score of group II (23.3±6.4) and group III (24.17±7.7) were statistically more than group I (18.23±3.9). This correlated with higher serum lactate levels in groups II and III (groups with higher base deficit) and group I at admission (group with lower base deficit). The significant decrease (p < 0.001) in the lactate levels was observed 6 hours onward in all the groups. However they did not drop to the control values even at the end of 48 hours (table-3).

The volume of blood lost and fluid administered during resuscitation is shown in table-4. Totally there were 7 deaths among the 50 trauma patients. The blood loss (3940±1455 ml) and fluid administered (13430±2218 ml) during resuscitation were statistically (p < 0.001) more in non survivors than survivors (1749±597, 7997±1248 ml respectively). On admission serum lactate levels among survivors (0.535±0.182 mmolL⁻¹) and non survivors (0.557±0.126 mmolL⁻¹) were comparable. Following treatment there was a significant (p<0.001) decline in these
values of survivors, which was not seen in non survivors (table-5). Three patients died of uncontrolled bleeding and four others developed Multi System Organ Failure (MSOF) as terminal event.

Table - 4 : Blood loss (ml) and fluid infused (ml) during 24 hours in trauma patients (n= 50).

<table>
<thead>
<tr>
<th>Group</th>
<th>Blood Loss (ml)</th>
<th>Crystalloids (ml)</th>
<th>Colloids (ml)</th>
<th>Blood (ml)</th>
<th>Total (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>1948±1056</td>
<td>4547±1591</td>
<td>773±388</td>
<td>876±449</td>
<td>6213±2122</td>
</tr>
<tr>
<td>II</td>
<td>1954±791</td>
<td>5454±1942</td>
<td>890±429</td>
<td>1185±595</td>
<td>7530±2582</td>
</tr>
<tr>
<td>III</td>
<td>2304±1320</td>
<td>5658±2389</td>
<td>997±495</td>
<td>1505±978</td>
<td>8529±3647</td>
</tr>
</tbody>
</table>

Statistical significance: ** p < 0.01

Table - 5 : Serum lactate (mmolL⁻¹) of survivors and non survivors in 48 hours.

<table>
<thead>
<tr>
<th>Time in hrs</th>
<th>Survivors (n=43)</th>
<th>Non Survivors (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 hr</td>
<td>0.53±0.182</td>
<td>0.55±0.126*</td>
</tr>
<tr>
<td>8 hrs</td>
<td>0.46±0.161**</td>
<td>0.58±0.200*</td>
</tr>
<tr>
<td>12 hrs</td>
<td>0.40±0.129**</td>
<td>0.52±0.198*</td>
</tr>
<tr>
<td>24 hrs</td>
<td>0.34±0.119**</td>
<td>0.52±0.232*</td>
</tr>
<tr>
<td>8 hrs</td>
<td>0.28±0.107**</td>
<td>0.53±0.247*</td>
</tr>
</tbody>
</table>

Statistical significance: ** p <0.001, * not significant

Discussion

Shock has been defined in many ways, and the classic definition has been “peripheral circulatory failure, resulting from a discrepancy in the size of the vascular bed and volume of the intravascular fluid”. In the Advanced Trauma Life Support manual of the American College of Surgeons shock is defined as an abnormality of the circulatory system that leads to inadequate tissue perfusion and oxygenation. The resultant oxygen debt that occurs is responsible for anaerobic metabolism and the consequent raised serum lactate levels in all the cases of shock. Clinical studies have shown that, the patients with Injury Severity Score (ISS) more than 25, systolic blood pressure less than 90 mmHg had base deficit of more than 8 meqL⁻¹ and lactate levels more than 2.5 mmolL⁻¹ at 0-12 hrs of admission. The serum lactate levels in our study were also markedly raised and there was severe base deficit in our trauma patients on admission, suggesting tissue hypoperfusion and oxygen debt (table-1,2).

In an another study by Rutherford et al,⁵ in shocked patients of less than 55 yrs of age without head injury, a BD of 15 meqL⁻¹ was found where as in those with head injury or patients more than 55 yrs, the BD was less severe ie., 8 meqL⁻¹. It was inferred that base deficit can be a reliable marker of severity of injury and also of the risk mortality. Davis James et al² in their study demonstrated that the patients with higher BD on admission had higher ISS and required greater volume of fluid for resuscitation. In our study, patients in group II and III (the group with higher BD) had higher ISS and also serum lactate levels of less than group I (the group with lower BD). This suggest that base deficit levels on admission can be correlated well with severity of injury; the inadequate tissue perfusion results in raised lactate levels. Devis James et al³ have also shown the importance of BD as a useful guide to volume resuscitation in trauma patients. In their study BD was found to decrease with resuscitation in 160 patients, at the time of second estimation and was found to increase (became more negative) in 49 patients despite fluid resuscitation; 32 patients out of 49 had ongoing haemorrhage. We also observed in our study that as resuscitation proceeded towards adequacy, BD and Lactate levels decreased gradually (table-1,3 ).

Randolph et al⁶ studied the paediatric trauma patients and demonstrated that patients with admission BD of - 5 or higher died (37%). Of the 13 patients who died, 8 never cleared their BD and died within 33±18 hrs of admission. Failure to clear BD was associated with 100% mortality. Angela et al⁴ stated that persistent high lactate level (lactate value>2.5 mmolL⁻¹) at 12 to 24 hrs of admission is a predictor of Multi System Organ Failure (MSOF) in trauma patients. Durham et al⁷ documented that lactate levels on entry to study did not correlate with the occurrence of the death but levels at 24 hrs after admission were strongly correlated with death. In their study at 24 hrs mean values were 6±3.6 mmolL⁻¹ in non-survivors and 1.9±0.7 in survivors. They were 1.5±0.5 mmolL⁻¹ in patients without MSOF and 2.6±1.9 mmolL⁻¹ for patients who developed MSOF. Abramson et al⁸ studied serial lactate levels of trauma patients for 48 hrs and documented that subsequent levels were significantly lower in the survivors compared with the non survivors. They suggested that the ability to clear lactate predicts survival of the severely injured patients. Canizaro et al⁹ also documented in their study that the lactate levels correlated quite well with the depth of shock and rapidly returned to normal after successful resuscitation. The establishment of adequate tissue perfusion stops the excess production of lactate, the existing lactate is rapidly metabolized by the liver after restoration of hepatic blood flow. However levels remained higher in
non-survivors, who were never out of shock and perfusion never improved. In our study serum lactate levels of survivors were comparable to those of non survivors on admission, but subsequently following resuscitation, there was a decline in these values of surviving patients only. They remained elevated in the non survivors suggesting continued hypoperfusion (table-5). Seven patients died in our study, three had uncontrolled bleeding, whereas in other four patients hypoperfusion, as reflected by sustained rise in lactate, probably triggered MSOF. Hence trend in lactate clearance or decline from admission values, is a better predictor of survival, onset of MSOF or death in severely injured patients, than the lone lactate levels on admission.

Thus serial estimation of base deficits, a easily available investigation, is useful in judging the tissue perfusion during resuscitation of trauma patients in hypovolaemic shock; the trend in serum lactate values can predict subsequent outcome of these patients.

References

BOOK REVIEW

CLINICAL APPLICATIONS OF ANAESTHESIA

By Aparna Dalal, M.D.


This book with the contributions from a Biomedical engineer, a Cardiologist, an Intensivist and a Cardiothoracic Surgeon is unique in many ways.

It caters to the candidates taking a University practical / Vivavoce examination. The case proformas of various systems help the candidates to get oriented to the topic. The editor of the book has exercised lot of wisdom in touching each and every corner of the subject of anaesthesiology, resuscitation, critical care and pain management. The illustrations collected from various biomedical organizations is worth appreciable and is very much useful for the readers to understand the subject. Section on ‘Radiology and Anaesthesia’ with original roentgens and CT Scan photographs are quite informative. Comprehensive account of various procedures performed by anaesthesiologists including regional nerve blocks, the anaesthetic drugs, anaesthesia instruments, ventilators etc can be used as a ready recknor by the practitioners of anaesthesia.

I can say that everything about anaesthesia and the related sciences is found under one roof in this book. The book is very neatly printed and is moderately priced.

Dr. P.F. Kotur
Editor, IJA.