



EVALUATION OF ABG PARAMETERS IN PRE-TERM NEONATES IN NICU- A SINGLE INSTITUTION STUDY

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ABSTRACT **Aim:** To investigate the relationship between common acid-base parameters and blood lactate concentrations and their prognostic importance in sick pre-term neonates. **Methods:** Seventy five serial simultaneous measurements of arterial acid-base status and serum lactate concentrations were carried out in 30 neonates admitted to NICU with indwelling arterial catheters (gestational age and birthweight, range 27-32 weeks, and 1250-2080 g, respectively). **Results:** There was a correlation that was statistically significant between arterial blood lactate and pH. ($p < 0.001$). There was no correlation between peak lactate concentration (PLC) and base excess. Lactate concentrations were insensitively indicated by a negative base excess. At all levels, elevated lactate concentrations were associated with increased mortality. In six infants, an increase in blood lactate that was clinically significant preceded the development of clinical markers of deterioration and complications. **Conclusions:** pH or base excess cannot be used as surrogate measurements for blood lactate concentration; instead, independent measurements of blood lactate concentration are required. In sick neonates, blood lactate concentrations may provide an early warning signal and vital prognostic information. In this regard, serial blood lactate measurements are superior to a single value.

KEYWORDS :

INTRODUCTION

Hypoxia and hypotension are prevalent in sick neonates, especially premature infants receiving intensive care. Monitoring tissue perfusion is crucial for early recognition of circulatory failure, initiation of appropriate treatment, and response evaluation. Changes in heart rate, blood pressure, skin perfusion, and urine output, which are typically used to detect reduced tissue perfusion in this age group, are insensitive markers because clinically significant cellular hypoxia and reduced perfusion may be present well before clinically detectable changes in cardiovascular responses. In general, the size of these infants has prevented the use of more invasive haemodynamic monitoring, such as pulmonary artery thermodilution catheters to measure cardiac output, mixed venous oxygen saturations, and oxygen delivery.

The measurement of blood lactate concentrations is another method for assessing tissue perfusion in critically ill neonates. Serial measurements of blood lactate concentrations are superior to a single measurement, not only for providing a more accurate prognosis, but also for evaluating treatment response.⁷

Despite the abundance of information regarding the importance of measuring blood lactate in ill neonates, information regarding its significance in critically ill neonates is limited. Hyperlactatemia is linked to increased mortality in premature infants with respiratory distress. It has also been reported that elevated lactate concentrations in preterm infants are early indicators of sepsis⁸ and the need for blood transfusions.⁹ In clinical practice, the presence of acidosis and, more specifically, a negative base excess is commonly assumed to indicate impaired tissue perfusion and hyperlactatemia.

This study investigates the relationship between pH and negative base excess and arterial blood lactate concentrations in preterm neonates admitted to NICU.

Methods

This was a retrospective study performed on 30 preterm neonates in the NICU. Serially 75 ABGs were performed in these neonates till the point the neonate showed clinical improvement or succumbed to the illness. On admission, all these infants underwent monitoring of vitals, and umbilical arterial line, venous line was secured. All these infants received intravenous 10% glucose infusions, with the total fluid intake varying between 100-125 ml/kg/day.

Blood samples were collected from indwelling arterial catheters at approximately 12 hourly intervals for a maximum of five serial measurements per neonate. The study was terminated early in the event of death or removal of the arterial catheter either due to complications or a substantial improvement in the infant's status.

For the purpose of this study, hyperlactatemia was defined as an arterial blood lactate concentration of greater than 4 mmol/l. The relationship between acid-base parameters (pH and base excess) and blood lactate concentrations was studied between subjects (with the mean blood lactate concentrations for each subject in both groups) and within subjects (by calculating the residuals for each measurement from the mean for that subject). Peak lactate concentrations between various groups were compared using Student's t test after logarithmic transformation. Pearson's correlation coefficients were used to define the relation between acid-base parameters and blood lactate concentrations.

RESULTS

A total of 75 simultaneous measurements of arterial acid-base parameters and blood lactate were obtained from these 30 neonates. The initial sample was obtained at mean age of 12.2 +/- 1.23 hours after birth. The mean gestational age of the neonates was 30.32 +/- 2.3 weeks.

Twenty-two neonates had hyperlactatemia, defined as arterial blood lactate concentration in excess of 4mmol/L. Mean lactate level was 4.76 +/- 1.92 mmol/L, mean base excess was a negative value of 6.2 +/- 0.63. Neither pH nor base excess were sensitive indicators of hyperlactatemia.

There was no correlation between common acid-base parameters such as pH, base excess, bicarbonate concentrations and blood lactate concentrations. Neither pH nor base excess showed any association with blood lactate concentrations within subjects.

5 infants died during their hospital stay. Raised blood lactate concentrations, which was rising serially, but neither the lowest pH nor the worst base excess was associated with increased inpatient mortality. In two of the patients, lactates were > 6 mmol/l, and in the remaining it was less than 6 but more than 4 mmol/l.

Inpatient mortality in relation to the lowest pH, worst base excess (-10 mmol/l; $P=0.734$), and peak blood lactate concentrations using Fisher's exact test.

Of 8 who never became hyperlactatemic, none died during the hospital stay. Infants showing a consistent decrease or only a modest increase in blood lactate concentrations during the study period generally did well. This was true even for those infants with initial hyperlactatemia. In 8 infants that had serially rising lactates, there was complications such as hypotension in 2 and AKI in 1.

When we compared the survivors with the non-survivors in those neonates with serially rising lactates, we found that there was a

statistically significant difference. ($p=0.002$).

DISCUSSION

The presence of metabolic acidosis (negative base excess) is often assumed to reflect inadequate tissue perfusion and oxygenation. The main purpose of our study was to examine the relationship between common acid-base parameters and blood lactate concentrations, another indicator of tissue perfusion, in critically ill neonates.⁵ In this respect changes in oxygen extraction and delivery are more sensitive markers of tissue hypoperfusion. However, these require measurement of parameters such as mixed venous oxygen saturation and cardiac output which are technically difficult in small sick neonates, and, therefore, markers of tissue hypoperfusion such as lactate must be relied on in this population.⁵

It has been suggested that in acute lactic acidosis the blood lactate concentration and base excess correlate well.¹⁰ In our study, however, we could not show any clinically relevant relation between common acid-base parameters, such as pH and negative base excess, and arterial blood lactate concentrations. These results agree with the observations in adults.¹⁴ This is an important finding for two reasons. First, the presence of negative base excess, which is generally relied on to detect inadequate tissue perfusion, cannot be used as a proxy for blood lactate concentrations. Hence studies of interventions designed to improve tissue perfusion, such as the use of volume expanders or comparative trials of various inotropic agents, should include measurement of blood lactate as a marker of tissue perfusion.

Second, although many babies in our study had their negative base excess corrected by the administration of alkali, their blood lactate continued to be high or rose. Thus, correction of negative base excess may mask the continuing reduced tissue perfusion, and reliance on this parameter alone may provide a false reassurance of cellular wellbeing. Rohim MM et al⁹ has also demonstrated a poorer outcome with children in serial rising lactates despite adequate correction of base excess.

In this study, we did find an increased inpatient mortality associated with increasing blood lactate concentrations in this selected population. In many instances, blood lactate concentrations rose before any deterioration in the infant's condition had become clinically apparent. Changes in arterial pH and/or base excess were not informative of the subsequent outcome. In this regard, measurement of blood lactate concentrations, especially serially, may give important prognostic information and an early warning signal.

The mechanism of hyperlactatemia in sepsis is different to that in RDS, being related to limited oxygen extraction resulting from maldistribution of microcirculatory flow, but these observations emphasize the value of blood lactate concentration as an indicator of a baby's wellbeing under a variety of circumstances. The association between increased lactate and mortality in our mainly preterm neonatal population complements these findings.

Recently, normal ranges have been established for capillary blood lactate concentrations in healthy preterm and term infants.^{11,12} Furthermore, capillary blood lactate concentrations differ little from arterial concentrations in ill neonates.¹³ This would allow capillary blood samples to be used extensively for blood lactate measurements in ill infants.

CONCLUSIONS

In our study we noted that pH or base excess cannot be used as surrogate measurements for blood lactate concentration; instead, independent measurements of blood lactate concentration are required, especially serial measurements. In sick neonates, blood lactate concentrations may provide an early warning signal and vital prognostic information. With this information, the management strategies can be altered and help identify the underlying mechanisms of the illness and provide appropriate care. With appropriate and timely monitoring, we can help improve outcomes in sick neonates in NICU.

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