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Brief Report

Lactate

The role of lactate to guide resuscitation- A Clinician's perspective

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Measured lactate levels have been long considered to be a marker for the critical nature of the patient undergoing resuscitation. Serial measurement of the lactate levels has been shown to improve with the patient's clinical condition. However, the lactate level should only be used as adjunct to the ensuing resuscitation whilst appreciating that there may be a delayed rise in lactate levels as the perfusion status improves. In the context of sepsis, an absolute increased level of lactate untreated has been associated with increased morbidity and mortality and forms part of the initial management of the 'Sepsis Six campaign'. In the context trauma, lactate is also noted to be a vital parameter that guides the resuscitation effort highlighting the improvement in the anaerobic metabolism as oxygen delivery is improved to the end organs. This article aims to explore some of the concerns that emergency and critical care clinicians have a raised lactate.

Keywords: Resuscitation, Lactate, Physiology, Damage-controlled resuscitation

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Introduction

''*Doctor, this patient's lactate is raised at 4mmol/l, do you want to prescribe some fluid?''*. There have been many a time when we as clinicians have had similar encounters in our clinical journey. The reaction would be to prescribe intravenous crystalloids, however with time and recent emphasis on the importance of damage control/source control resuscitation this has been realised to be fraught with errors. The approach differs if the resuscitation is medical or trauma-based. This article aims to explore some of the concerns that emergency and critical care clinicians have a raised lactate.

Lactate- the basics: The classic model is that lactic acid is the by-product that is released from anaerobic metabolism at the cellular level due to deprivation of nutrients or oxygenation with ensuing signs of tissue hypoperfusion. However, if one studies the lactate shuttle hypothesis, Brooks highlighted that this once-thought-complete waste product is produced and utilised in an aerobic environment [1,2]. The presence of a hypoxic environment/tissue hypoperfusion would lead to a raised lactate however this is not the only cause of a raised lactate [3]. Through the process of glycolysis catabolism of glucose is carried out through which two ATP and two NADH molecules are produced. During the fermentation process, the NADH that is produced is used to convert pyruvate to lactate. The process proceeds without consuming oxygen [4]. Lactate has even been regarded as an important metabolite that is instrumental for brain functioning acting as a source of energy whilst protecting the neuron from acidosis [5].

Should we be 'normalising' lactate?

There is an increasing body of evidence in resuscitation medicine that the trend in lactate is more important than standalone levels. Increasing lactate levels are likely to represent a worse overall outcome than a down-trending value in blood lactate levels [6]. Aggressively treating the lactate may improve numbers however this is at the expense of the liberal use of crystalloids which is noted to be detrimental in the context of trauma.

Lactate in the context of trauma

In the context of trauma, there is a greater emphasis on the utility point of testing for blood gas than purely basing the resuscitation on physiological Parameters [7]. Lactate, Bicarbonate and base excess serial measurements are now noted to be superior to reliance on the absolute Hb level. Worsening acidaemia in the context of rising lactate is indicative of ongoing bleeding. Amongst the parameters, lactate is noted to have the best independent predictor for 72-hour mortality [8].

Lactate in the context of medical resuscitation

In the realm of medical resuscitation, we have noted that raised lactate is commonly seen in septic patients that is considered if undertreated are at risk of developing multiorgan failure and death. In a study carried out by Chen et al, it was noted that 1400ml of crystalloid fluid corrected lactate by 1mmol/l [8]. The authors also highlighted that the lactate load is an independent risk factor for 28-day mortality in adult septic patients with ensuing shock [9]. The choice of crystalloid used in the resuscitation process needs to be given consideration and there is much debate over the choice of balanced isotonic crystalloid (e.g., Ringer's lactate) vs Normal (0.9%) saline. Although the mechanism is fully understood, there are concerns about increasing lactate if resuscitated with large volumes of normal saline. It is thought that in the presence of high plasma chloride levels, the haemoglobin moiety readily binds to H+ to form HHb which invariably worsens the lactic acidosis [10,11]. However, this warrants a separate discussion which is not part of the focus of this article, however nonetheless should still be considered during resuscitation.

It is worth noting that the lactate level may not necessarily be representative of the critical unwell the patient may be due to one of the following causes: -

1. Initial hypoperfusion state with a falsely low lactate level, with the eventual rise in lactate as the perfusion state improves

2. In the context of mesenteric ischaemia, it is noted that the rise in lactate only occurs at the later stages of the ischaemic process.

There is a greater understanding that the continued persistence of a raised lactate in the septic patient is not strictly due to the continued process of anaerobic metabolism but rather ineffective lactate clearance [12]. Targeting the resuscitation based solely on the lactate level can lead to unnecessary

Excessive use of vasopressor support and blood transfusion which when considered in the long-term has a negative impact on the patient's prognosis [13].

In the context of sepsis, the drive has been to reduce the lactate levels by more than 20% within the first 2 hours, however there is no scientific basis for this arbitrary value [14]. The inexperienced clinician needs to understand that whilst this value is important, absolute values do not necessary reflect on how human physiology changes. Overzealous resuscitation may indeed lead to significant fluid shifts that may lead to worsening clinical state with worsening metabolic acidosis. Careful early administration of vasopressors may be better tolerated in the patient at risk of fluid overload.

Conclusion

Whilst the trends in lactate levels can be considered important biochemical parameters for resuscitation, the clinical context needs to be considered as part of the underlying process. Overzealous treatment in an attempt to 'normalise' lactate can prove to be a deleterious and negative impact on the patient's outcome. There needs to be a greater understanding of the multifactorial cause for a raised lactate.

Highlights

A raised lactate may be used as an independent marker of how unwell the patient is; however, an initially low lactate may be falsely reassuring the clinician.

Lactate whilst important should be considered in the patient's clinical context.

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