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EDITORIAL COMMENTARY

Severe hyperlactatemia after cardiac surgery: Sorting out the badness

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Central Message

Early, severe hyperlactatemia may be an important biomarker in the management of the post-operative cardiac surgery patient.

See Article page 825.

The “typical” patient population that is being referred for cardiac surgery continues to evolve. The performance of complex cardiac surgical procedures in octo- and nonagenarians, once deemed borderline, is becoming commonplace.¹⁻⁶ With this changing demographic, heightened vigilance is needed in perioperative care, to preserve organ perfusion and function in these patients, who often have a greater comorbidity burden and frailty.^{7,8} These patients have increased vulnerability to perioperative complications that may result in prolonged length of stay in the intensive care unit, and increased morbidity and mortality.⁹⁻¹¹

A desirable goal, therefore, is to identify biomarkers that detect when a postoperative patient has “gone off the tracks” at the earliest time point, to permit the healthcare team to intervene prior to the point at which irreversible organ injury has occurred. Perhaps the most commonly used biomarker to determine inadequate oxygen delivery to meet the metabolic demands of the body’s tissues and organs is the measurement of serum lactate. Serial measurements of lactate kinetics to guide adequacy of therapy have been associated with alterations in mortality rates in critical illness.¹²⁻¹⁴

In patients undergoing cardiac surgery, early hyperlactatemia (<6 hours) has been associated with a >3-fold increase in negative outcomes.¹⁵ Similarly, blood lactate measurements have been proposed as a useful tool in prognostication for patients who require extracorporeal life support.^{16,17} Indeed, a previous report attempted to highlight the “indispensability” of a blood lactate-based scale to complement existing risk prediction scores.¹⁸ However, the use of this biomarker remains controversial, as alteration of glucose metabolism in acute illness may result in elevation of serum lactate levels, independent of tissue hypoxia.¹⁹ Ultimately, although many groups are very interested in the use of

serum lactate, more work is needed, particularly in cardiac surgical patients.

Renew and colleagues present their retrospective, single-center data on 9580 consecutive, elective, cardiac surgery patients from a 5-year period, with a focus on the 121 patients who developed severe hyperlactatemia (peak lactate >10 mmol/L) in the postoperative period. This value was chosen based on seminal work by Madias, demonstrating that a serum lactate level of >10 mmol/L saturates the kidney and liver mechanisms to clear lactate. Hospital mortality was 40.5% in these patients, although perhaps this rate is better than expected for patients with an average serum lactate of 15.2 mmol/L. A total of 33% of patients were discharged to home. Cardiogenic shock was the most prevalent etiology (53.8% of cases), and earlier, with better outcomes than mesenteric ischemia or extremity compartment syndrome (9% of cases), which tended to develop later and were associated with 100% mortality.

The authors should be commended for increasing the granularity of analysis of this biomarker, which is used so commonly and is so readily available, which often portends the demise of patients. As they demonstrate, etiology, timing, and levels (the peak levels were higher in the mesenteric and limb ischemia patients) immediately provide more useful information to the treating team, and ultimately to substitute decision makers, as difficult decisions have to be made about these critically ill patients. According to the data presented by Renew and colleagues, 33% of patients with a peak serum lactate >10 mmol/L survived to hospital discharge; these outcomes are better than one would expect. Of course, a retrospective analysis such as this typically raises more questions than it answers.

Based on the limitations of their methodology, the authors were unable to provide a detailed analysis that included their entire population. Similarly, as serum lactate measurement was not performed in a standardized manner in all patients, nuances of lactate kinetics could not be studied. Such capabilities would have created the potential to look at risk across the spectrum of serum levels, identify a clinical definition for severe lactatemia based on minimal or nil survival, compare matched patients without lactatemia, to assess the hazard function as an independent risk factor, and ultimately generate a postoperative risk model that incorporates serum lactate. Finally, the influence of specific agents, such as epinephrine, in their contribution via type 2 lactatemia, could be evaluated.

Nonetheless, this study highlights the need for perioperative teams around the world to engage in these analyses, to not only help characterize severity of illness and

prognosticate for patients, but also pave the way for studies on early warning, early therapy, and assessment of treatment effectiveness. Understanding the relevance of this routine measurement in our patients, as in this study, is the first step to unlocking the potential utility of this biomarker in sophisticated patient management in the period after cardiac surgery.

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