Case Report

Unusually high serum lactate in patient presenting with septic shock

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Received: 21 July 2018
Accepted: 23 August 2018

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ABSTRACT

Septic shock has high mortality and can present atypically. Serum lactate, a useful biomarker for early recognition, has direct correlation with the mortality. It simply represents organ dysfunction and its relation with mortality has been widely studied. This case of a 65 year old man highlighting the extremely high lactate level with septic shock and its correlation with the clinical condition and outcome of the patient. He presented to the Emergency department (ED) with signs and symptoms of severe sepsis with initial serum lactate of 62.4 mmol/L measured in ED. Appropriate management was started including fluids and antibiotics, but his serum lactate remained elevated his condition deteriorated, eventually leading to death. High serum lactate levels can be a useful marker to timely identify and manage patients with severe sepsis, decreasing the mortality.

Keywords: Serum lactate, Septic shock, Mortality

INTRODUCTION

Severe sepsis and septic shock are still the common presentation in today’s emergency room with unacceptably high mortality. Admissions with sepsis or septic shock increases to more than double during the last decade, from 326,000 in 2000 to 727,000 during 2008, in spite of the fact that overall hospitalizations during the same period didn’t increase.5

Sepsis and septic shock may have atypical presentation and multiple differential diagnoses can be tracked down in the emergency room settings and the physicians must be aware of the confusing and overlapping presentation of sepsis. ED physician’s effort must be geared towards a structured and early diagnosis and treatment, similarly understanding the pathophysiological process of sepsis and septic shock is an integral part of improving clinical outcomes.3,4

The initial evaluation, early recognition and management of patients with sepsis and septic shock may reduce mortality and morbidity, hence prompt institution of empiric therapy is of utmost importance in this regard.5 Aggressive fluid expansion, early broad-spectrum antibiotics as well as inotropic and vasopressors support are the mainstay of sepsis bundle management all over the world. Serum lactate is potentially a useful biomarker to risk-stratify the patients with severe sepsis; however, it is plausible that elevated serum lactate is simply a manifestation of clinically apparent organ dysfunction and/or shock (i.e., refractory hypotension).6 However, the direct correlation of the mortality with septic shock and elevated serum lactate level has been extensively reported in the literature, irrespective of hemodynamic and oxygen-derived variables.7
In the reporting center we use to see around 20% of high acuity patients and many of them have ED presentation with severe sepsis and septic shock associated with high serum lactate but in this particular case the serum lactate levels was found to be unusually high and correlated well with the clinical condition and outcome of the patient. The aim of presenting this rare case is to highlight the extremely high lactate level in patient with septic shock.

CASE REPORT

Sixty five year old retired man known diabetic presented to Emergency Department of Aga Khan University hospital, Karachi with history of fever for three days duration, and was associated with chills, he was initially managed as outpatient with antipyretics, but fever persist, six hour prior to presentation, he also started vomiting and shortness of breathing. He was hypoglycemic at home, and homemade sugar syrup was given to him.

Patient was known diabetic for the last 15 years and was on oral hypoglycemic agents (sulfonylurea), his blood sugar level remain under control. He also had right knee replacement in 2007 and was in state of functional class 2/3 since then, his weight was 98kg. He didn’t have history of liver, renal or cardiac disease. Rest of the examination was normal. Nonalcoholic and non-significant family history.

His initial vitals were: heart rate 101/min, respiratory rate 30/min, BP 100/60 mmHg, temperature 37.3°C and oxygen saturation 84% at room air which improves with 10 L/min of oxygen supplement to 94%.

On examination he was lethargic, dehydrated, tachycardiac, tachypneic and pale looking middle age gentleman. Both heart sound were normal, no murmur. Chest on auscultation having bilateral equal air entry, no added sound. Abdomen was soft, non-tender, no visceromegaly, but distended, gut sounds audible. His GCS was 12/15, E=3, M=5, V=4, was confused and drowsy yet arousable. Planter were down going, pupils: PERLA and no signs of meningeal irritation were identified.

He was managed in the resuscitation room according to international guidelines and intubated, as he was not maintaining breathing and need assistant ventilation. Hypoglycemia was corrected with intravenous 25% Dextrose water. CVP line inserted for monitoring and fluid boluses along with antibiotics, considering the diagnosis of septic shock. ABG reveals severe metabolic acidosis. Dengue antigen was also found positive.

Initial serum (arterial) lactate (preservative bottle or chemical in collecting tube and time duration from sample drawn and run) level which was send from emergency was 62.4 mmol/l, as point of care test in the ED, later was reconfirmed from the laboratory analyzer with second blood sample within an hour of initial management and was found to be the highest ever serum lactate level observed in AKUH laboratory. (What was the level?)

The patient was admitted in Intensive care unit and continuous renal replacement therapy was started along with other management. Patient required continuous vasopressor support. However his serum lactate levels remained elevated (at 6 and 12 hours and level) despite of every possible management available. His lactate clearance was also monitored.

Support was continued till the family decided for withdrawal of support, death was declared at around 1349 hours on 7th November 2013.

DISCUSSION

Daily production of lactate is about 1500 mmols i.e. 20 mmols/kg/day, which is normally metabolize in liver. Normally the production and metabolism remain balance. All bodily tissues can produce lactate under anaerobic conditions; however tissues with active glycolysis produce excess lactate. Lactate production from pyruvate via the catalytic reversible reaction of lactate dehydrogenase is shown in Figure 1.

During resting stage skin, brain and muscles all can produce 25% of lactate, followed by RBC and gut, however during strenuous exercise skeletal muscles contribute to major production. Metabolism takes place in liver (60%) and kidney (30%), partially converted to glucose and partially metabolized to carbon dioxide and water.

Excessive tissue production or impaired metabolism/excretion lead to lactic acidosis.

During sepsis and septic shock, excessive production of lactate due to anaerobic metabolism, impaired tissue perfusion and impaired excretions, all may count to lactic accumulation. Serial lactate level measurement also help in assessing response to therapy. Half-life of lactate is 20 minutes. The persistently higher level either indicates excessive production or reduces elimination coupled with anaerobic metabolism. However acidosis usually resolves within an hour once the precipitating disorder is removed. In severe cases lactate levels can rise to very high levels of up to 30 mmol/l.

The persistently higher level of lactate in patients with severe sepsis or septic shock is associated with high mortality rate. Elderly patients with septic shock are more prone to have higher mortality at the lactate level 15 mmol/l or more.
Severe sepsis and septic shock are associated with estimated 751,000 cases and around 215,000 deaths annually in US and the estimated cost incurred was on sepsis related health was 16.7B US$. Elevated serum lactate has direct correlation with higher mortality. Serum lactate is useful biomarker in early diagnosis of severe sepsis. Mikkelsen et al identified that the initial serum lactate was associated with higher mortality independent of organ dysfunction in patients presented to ED with severe sepsis and septic shock. Serum lactate above 4.0 mmol/L is associated with 27% mortality rate, however this may drop to just 7% with a level of 2.5-4.0 mmol/L, even reduce to <5% with lactate level below 2.5 mmol/L. Six hours of treatment, lactate levels reduces by 10% with dramatic reduction of death <20%. Patients whose lactate levels decrease also require less vasopressor therapy.

The results from literature from 70’s identified that 10 meq/L of lactate level was not documented in patient died with septic shock.

CONCLUSION

High serum lactate levels can be a useful marker to timely identify and manage patients with severe sepsis, decreasing the mortality.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Afzal B, Siddiqui E, Kazi SG, Khan IQ, Daniyal M, Khan MAR. Unusually high serum lactate in patient presenting with septic shock. Int J Community Med Public Health 2018;5:4613-6.