

Case Report

Lactate-buffered dialysis in cardiogenic shock associated with severe combined lactic acidosis

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Abstract

The level of lactate that would serve as cut-off for contraindication of lactate buffer is so far unclear. An acute exogenous load of lactate does not affect the basal endogenous lactate production and metabolism. It is also well metabolized in patients suffering from acute renal failure and severe sepsis with a compromised haemodynamic status. We report a case of extreme lactic acidosis in a patient admitted with a combination of cardiogenic shock, uraemia and suspected accumulation of biguanide. The patient was successfully treated with lactate-buffered dialysis due to the accidental absence of the bicarbonate-buffered fluids.

Keywords: haemodiafiltration; intensive care; lactic acidosis; metformin

Background

Available studies have not shown a substantial impact of the bicarbonate-buffered fluid on morbidity and mortality of patients on continuous renal replacement therapy (CRRT) compared to the lactate buffer [1]. The daily load of exogenous lactate on CRRT often more than doubles the normal load of endogenous lactate for the liver's intermediate metabolism. We report a case of extreme lactic acidosis in a patient admitted with a combination of cardiogenic shock, uraemia and suspected accumulation of a biguanide. The patient was successfully treated with lactate-buffered dialysis due to the accidental absence of the bicarbonate-buffered fluids.

Case report

An 82-year-old male was admitted 5 days after outpatient surgery for hernia repair. The medical history

disclosed hypertension and type 2 diabetes mellitus treated with metformin. The patient was retrieved from bradycardia and hypotension, and required intubation and adrenalin at the scene. He allegedly complained of diarrhoea during the days after surgery and chest pain over 1.5 h before receiving rescue.

On admission, the immediate transthoracic echocardiography (TTE) showed motion abnormality of the inferior wall and right ventricular hypocontractility, and ECG confirmed STEMI (ST elevation myocardial infarction) of the inferior wall. The patient was given volume substitution, continuous drip of noradrenaline and short-lasting infusion of adrenaline, atrial flutter cardioverter to sinus after amiodarone. Urgent coronary angiography and PTCA for acute myocardial infarction in the right coronary artery was completed 55 min after admission. His haemodynamic status stabilized, left bundle branch block disappeared, repeated TTE found EFLV of 35%, no mitral regurgitation, moderate pulmonary hypertension and cardiac index of 2.5 l/min/m². The patient stayed in severe metabolic acidosis with a pH of 6.8–6.9 and lactate of 14 mmol/l despite haemodynamic improvement, normal liver function tests and euglycaemia with absence of ketones. Laboratory results also revealed severe uraemia (serum urea 43 mmol/l; creatinine 950 μmol/l); urgent sonography excluded obstructive nephropathy and confirmed adequate flow in renal vessels. Continuous venovenous haemodiafiltration (CVVHDF) was commenced with heparin anticoagulation. Intensivists had to use Na-lactate-buffered dialysis (Na-lactate 40 mmol/l) due to the accidental absence of bicarbonate-buffered dialysis bags. The decision was also supported by the fact that the patient had normal liver function tests and restored cardiac output. Ringer's solution was used as haemofiltration fluid to reduce the load of lactate and was supplemented with an excess of bicarbonate. Arterial lactate peaked at 34.5 mmol/l at 14 h after admission (Figure 1); at this time pH increased over 7.25. The total volume of 8.4% bicarbonate administered above the amount adequate to haemofiltration was 2630 ml during 72 h; 2400 ml of this amount was given during the first 24 h. During ongoing correction of acidosis, the patient required

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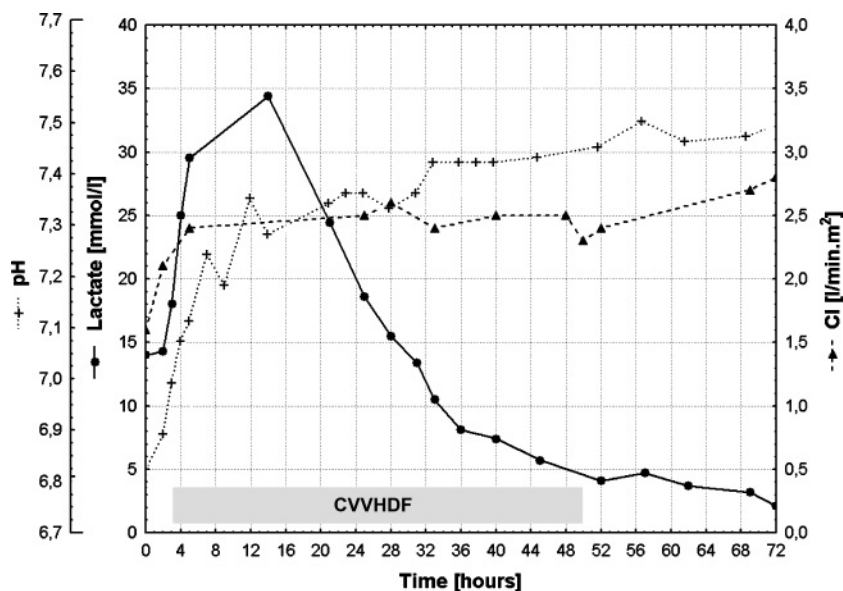


Fig. 1. The courses of arterial lactate level, arterial pH and cardiac index taken by doppler echocardiography during the first 72 h post-admission. Initial 47 h of CVVHDF are marked along the x-axis.

massive volume substitution; he also later presented with a critically low afterload that again required a high dosage of noradrenalin (0.8–1.0 $\mu\text{g}/\text{kg}/\text{min}$). His fluid balance during the first 48 h of ICU stay was +9900 ml. The arterial lactate cleared to 5 mmol/l while still being on CVVHDF with lactate dialysis. His cardiac index was gradually rising on noradrenaline and dobutamine drips towards 2.8 l/min/m² at 72 h post-admission (Figure 1). CVVHDF was interrupted after 47 h and finally ceased 101 h after admission; renal function gradually improved with no need for further renal replacement therapy. The last of multiple TTE exams showed severe hypokinesis of inferior and posterior walls, EFLV 40%, mild mitral regurgitation and moderate pulmonary hypertension. Bilateral pleural fluid was tapped 120 h after admission; the patient was extubated after 125 h. The patient was discharged to a coronary care unit on Day 8 and left the hospital for home after 28 days as NYHA II with no neurologic, renal or metabolic sequelae.

Discussion

The rather low incidence of lactic acidosis in patients receiving metformin [2] rises in patients who receive metformin despite major complications like renal insufficiency and/or cardiopulmonary compromise. The mortality in metformin-induced lactic acidosis is very high and has been estimated to be over 50% [3]. Metformin is excreted largely through kidneys and binds only negligibly to plasma proteins. Biguanides accumulated in renal failure may be effectively removed by dialysis [3,4]. So far, only a small number of metformin-induced lactic acidoses have been described in the literature [3–5].

In this case, early and aggressive treatment of the haemodynamic compromise, CVVHDF and large doses of sodium bicarbonate resulted in a successful outcome even in the

presence of severe acidosis and extremely high lactate levels. The most likely interpretation of the metabolic improvement is that lactate-buffered dialysis removed metformin and contributed to the effects of bicarbonate-buffered haemofiltration and extra high load of bicarbonate. The time to homeostasis correction was approximately equal to times described in available case studies [3–5]; however, most authors used intermittent dialysis first to remove metformin.

The authors are aware of a questionable suitability of lactate-buffered bags for dialysis in patients with severe lactic acidosis. The level of lactate that would serve as cut-off for contraindication of lactate buffer is so far unclear [6]. Available studies on lactic acidosis do not suggest that lactate removal may counteract lactate production [1]; instead, the ideal treatment of acidosis is to stop acid production by treating the underlying disorder. If the underlying disorder is adequately treated and lactate metabolism through pyruvate restored then the exogenous lactate is readily metabolized in the organism. An acute exogenous load of lactate does not affect the basal endogenous lactate production and metabolism. It is also well metabolized in patients suffering from acute renal failure and severe sepsis with a compromised haemodynamic status [7]. The load of hypertonic lactate infusion is described as safe and well tolerated in patients undergoing elective cardiac surgery [8]. Recent experimental papers [9] confirm the importance of lactate as an energetic substrate, particularly for heart metabolism in sepsis.

The prognostic importance of lactate has been vastly explored in the current literature. Lactic acidosis with preserved lactate to pyruvate ratio associates with a better prognosis compared to the disproportionate increase of the lactate level [10]. The question is thus raised whether the preserved lactate to pyruvate ratio may help a clinician to distinguish lactic acidosis with a place for lactate-buffered CRRT. Another scenario could be acidosis with the severely

increased lactate to pyruvate ratio where the input of exogenous lactate should be limited.

Conflict of interest statement. None declared.

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