Case Report

Hyperlactataemia induced by CVVHDF with low lactate bicarbonate-buffered solutions in patients with liver dysfunction

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Introduction

Critical illness is often complicated by hyperlactataemia, acute renal failure, and multi-organ failure [1]. Sodium lactate is the most commonly used nonbicarbonate buffer in both dialysate and replacement solutions and as a result, significant quantities of lactate could be transferred to the patient resulting in hyperlactataemia during continuous veno-venous haemodiafiltration (CVVHDF) [2]. Hyperlactataemia is well described during CVVHDF when the lactate load exceeds the capacity of lactate metabolism in critically ill patients [1]. The liver accounts for approximately 50% of the total lactate clearance of the body and therefore patients with liver dysfunction have a reduced capacity to metabolize lactate [1]. As such, bicarbonate-buffered solutions instead of lactate-buffered solutions are recommended in these patients [2–4]. Significant hyperlactataemia induced by the use of low lactate bicarbonate-buffered replacement and dialysate solutions during CVVHDF in critically ill patients has not been described [5]. We report on two critically ill patients, both with circulatory failure and acute liver dysfunction, who developed significant hyperlactataemia during CVVHDF with the use of low lactate bicarbonate-buffered solutions.

Case 1

A 63-year-old woman with a history of mitral stenosis, atrial fibrillation and chronic renal failure was admitted to the Royal Perth Hospital, Perth, Australia, with severe right-sided pneumonia. Her renal failure was normally treated with peritoneal dialysis and she was anti-coagulated with warfarin for the valvular heart disease. The patient deteriorated rapidly in the ward and developed severe hypoxaemic respiratory failure and septic shock requiring mechanical ventilation and noradrenaline infusion (0.1 μg/kg/min) in the intensive care unit (ICU). The liver function of the patient was deranged on admission to the ICU (bilirubin 33 μmol/l, ALT 144 U/l, ALP 303 U/l, INR 3.4). The INR remained elevated after treatment with 4 units of fresh frozen plasma, 2 vials of prothrombin complex concentrate (Prothrombinex-HT, CSL limited, Australia) [6] and 10 mg of intravenous vitamin K1. Ultrasound of the abdomen showed good hepatic blood flow to the liver and the blood ammonia concentration was mildly elevated (62 μmol/l, normal range: 10–50). Urgent tranoesophageal echocardiography showed severe mitral stenosis with a large clot adherent to the wall of the left atrium but no echocardiographic evidence of infective endocarditis.

Because the patient was normally anuric, CVVHDF was commenced immediately, through a femoral venous double lumen catheter by a blood pump at a flow rate of 180 ml/min, to facilitate fluid and electrolyte management. Bicarbonate dialysate solutions (Hemosol B0 by Hospal, Sondalo, Italy) were infused through the haemofilter, countercurrent to the blood flow, at a constant rate of 1 l/h. The replacement solutions were the same as the dialysate solutions in composition, infused continuously into the blood circuit in a prefilter fashion at a rate of 1 l/h, aiming at a neutral balance in the fluid status of the patient. Sixteen hours after the commencement of CVVHDF, the serum lactate concentration started to increase and it peaked at 8.5 mmol/l after 25 h of CVVHDF (Patient A in Figure 1). The haemodynamic status of the patients remained steady and there was no significant increase in requirement of noradrenaline infusion (0.1 μg/kg/min). Because the patient had a high risk of embolic complications due to the presence of atrial fibrillation and a large left atrial clot, the clinical diagnosis of ischaemic bowel disease was made despite her stable haemodynamic status. However, the bowel perfusion was normal and no abnormal pathology was found at the laparotomy. The serum lactate
concentration slowly returned to the normal range over a period of 8 h while the patient was not on CVVHDF after the operation. The hyperlactataemia reappeared again after 16 h of treatment with the same regimen of CVVHDF. Hyperlactataemia induced by CVVHDF was suspected and CVVHDF was withheld for another 24 h and the hyperlactataemia slowly resolved. The liver function and the cardiovascular status of the patient improved 2 days after the laparotomy and hyperlactataemia did not reappear when the same regimen of CVVHDF was recommenced again.

Case 2

A 64-year-old man with a history of hypertension was admitted to the Emergency Department of Royal Perth Hospital with a ruptured abdominal infra-renal aortic aneurysm. He was in profound hypovolaemic shock and had a short period of cardiac arrest at the Emergency Department before the emergency surgery. The patient had severe lactic acidosis during the surgery and was treated with 300 mmol of intravenous sodium bicarbonate. The patient was transferred to the ICU after emergency grafting of the ruptured abdominal aorta in the operating theatre. The patient developed signs of intra-abdominal bleeding soon after the operation and urgent re-laparotomy was required to achieve haemostasis. The patient was in circulatory failure requiring noradrenaline infusion (0.2 µg/kg/min) when he was transferred to ICU after the second operation.

The patient remained oliguric despite aggressive fluid resuscitation. There was no evidence of intra-abdominal compartment syndrome, the intra-abdominal pressure was measured at 10 cm H₂O. The liver function of the patient was deranged (bilirubin 13 µmol/l, ALT 505 U/l, ALP 51 U/l, INR 2.0) on admission to ICU. CVVHDF was commenced immediately, through a right subclavian vein double lumen catheter by a blood pump at a flow rate of 180 ml/min, to facilitate fluid and electrolyte management. Bicarbonate dialysate solutions (Hemosol B0 by Hospal, Sondalo, Italy) were infused through the haemofilter, countercurrent to the blood flow, at a constant rate of 11/h. The replacement solutions were the same as the dialysate solutions in composition, infused continuously into the blood circuit in a prefilter fashion at a rate of 11/h, aiming at a neutral balance in the fluid status of the patient. The serum lactate concentrations progressively increased over a period of 18 h during CVVHDF until it peaked at 8.6 mmol/l (Patient B in Figure 1). The dose of the noradrenaline infusion remained steady at 0.2 µg/kg/min during this period. Because there was no deterioration in the clinical status of the patient other than an elevated lactate concentration and, also with the experience of the first patient of this report, the CVVHDF regimen was CVVHD without the component of haemofiltration. The lactate concentrations slowly decreased and returned to the normal range after 22 h of CVVHD. CVVHDF was recommenced 2 days later after the liver function test had improved and the hyperlactataemia did not reappear again.

Discussion

Tissue hypoxia or ischaemia can induce hyperlactataemia and serum lactate concentrations have been used as a marker of tissue perfusion in critically ill patients [7]. Daily fluid exchanges in CVVHDF can reach more than 20–50 l, and as a result, the composition of the replacement and dialysate solutions can have a significant effect on the blood biochemistry of the patient. Lactate has a low molecular weight, and is readily dialysed or ultrafiltered [2]. However, the amount of lactate removed by CVVHDF using lactate free replacement and dialysate solutions is very small when compared with the total plasma clearance in patients with normal liver function (25 ml/min vs 1400 ml/min) [8]. It has been suggested that using lactate free dialysate and replacement solutions in CVVHDF will not mask any excessive endogenous lactate production even when there is tissue hypoxia and ischaemia [1,8]. Furthermore, in patients with liver dysfunction or failure resulting in reduced lactate
clearance, lactate accumulation does not seem to be a problem if the replacement or dialysate solutions used in CVVHDF is completely free of lactate [2]. However, some commercially available bicarbonate-buffered solutions are not completely free of lactate (www.usa-gambro.com: bicarbonatedialysate)(Table 1) [4,9,10]. The dialysate and replacement solutions used in our two patients contain 3 mmol/l of lactate. There was no report of hyperlactataemia induced by using such a low concentration of lactate bicarbonate-buffered replacement or dialysate solution during CVVHDF in critically ill patients. We observed a significant increase in lactate concentrations in both of our patients with the use of one of these low lactate bicarbonate-buffered replacement and dialysate solutions (Hemosol B0® by Hospal, Sondalo, Italy) that were used in our two patients contain 3 mmol/l of lactate. There was no report of hyperlactataemia induced by using such a low concentration of lactate bicarbonate-buffered replacement or dialysate solution during CVVHDF in critically ill patients. We observed a significant increase in lactate concentrations in both of our patients with the use of one of these low lactate bicarbonate-buffered replacement and dialysate solutions (Hemosol B0® by Hospal, Sondalo, Italy) during CVVHDF. Whether different low lactate bicarbonate-buffered solutions manufactured by other manufacturers will induce the same problem is uncertain. The markedly elevated lactate concentrations led to the clinical diagnosis of ischaemic bowel disease and a negative laparotomy in our first patient. With the experience of our first patient and also because the hyperlactataemia was not associated with any significant haemodynamic deteriorations, hyperlactataemia induced by the replacement and dialysate solutions was suspected in our second patient. This led to a prompt cessation of infusion of all exogenous lactate to the patient by removing the replacement component of the CVVHDF and resulted in resolution of the hyperlactataemia and avoided an unnecessary re-laparotomy.

The average daily lactate production in adults is approximately 1500 mmol [8]. However, in critically ill patients with acute liver failure and sepsis, there is an increased production as well as reduced clearance of lactate [11]. This explains why such patients do not tolerate the use of lactate-buffered solutions during CVVHDF because the excessive exogenous lactate load caused by the lactate-buffered replacement solutions can easily exceed the lactate metabolism or clearance by the patient. The lactate infused into the patient’s circulation through the replacement solutions in the CVVHDF seems to be the main cause of the hyperlactataemia in our patients. It is possible that even a small excessive amount of exogenous lactate in the low lactate bicarbonate-buffered replacement solutions can induce a slow accumulation of lactate when the total lactate load, from both exogenous and endogenous sources, has exceeded the capacity of lactate clearance of the patient. This scenario is most likely to occur when the patient has an increased production of lactate because of circulatory failure, and at the same time, a reduced capacity to metabolize lactate because of liver dysfunction.

In conclusion, the use of low lactate instead of no lactate bicarbonate-buffered replacement solutions in CVVHDF can potentially induce hyperlactataemia when the exogenous lactate load exceeds the lactate clearance in patients who have circulatory failure and liver dysfunction. When there is no other evidence of tissue hypoxia or deteriorations in haemodynamic status, physicians should interpret elevated serum lactate concentrations in this scenario with caution. Ceasing all exogenous source of lactate by removing the replacement component of CVVHDF or changing the replacement solutions to solutions that are completely free of lactate should be considered while investigating for other causes of hyperlactataemia.

Conflict of interest statement. None declared.

References


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