Deadly acidosis but home in 24 hours

Introduction

This article presents a case of severe alcohol-related lactic acidosis. The acidosis may have been worsened by underlying renal tubular acidosis. The patient recovered quickly with aggressive management.

Discussion

This patient had severe lactic acidosis apparently secondary to alcohol intake. Lactic acidosis is defined as a pH <7.35 and plasma lactate level >5 mmol/litre, and deemed severe if bicarbonate levels are <8 mmol/litre. Alcohol consumption increases production of lactate (Lien and Mader, 1999). Ethanol is quickly oxidized to acetaldehyde, then further oxidized to acetate generating nicotinamide adenine dinucleotide (NADH). NADH shifts the redox reaction catalysed by lactate dehydrogenase favouring conversion of pyruvate to lactate. Mortality from lactic acidosis has been correlated with lactate levels on admission to the intensive care unit (Cady et al, 1973). A lactate level of 22 mmol/litre predicts survival of less than 5%.

The acidosis was profound with little clinical effect; very few patients have been reported with a worse acidosis and most of those have died. This patient seemed to have a decreased ability for urinary acidification. He had a urinary pH of 5.0 with a blood pH of 6.73, thus he may have had coexistent renal tubular acidosis. Type 4 renal tubular acidosis is usually caused by congenital aldosterone deficiency or resistance. Aldosterone stimulates renal secretion of potassium and hydrogen and reabsorption of sodium, thus in type 4 renal tubular acidosis there is diminished distal hydrogen ion secretion and hyperkalaemia. It may be caused by the hyporeninaemic hypoaldosteronism of diabetic nephropathy, or a generalized tubular defect. In this patient urinary pH was <5.5, and serum potassium was high normal (5.1 mmol/litre), thus this may represent type 4 renal tubular acidosis.

The authors are not aware of any reports of simultaneous alcohol-related lactic acidosis and type 4 renal tubular acidosis. There is one report of D-lactate acidosis, a rare acquired metabolic disorder, occurring in a patient after small bowel resection with type 4 renal tubular acidosis (McNeil and Walmsley, 1984). It was thought that the renal tubular acidosis was transient. Hypocapnia caused by the metabolic acidosis could have affected distal tubular acidification. Experiments on dogs have demonstrated this in severe metabolic acidosis (Madias et al, 1977). Alternatively, the lactate could directly affect urinary acidification, although there is no experimental support for this. The combination of lactic acidosis and renal tubular acidosis could be coincidental.

Therapy of alcohol-related (type B) lactic acidosis is normally with fluid replacement, bicarbonate and treatment of the cause (Luft, 2001). With treatment lactate is metabolized back to bicarbonate, thus exogenous bicarbonate can cause an 'overshoot' metabolic alkalosis, as appears to have occurred with this patient.

Conclusions

Alcohol can cause severe acidosis under certain circumstances, even causing a pH of 6.73. Such an acidosis can be associated with a good prognosis. **BJHM**

Cady LD Jr, Weil MH, Afifi AA, Michaels SF, Liu VY, Shubin H (1973) Quantization severity of critical illness with special reference to blood lactate. *Crit Care Med* **1**(2): 75–80

Lien D, Mader TJ (1999) Survival from profound alcohol-related lactic acidosis. *J Emerg Med* **17**(5): 841–6

Luft FC (2001) Lactic acid update for critical care clinicians. *J Am Soc Nephrol* **12:** S15–19

Madias NE, Schwartz WB, Cohen JJ (1977) The maladaptive renal response to secondary hypocapnia during chronic HCl acidosis in the dog. *J Clin Invest* **60:** 1393–401

McNeil A, Walmsley RN (1984) A case of combined D-lactate and renal tubular acidosis. *Clin Chem* **30**(10): 1722

Case Report

A 51-year-old man drank two bottles of whisky and passed out. He woke with epigastric pain, which worsened during the day, and he vomited six times. Although normally teetotal, he binged sporadically. On presentation, he was tachycardic with a normal blood pressure, and inebriated with a Glasgow Coma Scale of 15. Urinalysis revealed a trace of ketones and pH of 5. Arterial blood gas analysis demonstrated severe metabolic acidosis with pH 6.73 (normal range (NR) = 7.35–7.45), pO_2 19.1 kPa (NR >10.6 kPa), pCO_2 2.54 kPa (NR 4.7–6 kPa), bicarbonate 4 mmol/litre (NR 22–28 mmol/litre) and lactate 22 mmol/litre (NR 0.5–1.6 mmol/litre).

He was diagnosed with severe alcohol-related lactic acidosis. Fluid resuscitation, broad-spectrum antibiotics and a proton pump inhibitor were commenced. He was given 1 litre of 1.26% sodium bicarbonate. The next morning after 7 litres of crystalloid and 75 ml of 8.4% sodium bicarbonate the patient was much improved with only mild epigastric pain. He had a pH of 7.53, bicarbonate of 27 mmol/litre and lactate of 1.7 mmol/litre. He was monitored for 24 hours and discharged.

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