Investigation vignette

A 58 Year old Man with Bleeding Oesophageal Varicies who Became Hypo-natraemic with Renal Replacement Therapy

CASE REPORT

A 58 year old man was admitted to the critical care unit in respiratory failure following an aspiration pneumonia that occurred during endoscopic banding for bleeding oesophageal varices. He had cirrhosis with portal hypertension caused by a long history of ethanol abuse. During the first three days of his admission he remained unconscious and mechanically ventilated. He was in sinus rhythm at a rate varying between 60 - 85 beats per minute, his mean arterial pressure varied between 50 and 70 mmHg on intravenous noradrenaline at 5 - 8 ug per minute and pitressin 1 U/hr. His PaO_2 varied between 65 and 92 mmHg (F_IO_2 0.5 and 8 cmH_2O PEEP).

As he remained anuric, continuous venovenous haemodiafiltration (CVVHDF) was commenced. However, over the next 3 days his arterial blood lactate level increased from 2.4 mmol/L to 12 mmol/L (Figure 1. 4.12). Following 48 hours of CVVHDF using a low lactate haemofiltration fluid his plasma lactate decreased to 2.2 mmol/L but he became hypo-natraemic (Figure 1. 6.12).

Name	Age	Sex
Mr. M. D.	58	M

	4.12	**6.12**		
Sodium	140	125	mmol/L	(135 - 145)
Potassium	4.5	3.8	mmol/L	(3.2 - 4.3)
Chloride	98	99	mmol/L	(99 - 109)
Bicarbonate	26	21	mmol/L	(21 - 32)
Glucose	5.7	6.4	mmol/L	(3.0 - 6.0)
Urea	28.4	19.3	mmol/L	(3.0 - 8.0)
Creatinine	0.348	0.212	mmol/L	(0.05 - 0.10)
Phosphate	1.05	0.94	mmol/L	(0.75 - 1.40)
Total Calcium	2.05	2.02	mmol/L	(2.00 - 2.55)
Albumin	25	24	g/L	(31 - 44)
Globulins	48	49	g/L	(21 - 49)
CK	105	92	U/L	(< 150)
ALT	60	52	U/L	(10 - 50)
AST	32	30	U/L	(10 - 40)
GGT	140	190	U/L	(< 40)
ALP	90	152	U/L	(30 - 110)
Total bilirubin	28	34	µmol/L	(4 - 20)
Amylase	56	37	U/L	(< 100)
Lactate	12.0	2.2	mmol/L	(< 2.0)

Figure 1. Plasma biochemical profiles on blood taken from the patient on lactate dialysis and 2 days later on a lactate free dialysis.

Diagnosis: Hyperlactataemia and hyponatraemia induced by dialysis solutions

The patient initially underwent CVVHDF using a dialysis solution with a lactate concentration of 45.5 mmol/L (Baxter haemofiltration replacement fluid AHB 7864, Table 1) and developed hyperlactataemia. The CVVHDF was continued using a lactate-free dialysis solution (Baxter haemofiltration replacement fluid AHK 5502, Table 1) without the addition of sodium bicarbonate and led to hyponatraemia.

Table 1 Baxter haemofiltration replacement fluid

	Lactate (AHB 7864) mmol/L	*Lactate free (AHK 5502) mmol/L*
Sodium	140	109
Potassium	1	
Lactate*	45.5	
Acetate		3.19
Chloride	100	110
Calcium	1.63	1.59
Magnesium	0.75	0.75
Glucose	10.88 (0.196%)	10 (0.18%)
Osmolality†	280	220

*ratio of L and D forms 20:1
† mOsmol/kg

Apart from nitrogenous compounds (e.g. urea, creatinine, amino acids) and other trace compounds (e.g. trace metals, vitamins), dialysate solutions usually contain electrolytes in concentrations that largely emulate extracellular fluid, although acetate or lactate have been used rather than bicarbonate because they are more stable in solution.[1] While plasma bicarbonate is removed during dialysis with bicarbonate-free solutions, the bicarbonate levels are usually maintained as both acetate and lactate are metabolised to regenerate the bicarbonate lost. Healthy individuals can metabolise up to 300 mmol/hr of acetate,[2] and 300 mmol/hr of lactate[3] although the maximum rate of metabolism is reduced in patients with liver disease, diabetes or poor peripheral perfusion.[1]

Initially acetate solutions were used.[4] Normally, the serum acetate level is less than 1 mmol/L (0.01 - 0.4 mmol/L),[5] rising to about 2 - 5 mmol/L during dialysis and increasing to 15 mmol/L or more in patients who metabolise acetate slowly.[1] In critically ill patients, the rate of acetate metabolism is reduced, and dialysis with acetate solutions has been associated with hypoxia, hypotension (due to a negative inotropic effect, chelation of calcium and peripheral vasodilation caused by acetate),[6,7] fatigue, dizziness, headache, and nausea.[8] Subsequently, lactate replaced acetate as the solution of choice in acutely ill patients requiring dialysis.

Lactate exists in two forms (D-lactate and L-lactate) and while it is assumed that the predominant racimate in intravenous and dialysis solutions is L-lactate, the composition varies widely depending on the method of preparation, with some solutions having a true racemic (1:1) mixture of each isomer.[9] In a normal 70 kg individual, the maximum rate of L-lactate metabolism is approximately 300 mmol/hr,[3] and the maximum rate of D-lactate metabolism is approximat-ely 100 mmol/hr.[10] However, as both D- and L-lactate metabolism is reduced in patients who have liver failure or lactic acidosis, and as hyperlactataemia has been reported to be associated with adverse haemodynamic effects,[11] increased protein catabolism (with increased urea generation)[12] and decreased ATP regeneration rate,[13] these solutions are not ideal in the management of the critically ill patient.

Some believe that lactate dialysis solutions are safe in acutely ill patients who do not have liver failure or lactic acidosis,[14] although others have found that compared with bicarbonate dialysis solutions, lactate solutions are associated with an increased number of cardiovascular events (e.g. hypotensive episodes) particularly in patients with previous cardiovascular disease or heart failure.[15]

As bicarbonate dialysate solutions do not cause hypoxaemia or hypotension, they are often currently suggested as the solution of choice for the critically ill patient requiring dialysis.[16] However, bicarbonate solutions do not have prolonged stability and form insoluble magnesium and calcium salts; therefore sodium bicarbonate has to be added separately to the dialysate solution just before its use.[14]

The addition of 100 mL of an 8.4% sodium bicarbonate solution (1 mmol/mL) to the 3 L bag of lactate-free solution used in our case (Baxter haemofiltration replacement fluid AHK 5502, which is packaged with a warning "this product requires the addition of sodium bicarbonate as a buffer") will provide a bicarbonate concentration of 33 mmol/L and a sodium concentration of 142 mmol/L.

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