Accidental Substitution of Acidic Concentrate for Acetate in Dialysis Fluid Concentrate: A Cause of Severe Metabolic Acidosis

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Dear Sir,

Potential errors with dialysate concentrate are numerous and may result in undetected cause of morbidity and mortality in patients undergoing haemodialysis therapy. Up to date, 5 cases of error in the haemodialysis procedure using the acidic concentrate jug, used in bicarbonate dialysis, instead of the acetate bath have been reported [1, 2]. The severity of the consequences of this mistake prompted us to report our experience with 2 cases, to simulate the error in vitro and to revise the optimal treatment.

In both patients, the symptoms began towards the first or second hour of the session, with obvious clinical deterioration. Blood chemistries (table 1) showed severe metabolic acidosis – with hyperkalaemia and hyperchloraemia – and hyponatraemia, while proper conductivity was obtained without setting off alarms. In both cases, a Hospal Monitral® N without pH meter was used (the last Monitral S series are now provided with it). In both cases, an acidic concentrate (Pa^lex, Renofundin® 802) had been used, by mistake, instead of the corresponding acetate concentrate (Renofundin 911) whose container happens to have the same volume (11 litres), same colour and coloured cap; the only difference lying in the package label.

To simulate the error in vitro, the above-mentioned machine was first put to work with acetate concentrate (Renofundin 911) and then run with two types of acidic concentrate (Renofundin 802 and 805) with a conductivity of 14.1 mS/cm. The simulation was repeated on 3 different days and 1-3 dialysate samples were taken each time. Sodium, potassium, chloride, calcium and glucose were measured with a Synchron CX3® (Beckman, USA); osmolality was measured with an Auto-Osmometer Osmostat OM-6020® (Kyoto Daiichi), Magnesium with a Clinical System® 700 (Beckman), pH with a Radiometer pH M62® (Copenhagen).

This simulation (table 2) showed that the acidic concentrate may replace the acetate without setting off alarms and with matching conductivity. The proportioning unit is capable of diluting it to the proper conductivity with a variation in its proportion: approximately 20 and 40% more concentrated than that obtained in normal conditions when Renofundin 802 and

805 are used, respectively. In acid dialysis, the dialysate Na⁺ concentration was much lower than that obtained when both acidic and bicarbonate concentrates were properly mixed and dialysate chloride concentration was much higher in acid dialysis than in haemodialysis with bicarbonate dialysate correct composition.

The two patients reported by Bruegge-meyer and Ramirez [1] in 1987 showed pH of 7.24 and 7.26, and plasma potassium of 5.6 and 5.2 mEq/l, respectively and plasma sodium concentration of 141 mEq/l in both of them. The more severe abnormalities in our cases might have been caused by the fact that these patients were dialysed with a different machine (Brueggemeyer and Ramirez used a Gambro, AK-10) or, more likely, because their patients had been dialysed for

a shorter period of time with an acidic concentrate, i.e. the replacement of the wrong liquid might have occurred near the end of the session. Huu et al. [2] in 1990 have simulated in vitro errors with acidic concentrate in a Monitral and an AK-10 machine and they obtained results very similar to ours.

We want to remark upon the large decrease in plasma potassium in patient 2 after he was redialysed. He was dialysed for 4 h with a bath containing 2.4 mEq/l of potassium (table 2). Six hours after the session, he was hospitalised. Blood analysis revealed 9.7 mEq/l plasma potassium concentration (the highest level published in this situation) without exogenous source, taking into account that the patient had suffered from nausea and vomiting. We think that the greater amount of plasma potassium was liberated from the intracellular compartment. Changes in acid-base balance exert a major influence on extrarenal potassium homeo-stasis by shift of potassium from intracellular to extracellular space as hydrogen ions move into cells to be buffered [3]. The plasma bicarbonate concentration on its own, independent of blood pH changes, also influences plasma potassium concentration. If acid is infused to reduce the bicarbonate concentration and the blood pH is maintained constant by a simultaneous reduction of the PCO₂, the plasma potassium concentration increases substantially [4]. After being redialysed with a bicarbonate bath, this patient presented a plasma potassium level of 5.5 mEq/l. Five and 12 h after haemodial-

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Table 1. Blood chemistries obtained in 2 cases after erroneous acid haemodialysis and haemodialysis with a bicarbonate dialysate of correct composition

HDa = Erroneous acid haemodialysis; HDb = haemodialysis with a bicarbonate dialysate of correct composition.

a Gas arterial blood analysis. b Gas venous blood analysis.

Renofundin 802 'A' acidic concentrate jug (11 litres)

Table 2. Simulation of haemodialysis: biochemical results of dialysate fluids with acetate concentrate and acidic concentrates

Acetate

Renofundin911

concentrate jug (11 litres)

Mixture of only the acidic component with water

Renofundin 805

'A' acidic concentrate
jug (5 litres)

Glucose, g/l

Osmolality, mosm/kg (mosm/l)

Na⁺, mEq/l

K⁺, mEq/l

Cl⁻, mEq/l

pH

Ca²⁺, mEq/l

Mg²⁺, mEq/l

Dilution, parts

concentrate: %O

2.04 ± 0.02 (2) 294 ± 8.5(297)

135 ± 0.8 (136)

2.04 ± 0.06 (2) 106 ± 0.6(105) 6.9 ± 0.08 3.4 ± 0.02(3.5)

1.75 ± 0.03(1.5)

1:34(1:34)

2.45 ± 0.04 (2) 272 ± 3.9 (225)

122 ± 0.6(100)

2.37 ± 0.05 (2)

123 ± 2.1 (105.75) 4.58 ± 0.33

3.55 ± 0.07 (3) 1.06 ± 0.04 (0.75) 1:37(1:44)

2.19 ± 0.04(1.5) 256 ± 8.1 (184)

114 ± 1.8 (80) 2.19 ± 0.04(1.5)

123 ± 2.2(86) 3.73 ± 0.07

4.8 ± 0.16(3.5) 0.88 ± 0.07(1) 1:25(1:35.8)

Results are expressed as mean ± SEM.

In brackets: data obtained from the manufacturer: for these, estimated osmolality (no osmolality) is available.

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ysis, the plasma potassium had decreased to 2.9 and 3.6 mEq/l, respectively. The change in the K⁺ concentration might be caused by the following factors: first, correction of the metabolic acidosis tends to introduce potassium into cells. Second, hyperventilation and hypocapnia often persist for as long as 24-48 h during recovery from metabolic acidosis [5]. Sustained hypocapnia is more likely to occur when the metabolic acidosis has been severe. As the persistent hypocapnia is coupled with rising blood bicarbonate concentration during recovery, blood pH can rise to frankly alkaline values. Alkalaemia is a well-known cause of hypokalaemia [6]. Third and last, the patient was redialysed with a dialysate K⁺ concentration of 1.5 mEq/l and had suffered from a negative potassium balance.

In this complication, intravenous administration of bicarbonate and haemodialysis with bicarbonate dialysate of correct composition are the appropriate therapeutic measures [7].

The delayed recovery of PO₂ during treatment of metabolic acidosis and consequences (severe delayed hypokalaemia) have important therapeutic implications. Overtreatment with bicarbonate bath should

be avoided. Attempts to correct plasma bicarbonate completely by means of rapid administration of bicarbonate solution can produce severe alkalaemia [5]. The goal of therapy should be to increase the bicarbonate concentration just up to an intermediate level between 10 and 15 mEq/l. We propose, as treatment, haemodialysis with a bicarbonate bath for a 1- to 2-hour period followed by slow 1/6 molar bicarbonate infusion during the next 24-48 h so as to gradually reach a normal pH. It might be convenient to use a dialysate fluid with higher a K⁺ concentration than normally used (1.5 or 2 mEq/l), and to closely monitor blood gas and potassium.

As demonstrated above, if acidic concentrate is used instead of acetate concentrate, the proportioning equipment will be able to dilute it to the proper conductivity. We propose that all proportioning equipments should be fitted with a pH-meter, with an alarm, especially in centres where both types of concentrate are prescribed. According to previous reports [1, 2], being aware of the problem and knowing that it exists is probably the best safeguard against human errors.

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