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The Case | Recurrent metabolic acidosis in a dialysis patient

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Table 1 | Excerpts from arterial blood gas and electrolytes at four different time points

	Before dialysis	After	24 h later	Before next dialysis
pH	7.19	7.47	7.33	7.09
$PaCO_2$ (mm Hg)	28	35	41	42
HCO_3^- (mmol/l)	10	25	21	12
Na^+ (mmol/l)	132	139	137	139
Cl^- (mmol/l)	95	101	98	102
AG (mmol/l)	27	13	16	25

Abbreviation: AG, anion gap.

The patient is a 49-year-old woman who has required dialysis for 3 years. Her renal disease was related to 30 years of anorexia nervosa.[1] Her urinary output was <100 ml/day. She was referred because of metabolic acidosis before every dialysis treatment. Her HCO_3^- was $\leqslant 10$ mmol/l and her arterial pH was <7.2. Occasionally lower values were observed. She denied any complaints and denied ingesting anything other then her prescribed medications; namely, a proton pump inhibitor, candesartan, $CaCO_3$, cinacalcet, erythropoietin, iron, and a vitamin preparation. $NaHCO_3$ tablets, 100 mmol/day, did not improve the acidosis. She was dialyzed with a bicarbonate bath and invariably left the unit with a normal serum HCO_3^-. The patient was referred to us for acidosis workup.

Before her next scheduled dialysis, her pH was 7.19, $PaCO_2$ 28 mm Hg, and HCO_3^- 10.4 mmol/l. The Na^+ level was 132, Cl^- 95, and K^+ 4.2 mmol/l. The osmolality was 287 mOsm/kg H_2O, creatinine 459 μmol/l, and lactate 0.54 mmol/l. The other laboratory values were within the normal range or commensurate with the dialysis state. As she had an anion gap of 27 mmol/l, she was examined for the presence of ethanol, ethylene glycol, and methanol, which were all found to be absent. Ketones were negative. Her salicylate level was 9.9 μg/ml. We reasoned that toluene intoxication was unlikely. Table 1 shows acid–base relevant blood gas and electrolyte results before dialysis on admission, after dialysis, on the next day, and before the next dialysis in the hospital.

What caused her acidosis?

SEE NEXT PAGE FOR ANSWERS

The Diagnosis | Tartaric acidosis

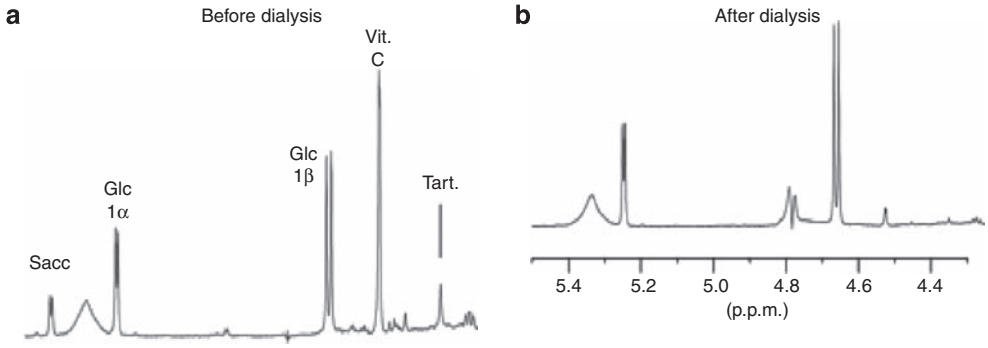

Figure 1 | Sections of the 1H nuclear magnetic resonance spectra showing the region of interest (5.5–3.5 p.p.m.) are shown. (**a**) The spectrum before dialysis shows a saccharose peak, two glucose peaks, a vitamin C peak, and a tartrate peak. (**b**) The spectrum after dialysis shows disappearance of the saccharose peak, vitamin C peak, and the tartrate peak. Glc1α, signal of proton in C-1α position; Glc1β, signal of proton in C-1β position; Sacc, saccharose/sucrose; Tart., tartrate; Vit. C, vitamin C.

We discussed the situation with the patient further. She admitted to ingesting three packages of Mentos chewable dragees, at least one tablespoon of vitamin C powder, and 10 bags of Ahoj Brause (seltzer that makes a fizzy drink) per day. The dragees were harmless and contained no notable acids. The vitamin C level in her admission serum was 500 mg/l (normal 4.6–14.9 mg/l) and might have contributed to oxalosis and renal failure, but this explained only about 1/3 of the increased anion gap.[2] Ahoj Brause is a generous source of tartaric acid and contains 1.044 g per bag. Tartaric acid provides 150 g/mol, so we calculated her daily intake at about 70 mM per day. We dissolved an Ahoj Brause bag in a glass of water; the mixture had a pH <3. After dialysis, the patient's acid–base status normalized.

We performed nuclear magnetic resonance spectroscopy in plasma before and after dialysis (Figure 1). We attributed the saccharose peak to the saccharose-containing Ahoj Brause. The glucose (Glc signals) peaks are dependent on proton positions. Thereafter came the vitamin C peak and a peak corresponding to tartrate. After dialysis, the saccharose peak, the vitamin C peak, and the tartrate peak were gone. The patient's daily tartrate load could account for the rest of the anion gap and the bulk of the metabolic acidosis.

Wine drinkers recognize tartaric acid as the source of 'wine diamonds,' the small potassium bitartrate crystals that sometimes form spontaneously on the cork. The German word is indeed, '*Weinsäure*'. The tartrate diamonds are harmless, despite sometimes being mistaken for broken glass. Yeast and bacteria metabolize tartrate with various facilities depending on the concentration of the D or L isoform. Glyceric acid is a breakdown product, but oxalo-acetic acid and glyoxylic acid are intermediates. The rate of tartrate metabolism in humans is unclear. However, renal failure interferes with tartrate elimination. Recently, the effects of dietary potassium tartrate were investigated in rats.[3] The animals excreted about half the ingested tartrate unmetabolized in the urine. Our patient could of course excrete no tartrate, which probably contributed to her clinical picture. There are no reports of tartaric acid or ascorbic acid-induced metabolic acidosis in the literature. Thus, we venture to claim the first report of Ahoj-Brause tartrate-associated metabolic acidosis, although we cannot dismiss a small contribution by vitamin C.

DISCLOSURE

All the authors declared no competing interests.

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SE, RK, and FCL managed the patient, HH identified the problem and referred the patient, ST and DL were responsible for the magnetic resonance spectroscopy, SE and FCL prepared the report.

REFERENCES

1. Luthra M, Davids MR, Shafie MA *et al.* Anorexia nervosa and chronic renal insufficiency: a prescription for disaster. *Q J Med* 2004; **97**: 167–178.
2. Nasr SH, Kashtanova Y, Levchuk V *et al.* Secondary oxalosis due to excess vitamin C intake. *Kidney Int* 2006; **70**: 1672.
3. Sabboh H, Coxam V, Horcajada MN *et al.* Effects of plant food potassium salts (citrate, galacturonate or tartrate) on acid-base status and digestive fermentations in rats. *Br J Nutr* 2007; **98**: 72–77.