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Teaching Point
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Please advise on infusing hydrochloric acid

Ralph Kettritz and Friedrich C. Luft

Department of Nephrology, Campus Virchow Klinikum, and Experimental and Clinical Research Center, Charité Medical Faculty, Berlin, Germany

Correspondence and offprint requests to: Friedrich C. Luft; E-mail: Luft@charite.de

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Introduction

The approach to and treatment of metabolic alkalosis has made great strides in recent decades and molecular mechanisms have been unraveled. Thus, we were perplexed to receive a request for assistance with hydrochloric acid management. This treatment was not that unusual in the distant past but is nowadays thankfully uncommon.

A double-take is an expression of surprise in body language. The consult request encompassed the title of this report and elicited a double-take response in both of us. We found the request a bit out of the ordinary and hastened to our intensive care unit. Sometimes strange requests turn out having a basis in fact. However, treating the underlying disorder remains the best regimen for ameliorating acid-base disturbances.

Case

The patient was a 50-year-old woman with terminal metastatic cervical cancer. The tumour had infiltrated her bladder, the ureters were obstructed bilaterally and the large bowel was involved. Both ureters had been redirected into an ileal conduit and a colostomy had been placed. The patient was somnolent and could not give a history. However, the nurses' notes indicated profuse vomiting, anuria and now a steadily increasing creatinine concentration. Her physicians were busy in the operating room. However, the chart indicated the physicians' order for two ampules (40 mmol/ampule) of hydrochloric acid to be infused in 500 mL Ringer's acetate solution through a central venous catheter. We also observed that the patient was receiving a parenteral nutritional solution (StructoKabiven®, Fresenius Kabi) supplying 1600 calories per day in a volume of 1477 mL/24 h.

Physical examination disclosed blood pressure 90/60 mmHg, heart rate 90 b.p.m. and respiratory rate 8/min. The patient could not be raised to a seated position without fainting, the neck veins were entirely flat and no hepato-jugular reflux could be elicited. The lungs were clear and there were no murmurs or extra heart sounds. She had no peripheral oedema. The sodium was 136, potassium 2.7 and chloride 80 mmol/L. The creatinine concentration was 434 µmol/L. The pH was 7.56, PaO_2 64 mm Hg, $PaCO_2$ 53 mm Hg and HCO_3 47 mmol/L. Urinary electrolytes had not been monitored and her anuria precluded our doing so.

We observed that StructoKabiven® is a remarkable product with an excellent amino acid and caloric profile. We were interested in the fact that the product she received contained 157 mmol/L of acetate. The preparation also contained sodium 60 mmol/L, potassium 45 mmol/L and chloride 52 mmol/L. We concluded that our patient had an appropriately compensated metabolic alkalosis, and so therefore recommended that the order for hydrochloric acid infusion be discontinued and made some alternative recommendations regarding her care.

Discussion

In patients with no kidney function, alkali addition gain or acid losses increase serum bicarbonate levels, and the excess alkali is retained until it is consumed eventually by endogenous acid production [1]. When kidney function is adequate, excess alkali is also excreted restoring serum bicarbonate to normal levels. Particularly important in that regard is the luminal Cl/HCO_3^- exchanger, Pendrin. Persistent alkalosis occurs only when tubular ion transport is altered in a way that limits or prevents bicarbonate excretion. Individuals receiving no chloride in the diet, subjects undergoing gastric suction or patients suffering from persistent vomiting develop metabolic alkalosis, associated with excess acid and potassium excretion into the urine. The disorder persists until chloride is given to replete the losses that were induced. The overriding role of chloride depletion, rather than volume contraction *per se*, is outlined in detail elsewhere [2]. We initially believed that the clinical picture was complete, until we reviewed the contents of StructoKabiven®.

Acetate is a derivative of acetic acid. The term includes salts and esters, as well as the anion found in solution.

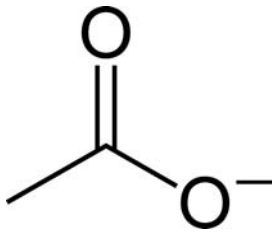

Fig. 1. Acetate ion.

The acetate anion $[CH3COO]^-$ belongs to the carboxylate family. Acetate is the conjugate base of acetic acid (Figure 1) and is metabolized to CO_2, H_2O and HCO_3^- ($CH_3COO^- + 2O_2 = CO_2 + H_2O + HCO_3^-$) at a 1:1 ratio. Similar to administration of bicarbonate, acetate can induce metabolic alkalosis. Generally, metabolic alkalosis does not occur with bicarbonate excretion because the capabilities of excreting bicarbonate are substantial. However, in patients with pre-existing chloride or potassium depletion (both were present in our patient), alkali ingestion results in a sustained increase in bicarbonate levels. Finally, in patients with renal failure, administered alkali is retained independent of body chloride or potassium stores. In our patient, the anuria also eliminated her ability to lose any bicarbonate in exchange for chloride, via her ileal conduit.

Chloride repletion is a good idea. Although strategies to deliver hydrochloric acid have been developed [3], hydrochloric acid is unnecessary. The literature is replete with therapeutic misadventures including breakdown of pulmonary artery catheters, tissue and skin necrosis, and erosion of recipient vessels [4, 5]. Normal saline, 4 L of which can increase serum hydrogen ion concentration by 10 nmol/L [6, 7], and potassium chloride sufficed in our patient, who unfortunately succumbed to her illness 10 days later. Our patient illustrated several aspects of metabolic alkalosis simultaneously and taught us several lessons, including the effects of chloride and potassium depletion on key ion transporters, the physiological role of Pendrin, the epithelial sodium channel, the H^+-ATPase and H^+/K^+-ATPase, stimulation of collecting duct ion transport and effects of alkali administration.

Teaching points

(i) An acid-base disturbance is not a diagnosis. The diagnosis lies in identifying the underlying cause of the disturbance.
(ii) Clinicians must know the contents of everything that they prescribe their patients. Trade names of products, such as StructoKabiven®, is no substitute of knowing the contents.
(iii) Hydrochloric acid has been infused therapeutically, but only clinicians over 50 years of age would be expected to know this.
(iv) An acid-base disturbance not uncommonly has more than one mechanism, as was the case in this patient.

Conflict of interest statement. None declared.

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