Conventional bicarbonate haemodialysis in postgastrocystoplasty metabolic alkalosis

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ABSTRACT

Metabolic alkalosis (MA) is an uncommon condition in chronic kidney disease (CKD) patients. The commonest cause is gastric acid loss. The normal compensatory urinary bicarbonate loss is absent in patients with CKD, and haemodialysis, being an alkalising process, is even more challenging in such a situation. We report a 44-year-old man with MA and acute-on-chronic renal failure presenting with uraemia and dehydration caused by acid loss from a previous gastrocystoplasty and obstructive uropathy. The MA was safely and quickly reversed through use of conventional haemodialysis and normal bicarbonate dialysate of 35 mmol/L. We also prevented further MA with the use of a proton pump inhibitor.

Keywords: acute-on-chronic kidney disease haemodialysis, chronic kidney disease, gastrocystoplasty, metabolic alkalosis

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INTRODUCTION

Metabolic alkalosis (MA) is an unusual phenomenon in patients with renal failure. The commonest cause is excessive loss of hydrogen and chloride ions, along with volume contraction secondary to gastric fluid loss, from profound vomiting or passive nasogastric tube drainage.[1] It is estimated that if a 60-kg man is subject to continuous gastric drainage of 2 L per day over three days, he may end up with a net bicarbonate gain of 600 mmol and an elevated serum bicarbonate of 20 mmol/L as 1 L of gastric juice may contain 100 mmol of hydrogen ions.[2] Net gain of bicarbonate in the form of citrate haemodialysis (HD), massive blood transfusion, crack cocaine use, and ingestion of pica and nonabsorbable antiacids is an even rarer cause of MA in patients with established chronic kidney disease (CKD).[3-7] The compensatory mechanism of bicarbonaturia is absent in CKD patients. Furthermore, worsening volume loss from vomiting is associated with increased tubular bicarbonate reabsorption through activation of the renin angiotensin aldosterone system. Respiratory suppression is the only compensatory response in such patients.

Severe MA in CKD calls for urgent correction of the acid-base disturbance lest hypocalcaemia-related complications like tetany, seizure and stupor ensue. Indeed, in its extreme form (pH of 7.65–7.70), the mortality rate is as high as 80%.[8] The management of MA is however challenging given that HD is an alkalising process. Hydrochloric acid infusion, acid dialysis and low bicarbonate formulation dialysis, are some techniques which have been used with mixed results.[9-11] The use of conventional bicarbonate dialysate to treat MA however, has rarely been reported.[2] We report a unique case of severe MA in a patient with acute-on-CKD resulting from increased urinary hydrogen ion loss post-gastrocystoplasty, and who had successful HD with conventional bicarbonate dialysate.

CASE REPORT

A 44-year-old Chinese man, with known CKD (baseline estimated glomerular filtration rate [eGFR] 28 ml/min/1.73m^2), was admitted to our renal unit with a history of lethargy and anorexia for a month and confusion for two days. He denied any protracted vomiting or diuretic use and was urinating 4–5 times a day. He had had disseminated tuberculosis involving the left urogenital system, associated with recurrent obstructive uropathy and secondary urinary infections, five years prior to presentation and had undergone a left nephrectomy, partial cystectomy, splenectomy, gastric antrectomy and vagotomy, and a reconstructive gastrocystoplasty and right ureteric reimplantation. His eGFR was maintained at 28 ml/min over the next three years without any evident MA until he was lost to follow-up for another two years.

At presentation, he was clinically dehydrated and restless, but alert and afebrile. His supine blood pressure (bp) was 120/76 mmHg and he had postural bp drop of 21 mmHg. Central venous pressure (CVP) read 6 cmH_2O. His right kidney was ballotable. Respiratory rate was 18/min. His laboratory results are shown in Table I. Urine electrolytes, except urine bicarbonate, were measured and are shown in Table II. Ultrasonography of the kidneys revealed right hydronephrosis.

He was given normal saline infusion (0.9%) to achieve a CVP of 10 cmH_2O and intravenous antibiotics. Emergency haemodialysis was performed using normal dialysate bicarbonate of 35 mmol/L, using a Fresenius 4008S machine (Fresenius Medical Care-Asia Pacific Pty, Singapore) with blood flow (Qb) and dialysate flow (Qd) of 180 and 500 ml/min, respectively, over two hours with zero ultrafiltration. Conventional haemodialysis successfully reversed the MA (Tables I & II), and he did not require any further dialysis subsequently. The right kidney was decompressed through a percutaneous nephrostomy the following day. He was started on a proton pump inhibitor

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Table I. Pre- and post-normal bicarbonate dialysis and post PPI changes in arterial blood gas analysis and electrolytes.

	Pre-dialysis	Six hours post-dialysis	One month after PPI
Sodium (mmol/L)	136	140	137
Potassium (mmol/L)	5.7	3.4	4.9
Chloride (mmol/L)	48	93	102
Carbon dioxide (mmol/L)	> 40	29	27
Urea (mmol/L)	63.8	43.0	14.6
Creatinine (μmol/L)	993	506	306
pH, arterial	7.52	7.40	7.38
pCO_2 (mmHg)	61.1	46.6	39.9
Bicarbonate (mmol/L)	48.8	30.3	27.8
Corrected calcium (mmol/L)	1.57	2.30	2.29
Phosphate (mmol/L)	6.40	1.78	0.92

Table II. Urine pH and electrolytes profile pre- and post-haemodialyis.

	Predialysis	> six hours post-dialysis	One month after PPI
pH	5.1	6.1	6.9
Sodium	16	21	
Potassium	24	18	
Chloride	27	14	
Osmolality	365	328	

(PPI) as well and had not suffered any more episodes of MA at follow-up one month later.

DISCUSSION

Gastrocystoplasty is a historical procedure that has been abandoned by many institutions because of its complications. The implanted gastric patch has been associated with hypochloraemic MA and the haematuria-dysuria syndrome.[12] In one retrospective series, the incidence of MA was reported at 9% over a four-year follow-up.[13] Animal studies have shown that once in the bladder, the gastric mucosa maintains its normal gastrin and acid secretory function, irrespective of vagotomy.[14,15] On the other hand, demucolisation and PPI use are associated with less aciduria.[16,17] Our patient had no other explanation for his MA, and urine pH, electrolytes and CVP suggest aciduria and volume contraction. This could have been better corroborated through histochemical studies of the bladder mucosa.

By using conventional HD with formulated dialysate bicarbonate of 35 mmol/L, a Qd of 500ml/min and Qb of 200 ml/min for two hours along with saline infusion, MA was corrected quickly and safely. The role of HD with normal bicarbonate dialysate has been described in a case series involving three patients with vomiting-induced MA, where it was shown to be convenient and without ill-effects. A lower dialysate bicarbonate of 30 mmol/L but similar Qd and Qb was used in the previous cases. Conventional bicarbonate dialysate obviates the need for central lines and reproportioning of dialysate bicarbonate seen in acid infusion and low bicarbonate dialysis respectively.[9-11] There is no need for acid dialysis, which is only possible on certain systems as described by Gerhardt et al.[10] This case also illustrates the efficacy of PPI in blocking the gastric mucosa proton pump and hence mitigating the aciduria. Kirsch BM et al recently described the usefulness of PPI in a uraemic patient with bulimia-induced MA.[17]

This is the first known reported case of gastrocystoplasty-induced MA in a patient with acute on CKD caused by obstructive uropathy, and who had a successful conventional bicarbonate dialysis for two hours and whose normal acid base status was maintained with PPI. However, this case still does not prove the absolute safety of normal dialysate bicarbonate for cases of MA and uraemia. Conventional haemodialysis should still be considered high risk in such instances, and ideally a lower Qd of 300 ml/min should be used with patient consent obtained. The use of saline infusion is of paramount importance to allow for bicarbonaturia. Concomitant ultrafiltration can also be used to offset potential fluid overload in patients with limited GFR and who are not dehydrated.

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