Renal potassium handling in a patient with surreptitious vomiting

Sir,
We read with great interest the recently published Teaching Point ‘The tell-tale urinary chloride’ [1]. While differential diagnosis of this intriguing case was properly managed, focusing on urinary chloride concentration and the coexisting metabolic alkalosis, the teaching points reported are not clearly stated.

Concerning the first point: we should note that gastric losses were unlikely to contribute significantly to hypokalaemia, since only small amounts of potassium were present in gastric fluid (about 5 to 10 mEq/L). Most of the potassium deficit was initially due to urinary losses [2].

Concerning the second point: it is mentioned that the elevated potassium excretion was due to hypovolaemia and hyperaldosteronism. However, hypovolaemia is frequently associated with a marked decrease in distal fluid delivery, thereby diminishing potassium secretion despite the hypovolaemia-induced secondary hyperaldosteronism [3]. Kaliuria is due to the coexistent metabolic alkalosis and consequently to the elevation of plasma bicarbonate and the filtered bicarbonate load. The excess filtered bicarbonate exceeds reabsorptive capacity, which cannot increase acutely. As a result, NaHCO3 and water delivery to the distal nephron exceeds reabsorptive capacity, which cannot increase acutely.

Furthermore, Alexandridis and Elisaf comment on our second teaching point; in particular, they emphasize that increased renal potassium secretion in hypovolaemia is due to hypovolaemia-induced metabolic alkalosis rather than hypovolaemia alone. In this second teaching point we solely intended, without reference to pathogenetic mechanisms, to make clear that measurement of the urinary potassium does not help here. We appreciate their comprehensive discussion of the issue.

Finally, this discussion illustrates how various mechanisms may overlap in causing electrolyte or acid–base disorders. To make matters even more complicated, their relative importance may also change considerably during the course of the disease. The predominant mechanism may hence be impossible to pinpoint in a particular patient.

Reply

Sir,
We are grateful to Alexandridis and Elisaf for their comment on our paper [1]. They first take issue with the relative importance of gastric and urinary losses of potassium in causing hypokalaemia in surreptitious vomiting. Multiple mechanisms may indeed be responsible for hypokalaemia in this setting. Direct loss of potassium from gastric secretions, increased urinary potassium secretion in response to an increased load of filtered HCO3 in hypovolaemia-induced alkalosis, and hyperaldosteronism may all be responsible [2]. This was clearly stated in the last paragraph of the Discussion while it was omitted from the Teaching Point due to constraints of space. It is important to note that the relative importance of any of these factors may change considerably during the course of the disease. It is generally believed that, in this setting, urinary losses are the predominant cause initially while direct loss of potassium from gastric secretions prevails during later stages [2]. It was therefore, we believe, impossible to gauge the relative importance of gastric and urinary losses in the patient under discussion, more so as we were unaware of the precise frequency and duration of her vomiting.

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2. Rose BD. Clinical Physiology of Acid-base and Electrolyte Disorders. McGraw-Hill, New York, 1994; 796

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