

Metabolic alkalosis in patients with distal renal tubular acidosis

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To the Editors:

I read with great interest the article in your journal by Lucaccioni et al. entitled “Severe metabolic alkalosis due to diuretic treatment in a patient with distal renal tubular acidosis: a rare association” (1). The authors reported a neonate with distal renal tubular acidosis (dRTA) who presented with hyperchloremic metabolic alkalosis after hydrochlorothiazide (HCT) treatment.

Although extremely rare, 2 infants with dRTA who developed metabolic alkalosis have previously been reported (2, 3). McSherry reported a 4-month-old infant with dRTA who developed hypochloremic metabolic alkalosis and severe hypokalemia following HCT treatment (2). Hymes et al reported a 7-month-old infant with dRTA who presented with hypochloremic metabolic alkalosis and hypokalemia after a 4-day history of vomiting, without HCT treatment (3). The authors of both reports suggested that severe metabolic alkalosis in their patients appeared related to hypokalemia and extracellular volume depletion.

Metabolic alkalosis can be caused by potassium depletion and/or chloride depletion due to gastric fluid loss or the chloruretic effect of a diuretic (4). The patient in the report by Lucaccioni et al. presented with hypokalemia, hypochloremia, and dehydration following HCT treatment and vomiting, leading to severe

metabolic alkalosis (1). Patients with dRTA who have hypokalemia and are treated by a thiazide diuretic and/or have persistent vomiting episodes should be need to be caution for the onset of metabolic alkalosis.

References

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