



Metabolic alkalosis in peritoneal dialysis — beyond the obvious: Questions

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A 15-year-old African American male with a history of stage 5 chronic kidney disease secondary to focal segmental glomerulosclerosis (FSGS) and bilateral nephrectomy on peritoneal dialysis (PD) presented with a generalized tonic–clonic seizure. He was evaluated in the emergency room where his vitals showed a heart rate of 105, blood pressure of 142/104 mmHg, temperature of 37.6 °C, and O₂ saturation of 89%. On exam, he was agitated, confused, and oriented only to person and place. His neurologic examination was otherwise normal. Initial labs showed sodium 152 mmol/L (normal 136–145), potassium 3.7 mmol/L (normal 3.5–5.1), CO₂ > 44 mmol/L (normal 22–33), and chloride 77 mmol/L (normal 98–107). Arterial blood gas showed pH 7.64 (normal 7.35–7.45), CO₂ concentration of 60.6 mmHg (normal 35–45), HCO₃ 67.7 mmol/L, and base excess of 38.5 mmol/L (normal –2 to 2). After additional investigation, he was diagnosed with encephalitis due to COVID-19 infection.

History was carefully reviewed due to significant alkalemia. No history of emesis, or recent prescription of antacids, or bicarbonate supplements. He was prescribed sevelamer carbonate for phosphorus binding but denied taking calcium carbonate for heartburn or as a phosphate binder. Home PD was completed using continuous cycler-assisted peritoneal dialysis (CCPD) of 12 cycles using standard Dianeal PD-2 peritoneal dialysis solution (lactate

40 mmol/L). Upon admission, the patient received 2 doses of arginine chloride for metabolic alkalosis and severe hypochloremia, and nightly PD was resumed per his home prescription. Sevelamer carbonate was switched to sevelamer hydrochloride. Blood pH, carbon dioxide, and bicarbonate normalized after 48 h, with bicarbonate of 21 mmol/L at the time of discharge.

The patient was readmitted for peritonitis 2 days after discharge. Serum bicarbonate was normal during this hospitalization, ranging from 20 to 28 mmol/L. At the follow-up appointment 1 week after discharge, labs showed bicarbonate of 46 mmol/L.

Questions

1. What acid–base disturbances lead to elevated serum bicarbonate?
2. What are the causes of metabolic alkalosis?
3. Which of these apply to patients on dialysis?

The answers to these questions can be found at <http://dx.doi.org/10.1007/s00467-022-05498-1>

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Declarations

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