Solutions to *e*AJKD's Test Your Knowledge: Acid-Base Disturbances in the Hemodialysis Patient

1. B. Hypercapnea

In patients with ESRD, the kidney does not play a role in acid-base homeostasis. Chronic hypercapnea does not produce a secondary elevation in serum bicarbonate level since this compensatory response requires kidney function. In the absence of kidney function, metabolic alkalosis can occur from either exogenous administration of alkali or loss of HCL from the gastrointestinal tract (vomiting, nasogastric suction). Malnourished dialysis patients with poor protein intake and low muscle mass have low endogenous acid production resulting in metabolic alkalosis. Packed red cells and fresh frozen plasma are anticoagulated with citrate, which generates bicarbonate upon metabolism. Crack cocaine is prepared with an alkali base, and heavy abuse in the setting or poor kidney function can produce a metabolic alkalosis.

2. C. ≥22 mEq/L

The KDOQI Guidelines recommend checking the serum bicarbonate levels at least monthly in dialysis patients, with goal level ≥ 22mEq/L (>20mEq/L in patients <2 years of age). Based on limited evidence, it is the opinion of experts that correction of metabolic acidosis is required for bone health and maintenance of linear growth in children.

3. C. Bicarbonate and citrate

Historically, acetate was used as the principal dialysate buffer, but this was stopped due to cardiovascular complications. Currently, bicarbonate constitutes the predominant buffer with concentrations in standard hemodialysis solution being 33 to 35 mEq/L. Lower concentrations of citrate in dialysate also act as buffers as each citrate molecule is metabolized into 3 bicarbonate molecules. Chloride and glucose do not have any buffering properties.

4. D. Fluid restriction and low-sodium diet to decrease inter-dialytic weight gain.

For most hemodialysis patients, pre-dialysis serum bicarbonate levels range between 19 and 22 mEq/L after the long inter-dialytic interval. Studies have shown that low pre-dialysis serum bicarbonate levels are independently associated with mortality, although the cut-off levels are variable. Continuing the current prescription would be inappropriate

since persistent metabolic acidosis warrants further investigation. In this patient, the post-dialysis serum bicarbonate levels reflect adequate alkali delivery with hemodialysis. A protein catabolic rate of 1 g/kg/day excludes excessive dietary protein intake as a source of endogenous acid. The next step would be to minimize inter-dialytic weight gain, since large fluid intake between treatments can lower serum bicarbonate levels by up to 3 mEq/L. Raising the dialysate bicarbonate level would be appropriate only after ruling out reversible causes.

5. A. Assess normalized protein catabolic rate

In this elderly dialysis patient, the absence of any active symptoms rules out obvious causes like vomiting. In view of his recent severe illness, malnutrition is a potential cause of his chronic metabolic alkalosis. An assessment of the normalized protein catabolic rate (nPCR) would give an estimate of his dietary protein intake and nutritional status. Checking arterial blood gases would not be useful because hypercapnea is unlikely to cause secondary metabolic alkalosis in this patient with ESRD. It would be inappropriate to ignore his chronic alkalosis, as elevated serum bicarbonate levels have been associated with increased mortality. Reducing the dialysate bicarbonate concentration is recommended only when alkalosis is severe and acute, i.e. bicarbonate levels >40 mEq/L.