Severe Hyperkalemia in Nephrotic Syndrome

With the exception of dilutional hyponatremia, electrolyte abnormalities are uncommon in children with nephrotic syndrome (NS)(1). With profound hypoalbuminemia leading to intravascular volume depletion, there is upregulation of the renin-angiotensin-aldosterone axis leading to avid proximal tubular sodium reabsorption(2) and stimulation of antidiuretic hormone. Abnormalities in serum potassium are not reported to occur if glomerular filtration (GFR) is preserved. We recently encountered a 4-year old Caucasian girl with new onset of NS. Her serum creatinine at diagnosis was normal (0.4 mg/dL) and so were her electrolytes; screening tests for secondary causes of NS were also normal (normal complement C3 and C4, and negative hepatitis B and C serologies), with the exception of a positive anti-nuclear antibody screen. The child had been started on steroid therapy before the test results were back. She returned a week later with worsening edema and a weight gain of 1.3 kg. Blood tests were obtained to rule out systemic lupus erythematosus; at the same time, an electrolyte panel was repeated. This showed that the patient was hyponatremic (sodium 127 mEq/L) and had significant hyperkalemia (potassium 7.2 mEq/L). Her serum creatinine was elevated at 0.7 mg/dL, corresponding to a calculated GFR of 83 mL/min/1.73 m^2 surface area, while her blood urea nitrogen (BUN) was 76 mg/dL(3). She was only mildly acidotic with a serum bicarbonate of 21(normal 24-32) mEq/l. She received oral loop diuretics, resulting in a drop in her potassium to 6.6 mEq/L the next day. She was subsequently admitted for intravenous albumin and furosemide and with that normalized all her electrolytes. Her BUN fell to 27 mg/dL but her serum bicarbonate remained depressed at 22 mEq/l. Hyperkalemia has never been reported before as a complication of a severe hypovolemic state in children or adults with NS. My hypothesis is that, in spite of secondary hyperaldosteronism from volume depletion, due to the poor sodium delivery to the distal tubule, potassium excretion was impaired even though her GFR was well preserved. This also explains her prompt response to loop diuretics, which increase the delivery of sodium to the collecting duct allowing potassium excretion. The purpose of this letter is to make health care professionals more aware of this potentially life-threatening complication of NS, which should be monitored for, in patients with NS who are becoming progressively more edematous.

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