Hyperkalemia in Children With Nephrotic Syndrome

IJKD 2013;7:326-34
www.ijkd.org

Dear Editor,

Serum electrolytes are usually within the normal range except dilutional hyponatremia in nephrotic syndrome (NS). Abnormalities in serum potassium are rarely reported to occur if glomerular filtration is preserved. Herein, we report 2 cases of severe edematous relapsing NS with hyperkalemia.

Case 1 is a 9-year-old boy with steroid-dependent NS that was admitted due to generalized severe edema. His follow-up period was 2 years with nonspecific renal biopsy findings and he had a negative family history for kidney diseases. On physical examination, he had generalized edema with respiratory distress. Laboratory values were as follows: serum creatinine, 0.3 mg/dL; blood urea nitrogen, 19.3 mg/dL; serum albumin, 1.2 g/dL; serum sodium, 126 mEq/dL; serum potassium, 7.4 mEq/dL; serum triglyceride, 195 mg/dL; serum cholesterol, 322 mg/dL; and urine protein, 58 mg/m^2/h.

Case 2 is a 3-year-old boy with frequently relapsing NS who was hospitalized with severe generalized edema and ascites. Laboratory findings were as follows: serum creatinine 0.05 mg/dL; blood urea nitrogen, 17.7 mg/dL; serum albumin 1.1 g/dL; serum sodium 121 mEq/dL; serum potassium, 7.1 mEq/dL; serum triglyceride 370 mg/dL; serum cholesterol 338 mg/dL; and urine protein, 60 mg/m^2/h.

Both patients were treated with nebulized salbutamol due to hyperkalemia as a first step of therapy. However, serum potassium levels have been slightly decreased with this therapy (6.7 mEq/L and 6.5 mEq/L, respectively). When albumin infusion with furosemide treatment was started, the serum potassium levels normalized in our patients. Pulse-methyl-prednisolone (30 mg/kg/d) treatment was also started. During the follow-up period, this treatment protocol induced prompt remission with resolution of clinical symptoms and normalization of other laboratory findings such as serum albumin levels and proteinuria. To our knowledge, serum potassium levels in nephrotic patients have not been rigorously documented, thus, mechanisms of hyperkalemia in NS are not well known. The possible mechanisms of hyperkalemia include: (a) potassium excretion was impaired due to the poor sodium delivery to the distal tubule[1]; (b) increased renal outer medullary potassium channel (ROMK) clathrin-dependent endositosis due to with-no-lysine-kinase-4 (WNK-4), which inhibits ROMK by hypovolemia in spite of secondary hyperaldosteronism in NS[2]; and (c) downregulation of ROMK stems from phosphorylation of extracellular signal-regulated protein kinase arising from the presence of proteins in the luminal fluid in NS.[2] Our experience suggests that correction of hypovolemia seems to be the mainstay treatment of hyperkalemia in patients with NS. This also might explain patients' response to volume expansion with albumin, which induces inhibition or downregulation of ROMK by hypovolemia or proteinuria. Diuretic treatment can increase hypovolemia, so furosemide should not be used in case of hyperkalemia as first choice of treatment. The reduction of potassium levels with nebulized salbutamol seems to be temporarily. We concluded that health professionals should be monitored serum potassium levels in patients with severe edematous NS due to potentially life-threatening complication of hyperkalemia.

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