Correspondence

Treatment of nephrogenic diabetes insipidus due to hypervitaminosis D

To the Editor

We read the interesting report of hypervitaminosis D causing nephrogenic diabetes insipidus (NDI) in a 5-month-old infant published recently in Saudi Medical Journal.[1] In this report, the authors describe the management of an infant with NDI due to hypervitaminosis D. The cause of NDI in hypervitaminosis D is hypercalcemia. Two underlying mechanisms leading to NDI have been proposed. Calcium sensing receptor (CaSR) expressed on the basolateral (blood) side of the thick ascending limb cells indirectly inhibits the NKCC2 co-transporter BSC1 and impairs the generation of a medullary concentration gradient. Secondly, this receptor is also expressed on the luminal side of the collecting duct cells, and decreases aquaporin-2 expression on apical membrane.[2] The management of acquired NDI primarily consists of identifying and correcting the underlying disorder.[2] Hydration with normal saline, loop diuretics and glucocorticoids is the initial management of hypercalcemia.[3] The authors have used hydro-chlorthiazide for the management of NDI in this patient. Thiazides itself cause hypercalcemia and should not be used in the management of NDI when the underlying cause is hypercalcemia. Thiazides acts on NaCl co-transporter in distal convoluted tubule promoting excretion of NaCl in the urine and ultimately decrease effective intravascular volume which leads to increased passive proximal Ca absorption secondary to increase in proximal sodium and water re-absorption.[4]

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Reply from the Author

Thank you for your nice comment regarding our case report: Thiazide diuretics decrease distal convoluted tubule reabsorption of sodium and water, thereby causing diuresis. This decreases plasma volume, thus lowering GFR and enhancing the absorption of sodium and water in the proximal nephron. Less fluid reaches the distal nephron, so overall fluid conservation is obtained. We were concentrating in treating hypernatremia as it is hazardous to the brain and may cause disruption of capillaries and may lead to intravetricular hemorrhage and this what happened as sodium decreased dramatically with the use of thiazides and hydration without any increase in calcium or worsening of hypercalcemia. Meanwhile, we were forced to decrease the dose of thiazides during course of treatment as patient developed low calcium and sodium. All corticosteroid drugs, including prednisone, can cause sodium retention, resulting in dose-related fluid retention. Corticosteroid-induced fluid retention can be severe enough to cause hypertension. The principal mechanism of corticosteroid-induced hypertension is the overstimulation of the mineralocorticoid receptor, resulting in sodium retention in the kidney. This results in volume expansion and a subsequent increase in blood pressure. For this reason, we did not use steroids because of the presence of hypernatremia and fear of hypertension and hyperglycemia which may worsen polyuria and dehydration which will worsen hypernatremia.

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