A Case of Resolving Acquired Nephrogenic Diabetes Insipidus after Parathyroidectomy

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Nephrogenic diabetes insipidus (DI) is caused by partial or complete renal resistance to the effects of antidiuretic hormone. Hypercalcemia can cause nephrogenic DI, but extreme hypercalcemia usually is distinctive for malignancy, and rarely seen in primary hyperparathyroidism. Here, we present a case of acquired nephrogenic DI in a patient with benign parathyroid tumor. A 48-years-old male with diabetes mellitus visited the emergency department because of nausea and vomiting for 4 days. He suffered frequent urination and excessive thirst, and his daily urine output was 9 L. Laboratory values showed serum creatinine 1.39 mg/dl, random glucose 248 mg/dL, corrected calcium 18.4 mg/dL, phosphorous 2.7 mg/dL, and intact parathyroid hormone 1283 pg/mL. Emergent hemodialysis using low calcium dialysate was underwent and large volume saline was given with intravenous furosemide and bisphosphonate. Parathyroid ultrasound revealed 5.9 x 2.6 x 2.4 cm sized ovoid nodule in right parathyroid gland. Although serum calcium and other electrolyte values returned to near normal, he still presented polydipsia and polyuria. He was performed water deprivation test, and diagnosed partial nephrogenic DI. Pituitary mass was not found in bran magnetic resonance imaging, and genetic mutation in the aquaporin-2 (AQP2) and arginine vasopressin (AVP) gene was not detected. Right parathyroidectomy was conducted, and histopathologic finding was parathyroid adenoma. Two weeks after the parathyroidectomy, daily urine output decreased to less than 2 L/day. This is a rare case of nephrogenic DI caused by parathyroid adenoma and corrected by surgical resection. Hypercalcemia is a well-known but overlooked cause of polyuria. The polyuria caused by impaired concentrating ability of the renal tubule was not improved after the correction of the hypercalcemia through medical treatment and hemodialysis, but rapidly resolved after parathyroidectomy. Severe hypercalcemia produced a concentrating defect in the renal tubule and postoperative disappearance of the nephrogenic DI confirmed its reversible, purely metabolic causes