radiation and temozolomide 2 months after surgery. On this admission for nausea and vomiting, sodium noted to be elevated at 154 mmol/L (136-145 mmol/L). Initially thought to be due to dehydration and started on intravenous fluids. Urine output noted to increase to 2-3 liters/day with persistently elevated sodium. Urine studies and labs collected while sodium 155 mmol/L significant for urine osmolality 128 mOsm/kg (50-1200 mOsm/kg), serum osmolality 330 mOsm/kg (280-295), and urine sodium 34 mmol/L. Given patient's history of prior craniotomy, radiation, and temozolomide, suspected central diabetes insipidus but DDAVP challenge with 4 mcg did not result in a lower urine output and urine osmolality remained <300 mOsm/kg. Repeat DDAVP challenge produced similar results. Evaluation of other central axis deficiencies revealed normal Free T4 (1.21 ng/dL). Unable to assess for adrenal insufficiency due to patient already on high-dose steroids for malignancy. Workup was turned to potential nephrogenic etiology as patient had been started on pantoprazole during her initial diagnosis 4 months prior. Co-peptin while sodium 148 mmol/L returned at 27.6 pmol/L (<13.1 pmol/L), indicative for nephrogenic diabetes insipidus. Patient was started on amiloride 5 mg twice a day and hydrochlorothiazide 25 mg twice a day with improvement noted in both sodium and urine output. Did discuss renal biopsy to rule-out interstitial nephritis from pantoprazole but given patient was on anti-coagulation for sub-massive pulmonary embolism, biopsy was deferred. Ultimately patient was discharged on amiloride 10 mg and hydrochlorothiazide 25 mg daily with sodium 142 mmol/L. She continued to receive temozolomide but eventually both amiloride and hydrochlorothiazide were held during a subsequent admission about a month later due to hyponatremia from poor oral intake. Sodium remained stable for about 5 months off medication at which time patient was re-started on amiloride for hypernatremia. Unfortunately patient passed due to complications from thrombosis. Review of PubMed revealed only 1 additional case report of a patient on temozolomide who developed nephrogenic diabetes insipidus but that patient was also on sulfamethoxazole-trimethoprim which was most likely the cause.

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Nephrogenic Diabetes Insipidus in a Patient on Temozolomide

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A 54-year-old female admitted for nausea and vomiting found to have hypernatremia. Four months prior to this admission, patient had undergone emergent craniotomy for an intraparenchymal hemorrhage. Found to have a 7.8 cm hemorrhagic and partially necrotic mass. Pathology consistent with anaplastic oligodendroglioma. She began