4. Free T4 improved to 0.4 ng/dL on day 3. Her mentation improved slowly and returned to baseline by day 6.

Discussion: In patients with chronic, severe hyponatremia, treatment usually entails hypertonic saline delivered either by bolus or continuous infusion with a targeted sodium correction rate of ≤8 mEq/L in any 24-hour period. However, when hyponatremia is due specifically to hypothyroidism, hypertonic saline confers high risk of overcorrection, as was evident in this patient. The pathogenesis of hyponatremia in myxedema coma is multifactorial, involving reduced free water clearance due to lower renal perfusion, inappropriately elevated ADH levels due to decreased cardiac output, and tubular dysfunction. Therefore, isotonic IV fluids and levothyroxine therapy directly address the mechanisms underlying hyponatremia and avoid the undue risk of hypertonic saline. Myxedema coma constitutes a unique context for the etiology and treatment of severe hyponatremia.

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Thyroid

PSAT361

Severe Hyponatremia in a Patient with Myxedema Coma

Maryam Nemati, MD and Katya Rubinow, MD

Introduction: Patients with myxedema coma present with hypothermia, bradycardia, hypotension, altered mental status, and hyponatremia. We present an interesting case of myxedema coma with severe hyponatremia and discuss hyponatremia management.

Case presentation: Patient is a 67-year-old female with history of hypothyroidism following thyroidectomy for papillary thyroid carcinoma, hypertension, and medication nonadherence who was found down and brought to the hospital for altered mental status. On presentation she had a temperature of 35 °C, heart rate of 49, BP of 130/105, and intermittent O_2 desaturations due to hypoventilation. On exam she was somnolent with nystagmus and lateral gaze deviation. She was moving all extremities. Lung and heart exam were normal. Labs showed sodium of 102 mEq/L (135-145 mEq/L) and serum osmolality of 243 mOsm/kg (280-300 mOsm/kg). TSH was 35U/mL (0.4-5 U/mL), and free T4 was <0.2 ng/dL (4.8-10.8 ug/dL). CBC and creatinine were normal. Head CT and chest XR were unremarkable. In the ER, she received hypertonic saline 3% as a 150mL bolus along with 1L of isotonic saline. Her sodium rapidly corrected to 117 mEq/L over 2.5 hours. She was given 3 doses of desmopressin 2mcg to control the overcorrection. She received hydrocortisone 100mg IV followed by 50mg IV q8hr. After the first dose of hydrocortisone, she was given levothyroxine 100mcg IV once followed by 75mcg IV daily. Hydrocortisone was discontinued after her cortisol level returned at 49 ug/dL (5.0-22.6 mcg/dL). Plasma sodium stabilized at 114meq/L in 48hrs. With continued IV levothyroxine alone, plasma sodium increased to 122 mEq/L by day 3 and then rose to 134 mEq/L on day