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Severe Hyponatremia as the Presenting Feature of Partially Empty Sella

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No conflicts of interest.

INTRODUCTION

We report a case of severe acute on chronic hyponatremia meeting the laboratory diagnostic criteria of SIADH. Subsequent evaluation showed secondary adrenal insufficiency due to partially empty sella. Hyponatremia completely resolved following hydrocortisone treatment.

CASE PRESENTATION

A 76-year-old man with history of hypothyroidism and SIADH was admitted with weakness, fatigue, and leg cramps for the past 3 months. He was staying in a cabin with a broken air conditioner during the summer when his symptoms started. Physical exam revealed mild dry mucus membranes, normal skin turgor, capillary refill less than 2 sec., bradycardia, and no skin hyperpigmentation. He was found to have acute-on-chronic hyponatremia with a serum sodium of 115 mEq/L (baseline 124 mEq/L-130 mEq/L), which worsened with initial volume resuscitation. Lasix and salt tablets were not effective treatment.

Other labs showed a low serum osmolality (241 mOsm/kg), high urine osmolality (598 mOsm/kg), high urine sodium (121 mmol/L), low serum uric acid (2.7 mg/dL, n 3.3-8.5 mg/dL), normal TSH, and a low normal AM cortisol (5.7 ug/dL, n 4.0-17.0 ug/dL). A subsequent cosyntropin stimulation test confirmed adrenal insufficiency with a 60 min cortisol of 10.3 ug/dL. ACTH was inappropriately normal (23 pg/mL, n 7-69 pg/mL) indicating a central etiology.

In addition, he had a mildly elevated prolactin (23 ng/mL, n 4-15 ng/mL), inappropriately normal TSH (2.83 uIU/mL, n 0.30-4.50 uIU/mL), low normal total T4 (5 ug/dL, n 4.9-11.7 ug/dL), and a mildly low total T3 (52.3 ng/dL, n58.0-159.0 ng/dL) suggesting a possible secondary hypothyroidism. A pituitary MRI revealed partial empty sella without evidence of pituitary adenoma.

The patient was started on stress-dose hydrocortisone with subsequent increased urine output (4 L within 8 h of initial treatment) and increased serum sodium as well. To prevent rapid sodium correction, one dose of desmopressin was given with a low rate hypertonic saline to equilibrate the desmopressin effect. Hydrocortisone was tapered to maintenance dose, and the fluid restriction was discontinued to prevent dehydration. His home Levothyroxine dose was increased, and he was discharged with a serum sodium of 133 mEq/L. As an outpatient, his sodium has normalized with continued treatment.

TABLES AND FIGURES

Table 2. Management course: summary

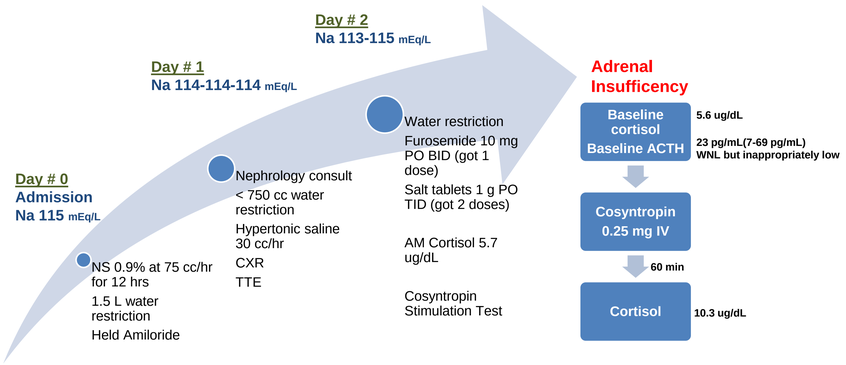


Table 3. Progression of Events: Endocrinology on Board

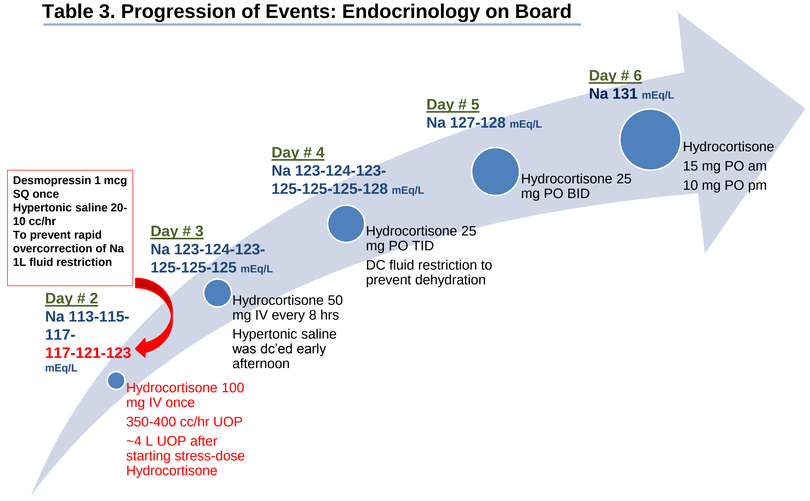


Table 4. Evaluating for Hypopituitarism

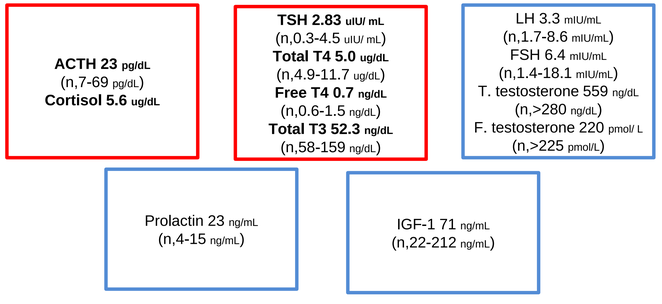


Fig. 1. Desmopressin and Hypertonic saline to prevent rapid sodium correction

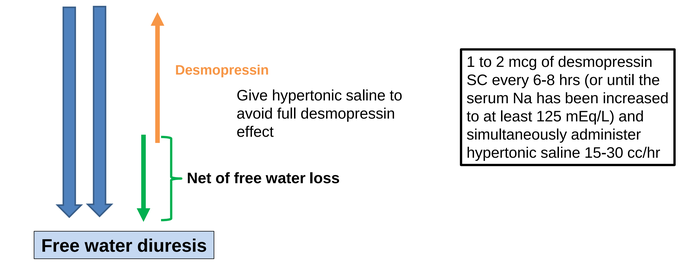
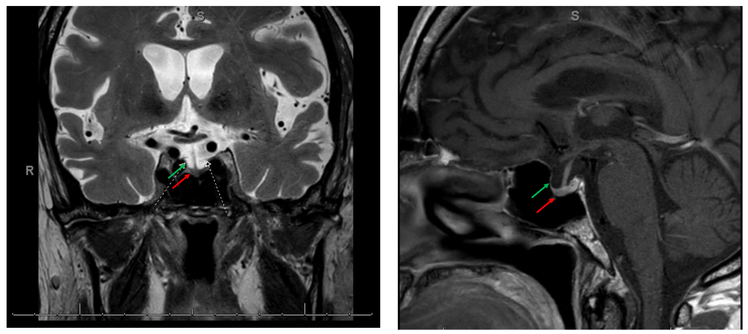


Fig. 2. Pituitary MRI showed partially empty sella turcica. No pituitary adenoma.



CONCLUSION

Secondary adrenal insufficiency is characterized by euvolemic hyponatremia with biochemical features of SIADH without hyperkalemia. ADH increases due to the lack of cortisol negative feedback loop and the reduction in systemic blood pressure and cardiac output from cortisol deficiency. Hyponatremia corrects rapidly with steroid treatment, which turns off ADH and causes free water excretion. Desmopressin and hypertonic saline can be effectively used to prevent or stop rapid correction. An interesting feature about this case is that partially empty sella is the cause for hypopituitarism.

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