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Complicated cases of hyponatremia

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Hyponatremia is the most common electrolyte imbalance encountered in clinical practice, occurring in 14%-42% of hospitalized patients. Although hypertonic saline is an effective treatment for symptomatic hyponatremia, the etiologies of hyponatremia finally should be thoroughly evaluated to prevent recurrence of hyponatremia. Hypotonic hyponatremia has many possible underlying causes. These include non-renal sodium loss, diuretics, third spacing, adrenal insufficiency, syndrome of inappropriate antidiuretics (SIAD), polydipsia, heart failure, liver cirrhosis, and nephrotic syndrome. The diagnostic algorithm is simple but does not guarantee completeness in each individual. If hyponatremia persists after cause-specific treatment, we called it as 'unresolved' hyponatremia. If hyponatremia is unresolved, we should check the initial diagnosis of the underlying cause was probably wrong or only part of the explanation because of complicated etiologies of hyponatremia. Therefore, if recurrent or refractory hyponatremia after cause specific treatment, it should be reassess using the diagnostic algorithm, and reviewing of the medications and underlying diseases that can cause SIAD. Today, I introduce two interesting cases of hypotonic hyponatremia with more than one cause.

A 49-year-old man visited emergency department with complaints of high fever (body temperature 41°C) and altered mentality on that day. He had frequency and thirst four days ago. He took celecoxib for 1 month because of a finger pain. Laboratory results showed hypo-osmolar hyponatremia (serum sodium 109 mmol/L). Further evaluation showed serum osmolality, 229 mOsm/kg; urine osmolality, 232 mOsm/kg; urine sodium, 43 mmol/L. Laboratory results for thyroid function test and rapid ACTH test were normal. The causes of hyponatremia were presumed to be SIAD due to bladder distension (urine: 1600 mL for 30 minutes after foley catheter insertion) and nonsteroidal anti-inflammatory drug, and hypovolemia due to heat stroke at the beginning. Administering hypertonic saline for symptomatic hyponatremia was started and then normal saline was additionally administered for volume expansion. The target correction was reached after initiation of treatment. Although half saline and electrolyte free water as maintenance fluids were administered for diuresis and volume expansion, he received re-lowering treatment as serum sodium reached 120 mmol/L at 18 hours. On 3-5th day of hospitalization, hyponatremia (120-122) recurred several times with huge amount of urine volume (4500-5500 mL, urine osmolality 103 mmol/L, urine Na 30 mmol/L). The cause of recurrent hyponatremia was presumed to be volume depletion due to diuresis. After normal saline infusion, serum sodium levels were gradually elevated, and urine output also gradually decreased as post diuresis improved.

A 66 year-old woman was admitted with poor oral intake and general weakness. She had a history of hypertension and took hydrochlorothiazide 12.5mg daily. Laboratory results showed hypo-osmolar hyponatremia (serum sodium 120 mmol/L). Further evaluation showed serum osmolality, 250 mOsm/kg; urine osmolality, 508 mOsm/kg; urine sodium, 173 mmol/L; basal cortisol, 0.2 mcg/dL; 30min cortisol, 2.20 mcg/dL; 60 min cortisol, 2.70 mcg/dL; ACTH, 11.3 pg/mL; free T4, 0.2 ng/dL; TSH, 8.58 mIU/mL. Magnetic resonance image showed that partial empty Sella. The causes of hyponatremia were presumed to be use of thiazide and panhypopituitarism (adrenal insufficiency and hypothyroidism) due to Sheehan syndrome. Hypertonic saline was continuously administered for symptomatic hyponatremia during first 12 hours until serum sodium reached 125 mmol/L. Serum sodium was gradually increased only by correcting the cause of hyponatremia without additional infusion of hypertonic saline (thiazide discontinuation, and administration of hydrocortisone and synthyroid).