Hypokalemia, distal renal tubular acidosis, and Hashimoto's thyroiditis

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To the editor: Renal tubular acidosis (RTA) is defined as the inability of renal tubule to acidify urine regardless of any reduction in glomerular filtration rate. Type I or distal RTA is a subtype characterized by an impaired hydrogen ion (H+) secretion in the distal convoluted tubule. This defect may be inherited or acquired, and causes H+ retention, with the resultant decrease in plasma bicarbonate and alkaline urine. Most common cause of RTA I include diabetes mellitus, Sjögren’s syndrome, multiple myeloma, primary amyloidosis, sarcoidosis, kidney transplant, obstructive uropathy, sickle cell disease, calcium metabolism disorders, and certain drugs.

Two patients with hypokalemia due to renal tubular acidosis secondary to Hashimoto’s thyroiditis are reported below. We suggest that this association is mediated by autoimmune mechanisms.

**PATIENT 1**

A 29-year-old female patient with progressive muscle weakness and quadriplegia, hyperchloremic metabolic acidosis with normal anion gap, and severe hypokalemia, which was corrected with intravenous potassium with clinical improvement. RTA type I, with high titers of anti-peroxidase antibodies (100 U/mL) and > 100 mU/mL of thyroid-stimulating hormone (TSH), was diagnosed. Despite adequate alkali administration, acid-base status was corrected when thyroid function was normalized. After treatment with levothyroxine and potassium citrate, the patient has been asymptomatic for the past 8 years.

**PATIENT 2**

A 30-year-old female patient with growth retardation due to type I RTA diagnosed in adolescence was admitted to hospital for a spontaneous hip fracture. Patient reported marked fatigue, weakness (quadripareisis), and muscle cramps for the past two years. Labora-
Hyponatremia secondary to cerebral salt-wasting syndrome associated to bacterial meningitis

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To the editor: Hyponatremia is the most prevalent water and electrolyte disorder in standard clinical practice. The cerebral salt-wasting syndrome (CSWS) is an uncommon cause of hyponatremia. The case of a male patient with bacterial meningitis who developed hyponatremia secondary to CSWS is reported below.

CASE REPORT
A 21-year-old male patient with an unremarkable history was admitted to the intensive care unit (ICU) with a diagnosis of meningococcal meningitis associated to sepsis with multiorgan failure that required mechanical ventilation, hemodynamic support with dopamine, treatment with human recombinant activated C protein, and anticonvulsant drugs due to a secondary irritative focus. Empiric treatment was started with ampicillin, cefotaxime, vancomycin, and dexamethasone, and was switched to cefotaxime alone after the causative germ was isolated (Neisseria meningitidis) and the results of susceptibility testing were known. Serum creatinine (SCR) levels at ICU admission were 3.2 mg/dL, and normalized after two days (0.9 mg/dL). Once hemodynamic stabilization, renal function control, and spontaneous breathing had been achieved, the patient was moved to the ward.

During the final days of stay at the ICU, a progressive decrease in natremia was documented (see table I), associated to polyuria of 4-5 liters daily, with normal serum levels of antidiuretic hormone (ADH) and high levels of brain natriuretic peptide (pro-BNP). Adequate intravenous volume replacement with physiological saline based on urinary sodium loss allowed for normalization of natremia and a normal volume status (table I).

DISCUSSION
In patients with central nervous system diseases, hyponatremia does not have to be necessarily related to a syndrome of inappropriate ADH secretion (SIADH), but may be secondary to a CSWS. Subarachnoid hemorrhage is the most common cause of CSWS, but this has also been reported to be associated to meningitis of an infectious origin. A new case of CSWS occurring in a young adult after resolution of a bacterial meningitis is reported.

Diagnosis of CSWS requires the presence of an inappropriate diuresis for circulating sodium levels and volume depletion. Diagnostic suspicion of CSWS is essential for hyponatremia control, because its treatment is totally different from that of SIADH. While volume and sodium replacement is essential in CSWS, SIADH responds to water restriction. In the case reported, CSWS was suspected based on the existence of polyuria associated to hyponatremia and elevated natriuresis. Elevated serum levels of pro-BNP confirmed diagnosis of CSWS. The increase in pro-BNP serum levels secondary to the inflammatory process in the central nervous system could be related to the inappropriately high natriuresis.

To sum up, occurrence of hyponatremia combined with increased natriuresis and volume depletion in patients with central nervous system disease should raise the suspicion of a CSWS.

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