tory tests results: TSH > 100 mU/mL, high titers of anti-peroxidase antibodies (300 U/mL), and low free thyroxin levels (table). Treatment was started with levothyroxin 150 mg/day and calcium citrate. Densitometry showed severe osteoporosis. Patient has not relapsed after 7 years of hormone replacement therapy.

Hashimoto's thyroiditis is an autoimmune disease, and type I RTA has also been related to autoimmunity.[6-8] Antibodies directed against collecting tubule cells could play an outstanding role in this setting, affecting acid-base status and potassium balance. These are the first cases reported in the literature of hypokalemic paralysis caused by type I RTA as a presentation form of Hashimoto's thyroiditis.

Taking into account that Hashimoto's thyroiditis is the most common cause of hypothyroidism, with a 1% prevalence, type I RTA could be an underdiagnosed associated condition with variable grades of clinical expressivity.

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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.[1] 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.[2,3] 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.[4] 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.[5] 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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Table I. Changes in laboratory test parameters during admission

	ICU →					WARD →				
	6/1/05	**7/1/05**	**9/1/05**	**10/1/05**	**11/1/05**	**13/1/05***	**17/1/05**	**18/1/05**	**19/1/05**	**21/1/05**
SCr (mg/dL) (N: 0.6-1.1)	0.7	0.8	0.7	1	0.8	0.7	0.7	0.5	0.5	0.5
PNa (mmol/L) (N: 135-145)	139	**134**	**133**	**130**	**131**	**133**	**134**	136	137	137
UNa (mmol/24 h) (N: 75-200)						**489**	**364**	**806**	**262**	**335**
POsm (mOsm/kg) (N: 275-300)						277				291
UOsm (mOsm/kg) (N: 300-1,300)								429		423
Pro-BNP (pg/mL) (N: 0-84)						**97**				**303**
ADH (pg/mL) (N: < 7.6)						3.5				2.5

Abbreviations: SCr: serum creatinine. PNa: plasma sodium; UNa: urinary sodium. POsm: plasma osmolarity. UOsm: urinary osmolarity. pro-BNP: brain natriuretic peptide. ADH: antidiuretic hormone. (*) Start of saline infusion adjusted to urinary losses.
Bold numbers indicate out of normal ranges.

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Disseminated histoplasmosis in a kidney transplant patient

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To the editor: Four years after receiving a kidney transplant from an unrelated living donor for polycystic kidney disease,[1] a 49-year-old female patient started to experience episodes of hyperthermia. The immunosuppression regimen consisted of methylprednisone 4 mg/day, micophenolate mofetil 2 g/day, and tacrolimus 2 mg/day. She was admitted to hospital for suspicion of complicated renal cysts and was found subcutaneous nodules that were biopsied and seen to be consistent with erythema nodosum. Nephrectomy of the right native kidney was performed, but fever persisted. Cultures were persistently negative. Patient developed massive ascites. Magnetic resonance imaging showed ascites and a complicated liver cyst, that was marsupialized, and two liters of ascites were drained. Peritoneum was normal upon inspection. After 22 days of incubation, subcutaneous nodules developed colonies of *Histoplasma capsulatum.* Fever persisted despite itraconazole administration, and amphotericin B was therefore added. Subsequent blood, urine, and ascites fluid cultures were positive for *Histoplasma capsulatum*. Fever and ascites resolved 29 days after hospital admission, and patient was discharged home. Two weeks later, fever and subcutaneous nodules in the arms recurred. Severe pericardial effusion with ultrasonographic signs of tamponade was diagnosed. The patient died during the procedure.

While early fungal infections are less commonly reported in kidney transplant patients than in other transplant patients, their mortality is high.[2] These patients have a high risk of suffering late opportunistic infections, particularly by *Listeria, Nocardia, Mycobacteria*, or fungi, associated to use of venous catheters, antibiotics, corticosteroids, surgical procedures, and diabetes mellitus.

Approximately after 6 months since transplant, incidence of opportunistic fungal infections decreases, but an increase occurs in the incidence of systemic endemic fungal disease, of which histoplasmosis and coccidioidomycosis are the most significant. Histoplasmosis is the most common endemic mycosis in immunosuppressed patients in Argentina,[3,5] with an incidence rate up to 40% in humid, temperate areas. In our country, the most common clinical presentations in healthy individuals include the pulmonary variant and the chronic disseminated variant associated to mucocutaneous lesions, adenomegalies, hepatomegaly, and splenomegaly.[6]

In normal subjects, primary infection is usually asymptomatic in more than 90% of cases. In a lower proportion, clinical signs include pericarditis, mediastinitis, erythema nodosum, or retinitis.[7]

Ascites is a complication of systemic histoplasmosis not previously reported