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

in the 48 cases of histoplasmosis in kidney transplant. It should be noted that surgeons did not take peritoneal biopsies during abdominal laparoscopy because of the normal characteristics of the serosa. This could be due to the abnormal inflammation patterns in immunosuppressed patients. We think random peritoneal biopsies should be performed despite absence of gross structural changes when the cause of ascites has not been diagnosed.[8]

Patients transplanted solid organs have an increased risk of suffering opportunistic infections. Systemic histoplasmosis is a rare and serious condition that should be considered as a long-term complication in transplant patients.

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Autoimmune thyroiditis, subclinical hypothyroidism, and nephrotic syndrome caused by menbranous nephropathy

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To the editor We report a new case of glomerular disease associated to thyroid disease, after the one recently published in this same journal.[1]

A 60-year-old female patient with an unremarkable history consulted her general practitioner complaining of fatigue, depressive symptoms, and mild ankle edema in the previous month. Laboratory test results included serum albumin levels of 1.9 g/dL, cholesterol levels of 338 mg/dL, creatinine levels of 0.9 mg/dL, proteinuria of 5.8 g/24 h, TSH 9.9 µIU/mL (normal 0.4-4), antiperoxidase antibodies 714 IU/mL (normal < 60). Treatment was started with levothyroxin 50 mcg/24 h and atorvastatin 20 mg/24 h, and patient was finally referred to hospital six months later for completing work-up. The only significant findings in physical examination were signs of venous insufficiency in the lower limbs with no noticeable ankle edema. At the time, the patient had normal renal function with the following laboratory test results: albumin 2.6 mg/dL, cholesterol 239 mg/dL, proteinuria 3.4 g/24 h, TSH 10.9 µIU/mL, free T4 0.98 ng/dL (normal 0.6-1.8), antiperoxidase Ab > 1300 IU/mL, antithyroglobulin Ab > 500 IU/mL (normal < 60). Autoimmune study was normal, and tumor markers and viral serologic tests (HBV, HCV) were negative. Colonoscopy and mammography were normal. A percutaneous renal biopsy showed lesions consistent with a stage II membranous kidney disease. Based on diagnosis of autoimmune thyroiditis, subclinical hypothyroidism, and nephrotic syndrome due to membranous nephropathy, levothyroxin was discontinued because of normal T4 levels and treatment was started with lisinopril 20 mg, candesartan 16 mg, and sustained release fluvastatin 80 mg. Six months later, the patient remained in a good clinical condition, with no edema, and laboratory tests showed a partial remission of nephrotic syndrome with proteinuria of 1.6 g/24 h, serum albumin 4.1 g/dL, cholesterol 177 mg/dL, as well as a virtually normal thyroid function (TSH 4.5 µIU/mL, T4 1 ng/dL).

Association of thyroid disease and glomerular diseases is known, though few cases have been reported. Particular mention should be made of association of Graves disease and membranous nephropathy.[2] Autoimmune thyroiditis has also been reported to be associated to this same nephropathy,[3,4] and also to IgA nephropathy,[5,6] minimal change disease,[1,7,8] and membranoproliferative glomerulonephritis.[9,10]

As reported by some authors,[1,10,11] simultaneous occurrence of thyroid and glomerular disease could be explained by the existence of an autoimmune pathogenesis common to both conditions, and incidence could be higher than suspected, with proteinuria being found in a high proportion of patients with autoimmune thyroiditis and Graves disease. In our case, thyroiditis was diagnosed based on positive antithyroid antibodies, and its clinical manifestation was a subclinical hypothyroidism. The parallelism seen between remission of nephrotic syndrome and TSH normalization should be noted. Use of levothyroxin, corticosteroids, or other immunosuppressants, that were administered in some previously reported cases,[3,6-10] was not required. The need for investigating thyroid function in cases of apparently idiopathic nephrotic syndrome should be stressed.

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