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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