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Systemic lupus erythematosus and hypothyroidism

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To the Editor,

The combination of systemic lupus erythematosus (SLE) and altered thyroid function has been described in several different studies. The most common alteration has been described as primary hypothyroidism. However, the presence of central hypothyroidism in patients with SLE is very uncommon. Here we present the case of a patient with SLE that, in the course of an outbreak of lupus nephritis, developed severe suprathyroid hypothyroidism

Our patient was a 33-year old male diagnosed with SLE in 2000 based on an analysis of polyarthralgia and cutaneous lesions. In 2001, he developed pure nephrotic syndrome. A renal biopsy indicated membranous glomerulonephritis (stage V) that went into complete remission following immunosuppressant treatment. In 2005, he had another bout of nephritis in the form of impure nephrotic syndrome; we performed another biopsy and detected membranous glomerulonephritis (GN), accompanied by necrosis and proliferation in half of the glomeruli and centres of fibrosis, as well as interstitial atrophy (stage 4-5). He responded partially to several immunosuppressants, and was stabilised at a plasma creatinine level of 1.5-1.8mg% and proteinuria in the non-nephrotic range. He then was treated with losartan, prednisone (5mg/day), and simvastatin.

In March 2011, the patient sought treatment for the appearance of tibiomalleolar oedema. The physical examination revealed blood pressure of 210/120mm Hg, pallor of the mucosa, and pitting oedema in both legs. A laboratory analysis revealed: haemoglobin (Hb): 10g/dl; creatinine: 4.3mg/dl (glomerular filtration rate [GFR]: 15ml/min/1.73m^2); albumin: 25g/l; antinuclear antibodies (ANA): 23 (positive >1); anti-DNA: 405U/ml (positive >15); C3: 28mg/dl (76-181); C4: 4.4mg/dl (12-49); proteinuria/24 hours: 11g, and sediments with haematuria. We started the patient on prednisone and mycophenolate, with anti-hypertensives to control the arterial hypertension. We observed the progressive disappearance of the oedema, as well as improved renal function (creatinine: 3mg/dl and reduction of proteinuria to 3g/24h). Seven days after starting treatment, the patient complained of severe asthenia that impeded mobility, constipation, and a constant feeling of cold. The thyroid analysis revealed: thyrotropin (TSH): 0.09μU/ml (0.34-4.9); free T4 thyroxin: 0.60mg/dl (0.69-1.48), free T3 triiodothyronine: 1.4pg/ml (1.71-3.71); reverse triiodothyronine: 0.19ng/ml (0.10-0.34), and thyroid peroxidase antibodies (TPO): 6.92U/ml (0-5.6). The measurements of gonadotropins (FSH, LH), prolactin, human growth hormone, testosterone, and somatomedin C (IGF-1) were all normal. A thyroid ultrasound and magnetic resonance of the hypophysis resulted normal. We started treatment with levothyroxine, observing a progressive disappearance of the hypothyroid symptoms, and normalised plasma levels of free thyroxin.

Several different studies have shown that altered thyroid function is more common in SLE patients than in the general population.[1-4] Primary hypothyroidism, both clinical and subclinical, is the most commonly observed alteration. Two recent studies compared patients with SLE with a control group and observed a prevalence of primary clinical hypothyroidism of 6% and 14%, and subclinical hypothyroidism of 12% and 17%, respectively.[5,6] The prevalence of clinical hypothyroidism in the general Western population is less than 1%. Based on the presence of antithyroid antibodies in patients with SLE and hypothyroidism, half of all cases have an autoimmune origin, and the percentage of positivity for antithyroid antibodies in patients with SLE and euthyroidism ranges between 6% and 47%.

On the other hand, the majority of these studies suggest that there is no difference in the prevalence of hyperthyroidism between patients with SLE and the general population. We must point out that the association between SLE and central hypothyroidism is rare. The cases described have been of patients with SLE that develop lymphocytic neurohypophysis, which also produces altered secretion of other hormones in addition to the thyroid hormones.[7]

Taking into account this prevalent association and the fact that clinical and laboratory manifestations of hypothyroidism can simulate a lupus outbreak,[8] we suggest performing an analysis of thyroid function in patients with SLE.

Conflicts of interest

The authors have no conflicts of interest to declare.

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ERRATA

In the article ***«La eritropoyetina recombinante humana en la enfermedad renal crónica: lecciones que aprender»*** (Recombinant human erythropoietin for chronic kidney disease: lessons to learn), published in the extra supplement ***NEFROLOGÍA BASADA EN LA EVIDENCIA***, volume 2, issue 4 from 2011, on page 7, it has been left out that content is accessible on the NEFROLOGÍA website: http://www.revistanefrologia.com/modules.php?name=articulos&idarticulo=11056&idlangart=ES

We apologize for the error to our readership.