

Concomitant Diagnosis of Primary Non-Autoimmune Hypothyroidism and Systemic Lupus Erythematosus

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Abstract: Primary hypothyroidism is the most common thyroid dysfunction during systemic lupus erythematosus (SLE). Its prevalence is estimated 3.9-6.6% depending on the series, is mainly due to autoimmune thyroiditis, and is often diagnosed in already known patients with SLE. Non-autoimmune primary hypothyroidism remains exceptional and concomitant forms are rare.

We report the original observation of a non-autoimmune primary hypothyroidism diagnosed concomitantly with SLE in a 19-year-old Tunisian woman. As rare as it is, this association deserves to be known by health professionals given its therapeutic and prognostic implications.

Keywords: Primary hypothyroidism, systemic lupus erythematosus, thyroid dysfunction, connective tissue disease.

1. INTRODUCTION

Thyroid dysfunctions are common in patients followed for systemic lupus erythematosus (SLE) [1, 2]. The clinical presentation of these dysfunctions can be of overt or subclinical hypothyroidism, overt or subclinical hyperthyroidism, sick euthyroid syndrome (isolated low triiodothyronine (T3), and euthyroid autoimmune thyroid disease [1-3].

Primary hypothyroidism is the most common thyroid dysfunction during SLE [4, 5]. Its prevalence is estimated 3.9-6.6% depending on the series [7]. It is mainly due to autoimmune thyroiditis (Hashimoto's thyroiditis) [1-7]. Non-autoimmune primary hypothyroidism remains exceptional [6, 7]. Thyroid dysfunction is most often diagnosed in already known patients with SLE; the concomitant forms are rare [6].

We report the original observation of a non-autoimmune primary hypothyroidism diagnosed concomitantly with SLE in a 19-year-old Tunisian woman.

2. CASE REPORT

19-year-old Tunisian woman, without pathological medical history, was explored for acute inflammatory polyarthritis associated with

Raynaud's syndrome, edema of the extremities, and recent constipation.

The somatic examination noted synovitis of the wrists, metacarpophalangeal and interphalangeal joints of both hands, a malar rash (vespertilio), discreet painless edema on both legs, and macroglossia.

Biology showed: lymphopenia at 1000/mm³, thrombocytopenia at 102,000/mm³, hemolytic anemia at 9.2 g/dl with positive direct coomb test, erythrocyte sedimentation rate at 89mmH1, C-reactive protein at 34mg/l, and proteinuria at 4.1 g/24h. The rest of the basic bioassays were within normal limits: leukocytes, neutrophils, blood sugar, creatinine, calcium, transaminases, muscle enzymes, uric acid, lipid parameters, serum ionogram, and serum protein electrophoresis.

Immunological tests revealed positive anti-nuclear antibodies at 1/160 with speckled fluorescence, positivity-native DNA, anti-Sm, and anti-nucleosome antibodies.

The radiographs of the painful and swollen joints were without abnormalities.

The thyroid assessment noted a primary hypothyroidism with a thyroid stimulating

hormone (TSH) at 14 μ IU/ml and total free thyroxine at 6.8 pmol/l. The anti-thyroid antibodies (anti-thyroperoxidase and anti-thymoglobulin antibodies) were negative. Ultrasound showed a thyroid gland of normal volume, homogeneous, and without focal lesions.

Exploration for systemic visceral involvement of lupus disease concluded to right pleurisy, minimal pericarditis, partial thrombosis of the inferior vena cava, and type Vb lupus nephropathy on renal biopsy.

Thus, the diagnosis retained was that of SLE with cutaneous, articular, hematological, cardiovascular, pleural and renal involvement, associated with a concomitant non-autoimmune primary hypothyroidism.

The patient was treated with systemic corticosteroids, intravenous cyclophosphamide, effective anticoagulation, and progressive hormone replacement therapy up to 125 μ g/d of levothyroxine normalizing her TSH. The evolution was favorable with complete remission of the lupus flare, and clinical and biological euthyroidism. No recurrence has been noted for five years.

3. DISCUSSION

The most common endocrine dysfunctions in patients followed for SLE are: hypothyroidism, type 2 diabetes mellitus, and hyperthyroidism (prevalence of 5.22%, 1.41%, and 1.41% respectively in Muñoz C et al series of 708 SLE patients) [6]. Among all thyroid dysfunctions, autoimmune thyroiditis is the most frequent in association with SLE [1-5]. The diagnosis of these endocrine dysfunctions is classically later than that of SLE; concomitant forms are rare: only 7.76% of cases in Muñoz C et al cohort [6].

Hypothyroidism is significantly more frequent in lupus patients compared to the general population: 5.7% versus 1% in Pyne D et al series [8]. Likewise, patients with hypothyroidism, particularly if autoimmune in nature, seem to be significantly more at risk of developing systemic lupus [9, 10]. Apart from autoimmune thyroiditis, primary hypothyroidism concomitant with SLE is exceptional [6-8]. Their origin/mechanism is not yet fully understood (glandular localization of lupus disease?) [8, 11].

The presence of hypothyroidism during SLE is associated with severe and active forms of the disease [5, 11]. Indeed, it was found a

statistically significant association between thyroid dysfunction and the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) [5]. In addition, hypothyroidism, even if subclinical, can be the cause of a delayed clinical complete response to specific treatment in patients with SLE [12].

4. CONCLUSION

As rare as it is, this association deserves to be known by health professionals given its therapeutic and prognostic implications.

Thyroid monitoring is essential in any patient followed for lupus, even if the anti-thyroid auto antibodies are initially negative. Similarly, regular monitoring for several years is desirable in hypothyroid patients to detect signs of SLE as soon as possible.

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