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HYPOTHYROIDISM AS INITIAL CLINICAL MANIFESTATION IN SYSTEMIC LUPUS ERYTHEMATOSUS- A CASE REPORT

General Medicine

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ABSTRACT

Hypothyroidism is an organ-specific autoimmune disease, whereas Systemic Lupus Erythematosus (SLE) is a multisystem autoimmune disorder. The two illnesses can develop one after the other or concurrently. Most previous studies found that thyroid illness was more common in SLE patients than in everyone else, especially in those who had higher rates of anti-thyroid antibodies. Here as we report a case of lady who came to us with hypothyroidism as the initial clinical presentation and was later diagnosed to have systemic lupus erythematosus Hence, awareness should be raised for screening SLE while making a diagnosis of hypothyroidism and the importance of thyroid disease should also be acknowledged in the treatment of SLE. This may help detect diseases at an early stage.

KEYWORDS

Systemic Lupus Erythematosus (SLE), Hypothyroidism, anti-nuclear antibodies (ANA), autoantibodies (AAbs)

INTRODUCTION

Systemic lupus erythematosus is a multisystem autoimmune disease of the connective tissue denoted by wide variety of manifestations, production of autoantibodies (AAbs) and chronic inflammation of many organs, with a higher incidence among females.[1] The pathogenesis includes local deposition of anti-nuclear antibodies (ANA) and activation of the complement system.[2]

The estimated world-wide prevalence is between 20 to 70 per 100,000 persons,[3] With a very high predisposition for women of childbearing age. In females between the age of 15 to 44 of age, the female to male ratio is most likely 13:1 whereas it is only 2:1 in younger and in the older age groups.[4,5]

The etiology is unknown, with a complex interplay between gender, genetic, ethnic, hormonal, and environmental factors. Despite significant progress in early detection and advancements in treatment, there is still significant early mortality associated with infections and organ-specific disease development.[6]

Numerous environmental triggers have been implicated in lupus. Drugs, Ultraviolet light, infections, smoking, silica, mercury, Epstein-Barr virus, and others.[7-10] Also, with 50% increased risk, psychological stress has been attributed in developing lupus.[11,12]

There are many end-organ effects of systemic lupus erythematosus (SLE) that are known. Many studies [13-15] have shown a correlation between thyroid illness and the presence of SLE. Given that both lupus and thyroid disease have nonspecific symptoms including fatigue, weight changes, dry hair, and skin signs, they can be confusing. The first description of the relationship between thyroid disorders and lupus date back to 1961.[16] In 1987, the first prospective research of thyroid abnormalities in SLE patients was conducted, and it was revealed that individuals with SLE usually had abnormal thyroid function test results.[17] Since then, studies have found time and time again that lupus patients are more likely to experience thyroid problems than the general population.[18] A case of SLE with hypothyroidism as the initial clinical manifestation is described in the given study.

Case Report

A 20-year-old female presented with complaints of fever with chills for 10 days, severe body and joint pain for 3 months, swelling of B/L lower limb for 1 month, accompanied by lethargy, memory loss, lack of concentration, recurrent oral ulcer, frequent hair loss, loss of appetite and shortness of breath. A red, nonpruritic rash on her face initially attributed to sunburn for 15 days. Her past medical history is unremarkable, and family history was not significant.

Physical examination revealed the following:

The patient is a thin young woman with a temperature 37.8°C, pulse 80 bpm, blood pressure 118/72 mm Hg, and respiration 16 breaths/minute. There is a well demarcated, erythematous, mildly scaly rash on bilateral cheeks over the malar eminences, across the nasal bridge, and on the chin with sparing of the nasolabial folds. Diffuse alopecia without scarring is present. There were oral ulcers with mild conjunctival pallor. The neck is supple without lymphadenopathy. There was a bilateral lower limb edema. Examination of the extremities reveals tender, swollen metacarpo phalangeal (MCP) and wrist joints bilaterally with mild effusions and intact range of motion. The lungs are clear, and there are no cardiac rubs or murmurs. The abdomen is soft and nontender without organomegaly. Weakened bilateral knee reflex and ankle reflex, with normal muscle strength.

Laboratory Findings

Laboratory testing on admission revealed Hemoglobin of 9 g/dL with mean corpuscular volume 87 fL, hematocrit 14%, red blood cell (RBC) count 1.6×106 mcL, white blood cell count was 2800/mm3, Platelet count 90,000/µL, serum electrolytes level were normal, serum creatinine was 1.1 mg/dL, erythrocyte sedimentation rate was 120 mm/hr, C-reactive protein was positive, urine routine microscopy showed 1+ proteinuria, 3 red blood cells (RBCs)/high-powered field (HPF), without casts, thyroid stimulating hormone was 12 mIU/L, rheumatology workup revealed ANA positive, dsDNA antibody positive, nRNP/Sm, SM, SS-A, Ro-52, Ribosomal-p Protein were positive. ANA titer was reported at 1:320 with a speckled ANA pattern. A plain radiograph of the chest and electrocardiogram (ECG) were unremarkable. Indirect coombs test direct coombs test were negative.

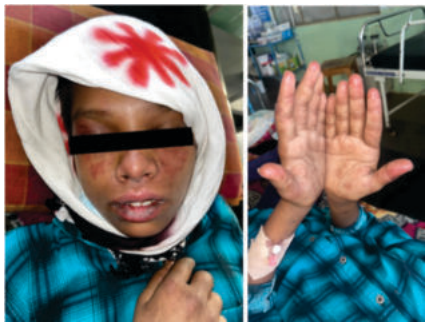
Procedure

The patient was initially treated with intravenous antipyretics and thyroid hormone replacement therapy with oral levothyroxine (25µg/day), hydroxychloroquine 400 mg/tds, methyl prednisolone pulse therapy of 500mg intravenous for 3 consecutive days. Later the patients were stared on oral corticosteroids with a dose of 35 (1 mg/kg). After 10 days the patient was discharged on oral thyroxine (25 µg/day) and oral prednisolone 35 mg. Oral prednisolone was tapered to 5 mg daily eventually. The patient was also prescribed sunblock SPF 50 daily over the limbs and face, betamethasone valerate cream 1:4 daily over the skin lesions. Subsequent follow-up showed that the patient had marked improvement in the skin lesions, oral ulcers had healed and she no more had joint pain.

DISCUSSION

A systematic literature in PubMed and CNKI databases was observed to identify research published from 1987 which contain information on the prevalence of hypothyroidism with SLE patients. Most of the research identified a high prevalence of hypothyroidism in patients

with SLE; the frequency of clinical hypothyroidism reported in the literature ranged from 3.0-21.4 percentage.[19] As compared to males the prevalence is higher in females.[20] Research has shown that arthritis and skin damage are most prevalent in patients with SLE with hypothyroidism, whereas, hematological abnormalities and neuropsychiatric symptoms were not that common.[21]



How the pro-inflammatory immunological response brought on by SLE affects thyroid function is unknown.[22] The immunological predominance of T helper 1 (Th1) cells appears to be a key similarity between thyroid illness and SLE.

Hypothyroidism is the most common thyroid disorder seen in patients of SLE. 15-19% of patients with lupus have hypothyroidism.[13, 23, 24] In comparison to male patients, female SLE patients have a tendency to have a higher prevalence of both subclinical and clinical hypothyroidism.[24] However also in male patients with SLE there is greater incidence of hypothyroidism as compared to healthy male controls (OR 5.26; 95% CI 3.61-7.68).[24]

There are studies that show clinical correlation between severity of outcome of both these disorders. In a six-month study of 363 SLE patients with subclinical hypothyroidism, Dong et al.[19] found that delaying the treatment of subclinical hypothyroidism delays the remission of SLE. In a case-control study involving 1006 SLE patients, Gao et al.[20] found that those with lupus nephritis had long-standing subclinical hypothyroidism.

Compared to the general population, persons with SLE appear to suffer from hyperthyroidism at a slightly higher rate, though less commonly than hypothyroidism. According to reports, 1.3% of the general population has hyperthyroidism.[14,25]Rates of hyperthyroidism in people with SLE range from 3% to up to 9% of the population.[23]

Regardless of the fact that people with SLE do not just have particular thyroid diseases a variety of thyroid diseases and SLE have been linked for more than 50 years, The most prevalent thyroid disorder among lupus patients is hypothyroidism, and both conditions likely have an immunological genesis in common. Patients with SLE are more likely to have thyroid nodules and hyperthyroidism than people without the disease.[26] Though the prevalence of thyroid cancer in lupus patients is less, it is twice as common in these individuals compared to those without SLE. Compared to women with SLE and no thyroid disease, women with SLE with thyroid disease have greater incidence of preterm birth.[27,28]

In conclusion, despite the wide range of symptoms and manifestations of thyroid illness, the rheumatologist should carefully assess the implications of any thyroid disease manifestation in any patient with lupus.

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