THYROID DISORDERS AND AUTOANTIBODIES IN SYSTEMIC LUPUS ERYTHEMATOSUS

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KEY WORDS: SLE, THYROID FUNCTION ABNORMALITIES, ANTITHYROID ANTIBODIES

ABSTRACT

Objective: To determine the patterns of thyroid dysfunction and autoantibodies associated with systemic lupus erythematosus (SLE).

Methodology: The study was performed on a group of 35 SLE patients and 10 matched apparently healthy subjects as a control group. Various clinical and laboratory parameters of SLE were measured for both groups. Also thyroid function tests were measured, which included TT₃, FT₄ and TSH. Antimicrosomal and antithyroglobulin antibodies were measured for patients. Patients were classified according to their thyroid function tests into 3 subgroups: subclinical hypothyroid (10 patients), subclinical hyperthyroid (8 patients) and euthyroid (17 patients).

Results: We found that the euthyroid subgroup showed lower hemoglobin level, higher ESR, and higher serum creatinine level that was of a high statistical significance (p<0.01). Also TSH level (2.4 ± 1.13) was higher than controls (1.72 ± 0.52) and this was of statistical significance (p<0.05).

In the hyperthyroid group, the level of the ESR, serum creatinine and FT₄ was higher than that of controls. This was also of a high statistical significance (p<0.01). The level of HB and RBCs was lower than in controls with high statistical significance. In the hypothyroid group, The levels of Hb and FT₄ was lower than in controls while the level of ESR, serum creatinine and TSH was higher than controls with high statistical significance (p<0.01).

Thirty percent (30%) of the patients showed positive antimicrosomal antibodies and twenty percent (20%) showed positive antithyroglobulin antibodies. The presence of these antibodies correlated positively with TSH levels. This might explain the thyroid disorders in SLE patients.

Conclusion: Performance of thyroid dysfunction tests in SLE patients, as part of biochemical and immunological profiles, may help in early detection of associated thyroid disorders.

INTRODUCTION

Autoimmune disorders occur together in the same patient. Autoimmune thyroid disorders have been shown to occur in association with connective tissue disorders such as juvenile rheumatoid arthritis Macejova et al. (2006). The association of thyroid disorders with systemic lupus erythematosus (SLE) has been confirmed Karmer et al. (2005). Autoimmune thyroiditis is often related to non-specific autoimmune organ diseases such as Rheumatoid Arthritis, systemic sclerosis, SLE or polymyalgia.
Thyroid disease is often difficult to diagnose clinically in the general population and could certainly be under-diagnosed in SLE patients because of overlapping symptoms of lupus and thyroid disease. Both lupus and thyroid disorders can cause fatigue, focal edema, weakness, myalgia, arthralgia and a variety of other non-specific complaints (Weetman, 2005).

Thyroid disorders may be the result of antithyroid activity of one of the antibodies produced in SLE (Innocencio R.M et al., 2004).

Asvold et al. (2006) indicated that the PRL level is higher in SLE patients and that in the presence of hyperPRL there is increased prevalence of thyroid antibodies. This gives evidence to the association of PRL and autoimmunity and pointing to the appropriateness of assessing and monitoring the progress of this marker in patients affected by these disorders (Karmer et al., 2004).

The results of the study of Koller et al. (2004) indicated that SLE patients and thyroid function abnormalities can demonstrate thyrotrophin binding inhibitory immunoglobulins and thyroid stimulating immunoglobulins activity in their serum. However, these antibodies do not necessarily correlate with specific abnormalities of thyroid function.

It is also possible that frequent abnormal thyroid tests may represent alternations that are secondary to the production of thyrotrophin by activated lymphocytes (Miyawaki et al., 2005), a peculiar metabolism of thyroid hormones in SLE patients. Also the pathogenesis of thyroid disease in SLE patients may be explained by the fact that HLA type B8:DR3 which occurs significantly more commonly among SLE patients or autoimmune thyroid disease. Therefore, there may be a subset of people who have a genetic predisposition to develop both disorders (Punzi & Betterle, 2004).

Lawind et al. (2004) stated that it is possible that during flares of SLE activity, elevated interferon levels lead to aberrant major histocompatibility complex antigen expression by thyrocytes provoking an autoimmune response and the development of antithyroid antibodies (Bastian et al., 2006).

Aim of Work:

We aimed at finding out the prevalence of abnormalities of thyroid function, the presence of antithyroglobulin and antimicrosomal antibodies and their correlation with different clinical and laboratory parameters of SLE patients.

SUBJECTS AND METHODS

Subjects:

Thirty five SLE diagnosed according to the American College of Rheumatology Criteria (Tan et al., 1982) who used to attend the Outpatient Clinic of the Rheumatology and Rehabilitation Department of Banha University Hospitals were included in the study. A group of 10 apparently healthy subjects matched for age and sex were included as a control group. None of the patients or controls had a history of thyroid disorder or clinical hyperthyroidism (marked weight loss, heat intolerance, neck swelling, tremors or eye signs) or clinical hypothyroidism (lethargy, weight gain, cold intolerance or bradycardia).

Methods:

All subjects included in this study were subjected to the following:

a) Thorough clinical examination with particular stress on the presence of malar rash, oral ulcers, skin lesions, arthritis, serositis, renal affection and CNS affection. Disease activity for SLE patients was assessed according to Bombardier et al. (1992).
Laboratory investigations: blood samples were withdrawn from all subjects and divided as follows:

- Samples on ethylenediaminetetraacetic acid (EDTA) for examination of red blood cells (RBC) count, hemoglobin (Hb), total leucocytic count (TLC) and platelet count.
- Samples on citrate for estimation of erythrocyte sedimentation rate (ESR).
- Clotted samples for estimation of serum creatinine, antinuclear antibodies, anti-DNA antibody, total T₃, free T₄ and thyroid stimulating hormone (TSH).
- Anti thyroglobulin antibody (Atg) and antimicrosomal antibody (Amic).
- Serum creatinine was assayed using modified Jaffe method on CX7 autoanalyzer.
- Serum total T₃ was assayed using competitive enzyme immunoassay (EIA) as recommended by Wisdom (1976) where both total T₃ in the patient serum and enzyme labeled T₃ compete for limited amounts of T₃ antibodies, then a wash was made to remove unbound labeled T₃. Finally color developer (Tetramethyl-benzidine) was added to give a blue color and then a stop solution was added to give yellow color and the result was read on an ELISA reader (450 nm).
- Serum free T₄ was assayed using competitive enzyme immunoassay according to Hansen et al. (1978) in a procedure similar to total T₃ assay and the result was read on an ELISA reader (450 nm).
- Serum TSH was assayed using non-competitive sandwich enzyme immunoassay Blomme et al. (1967). The assay system utilizes a unique monoclonal antibody directed against a distinct antigenic determinant on the intact TSH molecule. Mouse monoclonal anti- TSH is used as a second antibody in the conjugate solution. Finally, tetramethylbenzidine is used as color developer followed by a stop solution and the result is read on an ELISA reader (450 nm). The three thyroid hormones kits are supplied by Genzyme Diagnostics (1531, Industrial road, San Carlos, CA 94070 U.S.A).

Antinuclear antibody (ANA) was tested using the indirect immunofluorescence method Tan, (1982) where rat liver sections were employed. Substrate slides (rat liver sections) were overlaid with serum dilutions (1:20) and incubated to allow the ANA present in positive sera to bind nuclear antigens. Sites where ANA reacted with nuclear constituents are made visible by binding to them fluorescin isothiocyanate (FITC) labeled goat antihuman gamma globulin (the conjugate).

The patterns were recognized using the fluorescent microscope. Homogenous, peripheral or nucleolar patterns can be present in case of SLE. Anti- DNA antibody was tested using the indirect immunofluorescence method (Beutner et al., 1985). Crithedia Lucia sections were used as substrate on slide and a procedure similar to the previous one was followed.

Both anti DNA and ANA sections and reagents were supplied by SIGMA Diagnostics. Anti Thyroglobulin and antimicrosomal antibodies was performed using MT-fluoro-kit for indirect immunofluorescence supplied by INCSTAR Corporation, Stillwater, Minnesota, USA using the method recommended by Rose (1978).

Patients' serum samples were diluted in phosphate buffered saline and overlaid onto monkey thyroid cryostat sections fixed on a microscope slide. The resultant positive reaction was observed as apple-
green fluorescence of specific tissue when viewed with the fluorescence microscope.

**RESULTS**

Our patients were classified according to their thyroid function test into 3 subgroups: subclinical hypothyroid group, subclinical hyperthyroid and euthyroid subgroup. The number, age and sex of these subgroups and the control one are shown in table (1) and the percentage of patients in each group are shown in Fig.1.

Table (1): number, Age and Sex of all groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>Age in years</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subclinical hypothyroid</td>
<td>10</td>
<td>28.80 ± 7.29 Range (21-40)</td>
<td>10</td>
</tr>
<tr>
<td>Subclinical hyperthyroid</td>
<td>8</td>
<td>29.0 ± 7.45 Range (20-42)</td>
<td>8</td>
</tr>
<tr>
<td>Euthyroid</td>
<td>17</td>
<td>33.11 ± 8.56 Range (19-45)</td>
<td>17</td>
</tr>
<tr>
<td>Control</td>
<td>10</td>
<td>31.4 ± 6.0 Range (20-40)</td>
<td>10</td>
</tr>
</tbody>
</table>

![Fig. (1): Percentage of each group of patients.](image)

The hypothesis test for means (student's t test) was applied to the control group and all patients' subgroups regarding all parameters. The results are shown in table (2). The student's t test was re-estimated for the control group and each of the patients' subgroups separately and the results are shown in table (3).

The correlation matrix was performed for all patients including all parameters and r=0.3 was taken as the critical value. We found a positive correlation between FT$_4$ and serum creatinine ($r=0.3$). But there was a negative correlation between T$_3$ and disease activity index (DAI) ($r=0.5$). There were no other significant correlations between thyroid function tests and parameters measured. Student's t test was performed for thyroid function tests (T$_3$, T$_4$ and TSH) and antithyroid antibodies. The percentage of patients with +ve and –ve antithyroid are shown in Figs. 2 and 3.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>All patients</th>
<th>T</th>
<th>p</th>
<th>S</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin *g/dl</td>
<td>13.07±0.4</td>
<td>9.45 ± 1.06</td>
<td>10.3</td>
<td>&lt;0.01</td>
<td>Hs</td>
</tr>
<tr>
<td>Red blood Corpuscles mil./ml</td>
<td>4.18 ± 0.28</td>
<td>3.7 ± 0.72</td>
<td>1.25</td>
<td>&gt;0.05</td>
<td>Ns</td>
</tr>
<tr>
<td>Total leucocytic count</td>
<td>7.3 ± 1.34</td>
<td>6.8 ± 2.3</td>
<td>0.58</td>
<td>&gt;0.05</td>
<td>Ns</td>
</tr>
<tr>
<td>Platelet</td>
<td>233.1±45.7</td>
<td>231.0 ± 62.0</td>
<td>-0.05</td>
<td>&gt;0.05</td>
<td>Ns</td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate mm/h</td>
<td>7.6 ± 2.0</td>
<td>61.6 ± 22.8</td>
<td>-7.5</td>
<td>&lt;0.01</td>
<td>Ns</td>
</tr>
<tr>
<td>Creatinine mg/dl</td>
<td>0.6 ± 0.2</td>
<td>2.7 ± 1.4</td>
<td>-4.1</td>
<td>&lt;0.01</td>
<td>Hs</td>
</tr>
<tr>
<td>T3 ng/ml</td>
<td>1.33 ± 0.48</td>
<td>1.2 ± 0.3</td>
<td>0.23</td>
<td>&gt;0.05</td>
<td>Hs</td>
</tr>
<tr>
<td>T4 ng/dl</td>
<td>1.18 ± 0.39</td>
<td>2.63 ± 3.4</td>
<td>-1.31</td>
<td>&gt;0.05</td>
<td>Ns</td>
</tr>
<tr>
<td>TSH µu/ml</td>
<td>1.72 ± 0.52</td>
<td>3.2 ± 2.29</td>
<td>-2.13</td>
<td>&lt;0.05</td>
<td>s</td>
</tr>
</tbody>
</table>
Table (3): Comparison of the data of control group and each of the patient's groups.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Euthyroid</th>
<th>Hyperthyroid</th>
<th>Hypothyroid</th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>p1</th>
<th>p2</th>
<th>p3</th>
<th>S1</th>
<th>S2</th>
<th>S3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>13.07±0.04</td>
<td>9.5±1.0</td>
<td>9.01±1.19</td>
<td>9.61±0.99</td>
<td>10.00</td>
<td>9.9</td>
<td>9.8</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>HS</td>
<td>HS</td>
<td>HS</td>
</tr>
<tr>
<td>RBC</td>
<td>4.18±0.28</td>
<td>4.06±0.74</td>
<td>3.80±0.27</td>
<td>3.66±0.96</td>
<td>0.47</td>
<td>3.09</td>
<td>1.67</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
<td>&gt;0.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>TLC</td>
<td>7.3±1.34</td>
<td>6.7±1.87</td>
<td>7.85±1.6</td>
<td>6.37±3.42</td>
<td>0.87</td>
<td>-0.60</td>
<td>0.90</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Plt</td>
<td>232.1±45.7</td>
<td>240.8±43.9</td>
<td>250.7±67.11</td>
<td>205.1±79.1</td>
<td>-0.45</td>
<td>-0.68</td>
<td>0.96</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>ESR</td>
<td>7.6±2.0</td>
<td>60.87±23.9</td>
<td>64.4±26.3</td>
<td>64.0±19.11</td>
<td>-6.9</td>
<td>-6.7</td>
<td>-9.2</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>HS</td>
<td>HS</td>
<td>HS</td>
</tr>
<tr>
<td>CR</td>
<td>0.6±0.2</td>
<td>2.27±1.5</td>
<td>3.53±0.3</td>
<td>2.3±1.6</td>
<td>-3.23</td>
<td>-24.7</td>
<td>-3.09</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>HS</td>
<td>HS</td>
<td>HS</td>
</tr>
<tr>
<td>T3</td>
<td>1.33±0.46</td>
<td>1.51±0.3</td>
<td>1.12±0.14</td>
<td>1.12±0.48</td>
<td>-1.27</td>
<td>1.56</td>
<td>1.10</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>FT4</td>
<td>1.18±0.39</td>
<td>1.28±0.34</td>
<td>7.09±4.2</td>
<td>0.62±0.34</td>
<td>-0.64</td>
<td>-4.34</td>
<td>3.4</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>TSH</td>
<td>1.72±0.52</td>
<td>2.4±1.13</td>
<td>1.47±0.39</td>
<td>0.54±1.5</td>
<td>-2.10</td>
<td>1.29</td>
<td>9.1</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
<td>S</td>
<td>NS</td>
<td>HS</td>
</tr>
</tbody>
</table>

Table (4): Results of student's t test between +ve and –ve Amic Patients.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>-ve Antimicrosomal antibodies</th>
<th>+ve Antimicrosomal antibodies</th>
<th>t</th>
<th>p</th>
<th>S</th>
</tr>
</thead>
<tbody>
<tr>
<td>T3</td>
<td>1.2±0.35</td>
<td>1.2±0.4</td>
<td>0.35</td>
<td>&gt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>T4</td>
<td>3.2±2.8</td>
<td>1.03±0.24</td>
<td>1.06</td>
<td>&gt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>TSH</td>
<td>1.64±0.35</td>
<td>4.25±2.7</td>
<td>-3.1</td>
<td>&lt;0.01</td>
<td>HS</td>
</tr>
</tbody>
</table>

Table (5): Results of student’s t test between –ve and +ve Atg. patients.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>-ve Atg.</th>
<th>+ve Atg.</th>
<th>t</th>
<th>p</th>
<th>S</th>
</tr>
</thead>
<tbody>
<tr>
<td>T3</td>
<td>1.4±0.33</td>
<td>1.1±0.34</td>
<td>1.1</td>
<td>&gt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>T4</td>
<td>3.4±3.0</td>
<td>1.2±0.37</td>
<td>1.2</td>
<td>&gt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>TSH</td>
<td>1.5±0.37</td>
<td>3.3±2.5</td>
<td>-1.9</td>
<td>&lt;0.01</td>
<td>HS</td>
</tr>
</tbody>
</table>

Fig. (2): Percentage of patients with +ve and –ve antithyroglobulin antibodies.
SLE patients were subgrouped according to the presence or absence of clinical data and according to +ve or –ve DNA and ANA. Then both groups were tested for significance difference regarding T T3, FT4 and TSH to analyze the relation between the clinical findings of SLE, diagnostic tests of SLE and thyroid function tests diagnosing thyroid condition (Table 6).

**DISCUSSION**

Clinical thyroid disease is more frequent in SLE patients than the normal population. The results of studies differ as to whether both hypothyroidism and hyperthyroidism are common in SLE patients or whether this finding a restricted to hypothyroidism alone. SLE patients have a high frequency of biochemical abnormalities of thyroid function even when they do not have clinical disease. (Weetman, 2005). The underlying pathogenetic mechanism for this association is not clear (Karmer et al., 2005).

In many aspects, mechanisms leading to organ-specific autoimmune disease are identical with mechanisms causing organ-non-specific autoimmune disease. In many cases genetic disposition combined with specific antigens can be seen. Another possible factor in terms of endogenesis in
genes. External factors are important too, such as undergoing infection, stress situations, exposure to ultraviolet radiation (Melo et al., 2005).

In the present study the results showed that 28.90% of SLE patients showed subclinical hypothyroidism, 22.66% showed subclinical hyperthyroidism while 47.37% showed normal results. In a previous study, 6% of patients showed clinical hyperthyroidism. Over 45% of the patients had elevated TSH and more than 34% had low T4 (Weetman et al., 2005). The discrepancy between both results might be due to the fact that they only considered the clinical cases not the subclinical cases. Also, Koller et al. (2004) stated that SLE patients have a high frequency of biochemical abnormalities of thyroid function even when they do not have clinical thyroid disease.

On the other hand, a study on Asian SLE patients, the percentage of thyrotoxicosis (2.8%) was found to be higher than hypothyroidism (0.9%) or thyroiditis (Melo et al., 2005).

The prevalence of thyroid disorders in Korean SLE patients was classified by Park et al. (1995). Thyroid function and disease were evaluated in 63 SLE patients of these patients. Hashimoto's thyroiditis (9.5%) as well as euthyroid sicca syndrome (14.3%) was more common than Grave’s disease (4.8%). They concluded form this study that thyroid disease was not uncommon in SLE patients.

When the student's t test was applied to the parameters measured in the hyperthyroid and the control groups, and to the hypothyroid and control groups, the hemoglobin level, ESR and serum creatinine were higher in groups with thyroid function abnormalities. That was of high statistical significance, which means that in patients with abnormal thyroid function test, disease activity was higher.

Also the results of the correlation matrix in our study showed that T3 have a +ve correlation with age and disease activity index. Also FT4 showed good positive correlation with serum creatinine and –ve correlation with TSH. These results also showed that abnormal thyroid functions are associated with increased disease activity as suggested by Pyne & Isenbery (2002). He found that biochemical primary hypothyroidism had significantly higher ESR than in euthyroids.

In our results the TSH showed no correlation with any of the parameter of activity. To the contrary, in another study TSH level in active SLE patients were significantly higher than TSH levels in inactive or control groups (Innocencio et al., 2004). That might be explained by the fact that our patients showed subclinical hypothyroidism and TSH didn't show much elevation.

Twenty percent of our SLE patients had raised antithyroglobulin antibodies and 30% had raised antimicrosomal antibodies. Normally, the prevalence of antithyroglobulin antibodies in women is 18% (Pyne & Isenbery, 2004). Again the prevalence of antimicrosomal antibodies is about 7-9% of normal adult caucasians (Macejova et al., 2006). Our results were similar to those of Pyne & Isenbery (2002) who found that the percentage of SLE patients with antithyroid antibodies was 21%.

This was also consistent with the study of St. Thomas's Hospital in London on 100 SLE patients who were tested for thyroid antibodies 1.5 to 3.5 years after diagnosis (Vianna et al., 1991). Also in the study done by Weetman et al. (2005) the prevalence of antithyroglobulin antibodies in SLE patients was 27%. High titers autoantibodies were mainly detected in Hashimoto's thyroiditis that means that antithyroid autoantibodies may be a good predictor for the detection of Hashimoto's thyroiditis developing in SLE (Punzil &
Our results showed that patients with +ve antimicrosomal antibodies had higher TSH level than those with –ve antimicrosomal antibodies and this was of high statistical significance. Also patients with +ve anti thyroglobulin antibodies had higher level of TSH than patients with –ve antithyroglobulin antibodies. This means that increased antithyroglobulin and antimicrosomal antibodies are accompanied by an increase in the level of TSH.

This is in accordance with the study of Koller et al. (2004). The latter deduced from their study that antithyroglobulin and antimicrosomal antibodies were found with increased frequency in SLE patients as compared to controls. Again, subclinical elevation of the level of TSH correlates both with the presence and the level of antithyroid antibodies. This finding was obvious in the study of Pedersen & Herlin (1995) who described 2 SLE cases that developed thyroiditis with goiter, increased thyroid function test. These results suggested that autoimmune thyroid disease is possible manifestations in some SLE patients as a result of the production of autoantibodies directed against the thyroid Viann et al. (1991).

We also found that patients with photosensitivity and renal affection showed significantly higher T₄ level (subclinical hyperthyroidism) as compared to patients not complaining of photosensitivity or renal affection. This was in accordance with the study of Tektoni et al. (2004) in which there was a higher incidence of joint and mucocutaneous involvement, lymph adenopathy and renal manifestations in SLE patients with thyroid disease than the rest of SLE patients. Also Melo et al. (2005) found that SLE patients with abnormal thyroid function tests had a lower serum albumin level and showed renal affection. This is also confirmed by our results that FT₄ correlated positively with serum creatinine.

**Conclusions:**

Although the clinical diagnosis of thyroid disease is uncommon, yet abnormal thyroid function test results are frequent in SLE patients, which correlates with the disease activity in such patients. The underlying pathogenic mechanism for this association is not clear. It is important to know whether this case of subclinical thyroid disease would develop an overt clinical form of the disease and whether thyroid hormone replacement therapy would benefit patients in the subclinical hypothyroid group.

**Recommendations:**

Further studies are to be done to clarify the previous points and to ensure the correction between the thyroid function abnormalities and different clinical features of SLE especially renal affection.

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تغيرات وظائف الغدة الدرقية والأجسام المضادة في مرضى الذبحة الحمراء

الهدف: تحديد وظائف الغدة الدرقية والأجسام المضادة في مرضى الذبحة الحمراء.

الطريقة والبحث: نُجريت هذه الدراسة على 35 مريضاً بالإضافة إلى 100 شخص أصحاء كمجموعة مختلطة، وجدنا أن الأجسام المضادة لدواء القرقرية تظهر في مرضى الذبحة الحمراء في نسبة عالية. أظهرت الأجسام المضادة في مرضى الذبحة الحمراء وجود نسبة عالية من الأجسام المضادة لدواء القرقرية في مرضى الذبحة الحمراء.

الاستنتاج: أن هناك تغيرات في وظائف الغدة الدرقية في مرضى الذبحة الحمراء وتناسب مع نشاط المرضى والأخص إصابة الكلى.

التوصيات: ونصح بعمل وظائف الغدة الدرقية والأجسام المضادة في مرضى الذبحة كجزء من التحاليل المطلوبة لإثبات البركة وإخراج الغدة الدرقية في مرضى الذبحة الحمراء.